Original Article

Renoprotective Effect of Desloratadine on Streptozotocin-induced Diabetic Nephropathy in Adult Male Long-Evans Rats

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ABSTRACT

Diabetic nephropathy (DN) is a leading cause of end-stage renal disease (ESRD) worldwide and a major microvascular complication of diabetes mellitus (DM). Despite current treatments, particularly those targeting the renin–angiotensin system, the progression of DN remains inadequately controlled. Histamine H1 receptors have emerged as potential contributors to glomerular dysfunction, mediating inflammation and oxidative stress in diabetic kidneys. Desloratadine, a potent H1 receptor antagonist, exhibits anti-inflammatory and antioxidant properties, suggesting its therapeutic potential in DN. This experimental, randomized controlled study aimed to evaluate the renoprotective effects of desloratadine in streptozotocin (STZ)-induced DN in adult male Long-Evans rats. Seventy adult male rats were divided into eight groups. Group I served as a blank control, and Group II received vehicle for STZ. Groups III–VIII were rendered diabetic via a single intraperitoneal injection of STZ (50 mg/kg). Groups III, V, and VII served as diabetic controls, while Groups IV, VI, and VIII received daily oral desloratadine (10 mg/kg) for varying durations, initiated at different stages of DN progression. Serum creatinine was measured as a marker of renal function. Renal oxidative stress were assessed by malondialdehyde (MDA), reduced glutathione (GSH), and histopathological changes in renal tissue were also evaluated. In diabetic control groups, serum creatinine, and MDA levels were significantly elevated ($p<0.01$), while GSH was significantly reduced ($p<0.01$) compared to control. Histopathological examination showed marked renal damage, and body weight was significantly decreased ($p<0.01$). Desloratadine treatment significantly improved biochemical and histological parameters at all stages of DN ($p<0.05$ to $p<0.01$), though values did not return to normal control levels. Longer duration treatment, initiated after DN onset, yielded the most pronounced histological improvements. Desloratadine also significantly prevented diabetes-induced weight loss ($p<0.01$). Desloratadine attenuated the progression of diabetic nephropathy, likely through its antioxidant mechanisms. While it may not prevent DN onset, its therapeutic benefits in slowing disease progression warrant further investigation in advanced animal models and clinical trials.

Keywords: *Diabetic nephropathy, diabetes mellitus, desloratadine, long-Evans rats*

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INTRODUCTION

Diabetic nephropathy (DN) is one of the most serious microvascular complications of diabetes mellitus (DM), accounting for approximately 40% of cases of end-stage renal disease (ESRD) globally and remaining its leading cause.[1] In addition to being a primary contributor to renal failure, DN independently increases the risk of cardiovascular diseases, adding to its burden on global health With roughly 30–45% of diabetic individuals developing DN during the course of their disease,[2] it poses a major clinical and public health challenge, particularly in low- and middle-income countries, where 80% of diabetic patients reside.[3] DM is a chronic metabolic disorder marked by persistent hyperglycemia due to insulin secretion defects, insulin resistance, or both. Sustained hyperglycemia triggers a cascade of metabolic and hemodynamic disturbances that lead to long-term organ damage, notably to the kidneys. DN typically progresses from a state of glomerular hyperfiltration and microalbuminuria to overt proteinuria and eventually ESRD, characterized by a reduced glomerular filtration rate (GFR<60ml/min/1.73m²) and irreversible histological changes such as glomerular sclerosis, interstitial fibrosis, and tubular atrophy.[1,4] Current treatment strategies for DN primarily focus on strict glycemic control and management of associated risk factors like hypertension and dyslipidemia.[5] Although pharmacological interventions such as renin–angiotensin–aldosterone system (RAAS) blockers have been shown to delay progression, they are insufficient in halting disease advancement in many patients.[6] Consequently, there is growing interest in exploring alternative therapeutic targets, particularly those addressing underlying oxidative mechanisms. In recent years, increasing evidence has highlighted the pivotal role of oxidative stress in the pathogenesis of DN.[6] Interestingly, histamine has recently been identified as a novel player in the pathophysiology of DN. Released predominantly from mast cells in response to AGEs and ROS, histamine exerts potent pro-inflammatory effects via histamine receptors (H1–H4), with the H1 receptor (H1R) being most abundantly expressed in renal tissues.[7,8] Studies have shown that blocking H1R signaling can modulate inflammatory responses, reduce oxidative stress, and confer renoprotection in diabetic models.[9-11]

Desloratadine, a second-generation, non-sedating H1 receptor antagonist, is widely used for allergic disorders. In addition to its antihistaminic effects, desloratadine has demonstrated antioxidant properties through inhibition of NF-KB activation, reduction in pro-inflammatory cytokine release, and attenuation of ROS generation.[12-14] Animal studies further support its protective effects in various models of oxidative and inflammatory injury, including renal ischemia-reperfusion and toxin-induced nephropathy.[15,16] However, its potential role in mitigating diabetic nephropathy has not yet been explored. Given the promising anti-inflammatory and antioxidant profile of desloratadine and the multifactorial pathogenesis of DN, the present study aims to evaluate the renoprotective effects of desloratadine in a streptozotocin (STZ)-induced diabetic nephropathy model in adult male Long-Evans rats. STZ is a widely used compound for inducing type 1 diabetes in animal models by selectively destroying pancreatic β-cells, resulting in persistent hyperglycemia and renal injury closely resembling human DN.[17] Exploring the therapeutic impact of desloratadine in this model may provide valuable insights into its utility as a repurposed drug for managing DN, especially in resource-limited settings where drug affordability and availability are major concerns.

METHODS

This experimental, randomized controlled study was conducted in the Department of Pharmacology & Therapeutics, Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh, between March 2021 and January 2023. Seventy adult male Long-Evans rats (7–10 weeks old, 180–220 gm in weight) were used. Male rats were selected based on greater susceptibility to STZ-induced diabetes and diabetic nephropathy (DN). The rats were housed under standard laboratory conditions (24±1°C, 12:12 h light-dark cycle), with free access to water and standard pellet diet. Animals were randomly assigned into eight groups. Two groups served as controls (blank and vehicle), while the remaining six were diabetic groups induced by streptozotocin (STZ, 50 mg/kg, intraperitoneal), divided into three untreated and three desloratadine-treated subgroups:

Group I: Blank control (n=8).

Group II: Vehicle control (0.5 ml sodium citrate buffer, pH 4.5; n=8).

Group III, V, VII: Diabetic controls (n=9 each, sacrificed at weeks 4, 8, and 12).

Group IV (STZ+DESL1): Desloratadine-treated from diabetes onset, for 3 weeks (n=9).

Group VI (STZ+DESL2): Desloratadine-treated from week 4 to 7, for 4 weeks (n=9); and

Group VIII (STZ+DESL3): Desloratadine-treated from week 4 to 11, for 8 weeks (n=9).

A total of five rats died during the study due to suspected STZ toxicity or complications; these were excluded from analysis.

Diabetes was induced by a single intraperitoneal injection of freshly prepared STZ (50 mg/kg) in 0.1 M sodium citrate buffer (pH 4.5). Rats were fasted for 12 hours prior to injection. Blood glucose was measured from the tail vein using a glucometer (Accu-Chek Instant S, Roche, Germany). Rats with fasting blood glucose ≥15 mmol/L on day 7 post-STZ injection were included in the diabetic groups.

Desloratadine tablets (Deslor 5 mg, Orion Pharma) were crushed and dissolved in distilled water. Based on an average body weight of 200 g, each rat received 2 mg/day (10 mg/kg/day) orally via gavage using a flexible ball-tipped feeding needle. Dosing volumes were within recommended limits (10–20 ml/kg/day). Treatment schedules varied by group, as outlined above.

Random blood glucose was monitored twice weekly. Rats with RBS >30 mmol/L received subcutaneous long-acting insulin (2–4 units) per a predefined scale. Hypoglycemic rats (RBS <15 mmol/L) were treated with 20% glucose solution. At designated time points (weeks 4, 8, and 12), rats were euthanized under light anesthesia using chloroform. Blood was collected via carotid artery into plain test tubes, allowed to clot, and centrifuged to obtain serum, stored at –20°C. **Blood glucose was m**easured using glucose oxidase strip method, while s**erum creatinine was e**stimated by Jaffe's Alkaline-Picrate method. For r**enal oxidative stress,** kidney homogenates were assayed for malondialdehyde (MDA), reduced glutathione (GSH), using standard spectrophotometric methods and ELISA kits (SunLong Biotech, made in China). Both kidneys were excised; the left kidney was stored at –80°C for biochemical assays, and the right kidney fixed in 10% formalin for histopathological analysis. Kidney sections were stained with hematoxylin and eosin (H&E) and periodic acid-Schiff (PAS) for assessment of glomerular and tubular changes.

Data was analyzed using SPSS software version 26.0 for Windows. Results were expressed as mean±SD (standard deviation). One-way ANOVA followed by post-hoc unpaired and paired t-tests were used for group comparisons. A p-value <0.05 was considered statistically significant.

RESULTS

Table-I shows fasting blood glucose levels were significantly elevated in all STZ-induced diabetic groups (Groups III–VIII) compared to the control Group II (5.24±0.60 mmol/L; $p<0.001$), confirming successful induction of diabetes. No significant differences were observed among the diabetic groups themselves ($p>0.05$). To evaluate the progression of diabetic nephropathy over time, serum creatinine levels were measured in STZ-induced diabetic rats sacrificed at three different time points and compared with the non-diabetic control group (Table-II). In Group II (control), the mean±SD of serum creatinine level was 0.54±0.06 mg/dl. A time-dependent elevation in serum creatinine was observed in diabetic rats. Desloratadine significantly reduced serum creatinine levels in diabetic rats at all time points. At week 4, levels decreased from 0.90±0.15 mg/dl (Group III) to 0.73±0.05 mg/dl in Group IV ($p<0.05$). At week 8, creatinine dropped from 1.82±0.09 mg/dl (Group V) to 1.62±0.14 mg/dl in Group VI ($p<0.01$). At week 12, levels declined from 2.35±0.15 mg/dl (Group VII) to 2.06±0.16 mg/dl in Group VIII ($p<0.01$). Despite improvements, all desloratadine-treated groups remained above the control Group II level (0.54±0.06 mg/dl) (Table-III). Table-IV shows renal tissue MDA levels significantly increased with the progression of diabetes. Compared to the control Group II (0.84±0.11 µmol/L), MDA concentrations were elevated in Group III (1.83±0.34 µmol/L; $p<0.01$), Group V (2.82±0.31 µmol/L; $p<0.001$), and Group VII (4.06±0.21 µmol/L; $p<0.001$). The increases were also significant between Group V vs. III and Group VII vs. V ($p<0.001$). Renal GSH levels decreased progressively with the duration of diabetes. Compared to the control Group II (3.33±0.18 mg/ml), significant reductions were observed in Group III (2.23±0.20 mg/ml; $p<0.01$), Group V (1.60±0.15 mg/ml; $p<0.001$), and Group VII (1.16±0.16 mg/ml; $p<0.001$). The decline was also significant between Group V vs. III and Group VII vs. V ($p<0.001$), indicating worsening oxidative stress with prolonged diabetes. Table-V shows that desloratadine treatment significantly reduced renal MDA concentrations in all diabetic

groups. At week 4, MDA levels decreased from 1.83±0.34 µmol/L in Group III to 1.43±0.22 µmol/L in Group IV ($p<0.05$). At week 8, Group VI showed reduced MDA (2.47±0.19 µmol/L) compared to Group V (2.82±0.31 µmol/L) ($p<0.05$). Similarly, at week 12, Group VIII had lower MDA (3.77±0.17 µmol/L) than Group VII (4.06±0.21 µmol/L) ($p<0.05$). Despite these reductions, MDA levels remained higher than the control (Group II: 0.84±0.11 µmol/L). Desloratadine significantly improved renal GSH concentrations in diabetic rats at all time points. At week 4, GSH increased from 2.23±0.20 mg/ml (Group III) to 2.66±0.22 mg/ml in Group IV ($p<0.01$). At week 8, GSH rose from 1.60±0.15 mg/ml (Group V) to 1.96±0.10 mg/ml in Group VI ($p<0.01$). At week 12, GSH increased from 1.16±0.16 mg/ml (Group VII) to 1.56±0.28 mg/ml in Group VIII ($p<0.01$). Despite these improvements, GSH levels remained lower than the control Group II (3.33±0.18 mg/ml). Table-VI illustrates results of histopathological examination of kidney tissues in different groups of rats. Group I (blank control) and Group II (vehicle control) showed normal glomerular and tubular structures with no pathological changes (mean scores: 0.00±0.00 for all parameters). In Group III (STZ-induced, 4th week), mild renal changes were observed, including glomerular hypertrophy (0.86±0.38) and mesangial expansion (0.71±0.49), with a significant increase in mesangial expansion ($p<0.01$). Group IV (STZ+desloratadine, 4th week) showed reduced histological scores, with a significant decrease in mesangial expansion (0.13±0.35) ($p<0.05$). In Group V (STZ-induced, 8th week), further deterioration occurred with significant increases in glomerular hypertrophy (2.38±0.52, $p<0.001$), GBM thickening, and mesangial expansion (both $p<0.05$). Group VI (STZ+desloratadine, 8th week) showed improvements, with significant reduction in glomerular hypertrophy (1.00±0.54) ($p<0.01$), though other changes were not statistically significant. By the 12th week, Group VII (STZ-induced) exhibited severe damage, including nodular sclerosis (0.25±0.46), with significant increases in all other parameters ($p<0.001$). Group VIII (STZ+desloratadine, 12th week) showed marked renoprotection, with significant reduction in glomerular hypertrophy, mesangial expansion, GBM thickening, and mononuclear cell infiltration ($p<0.05$). Nodular sclerosis was absent. Group II (Vehicle control, sacrificed on Day 1 of Week 4): H&E stained sections showed normal histoarchitecture with well-defined glomeruli, intact renal tubules, and unremarkable interstitium (Fig. 1). Group III (STZ-induced diabetic group, sacrificed on Day 1 of Week 4): H&E stained sections demonstrated mild glomerular hypertrophy and early mesangial expansion (Fig. 2). Group IV (Desloratadine-treated diabetic group, sacrificed on Day 1 of Week 4): H&E stained sections showed mild amelioration of glomerular hypertrophy, with near-normal mesangial appearance and preserved tubular structure (Fig. 3). Group V (STZ-induced diabetic group, sacrificed on Day 1 of Week 8): H&E stained sections revealed glomerular hypertrophy, marked mesangial expansion, prominent mononuclear cell infiltration, and glomerular basement membrane thickening (Fig. 4). Group VI (Desloratadine-treated diabetic group, sacrificed on Day 1 of Week 8): H&E stained sections displayed amelioration of glomerular hypertrophy, reduced mesangial expansion, and decreased mononuclear cell infiltration compared to Group V (Fig. 5). Group VII (STZ-induced diabetic group, sacrificed on Day 1 of Week 12): H&E stained sections exhibited severe mesangial expansion, podocyte loss and vacuolization, mononuclear cell infiltration, and pronounced glomerular basement membrane thickening (Fig. 6). Group VIII (Desloratadine-treated diabetic group, sacrificed on Day 1 of Week 12): H&E stained sections revealed notable amelioration of me1sangial expansion, improved podocyte morphology, and decreased mononuclear cell infiltration compared to Group VII (Fig. 7).

Table-I: *Comparison of blood glucose level among control and experimental groups of rats*

	Group II n=8 Mean±SD	Group III n=7 Mean±SD	Group IV n=8 Mean±SD	Group V n=8 Mean±SD	Group VI n=8 Mean±SD	Group VII n=8 Mean±SD	Group VIII n=7 Mean±SD
Blood glucose (mmol/L)	5.24±0.60	24.49±4.65	25.58±3.54	24.78±4.61	25.41±3.62	25.66±4.30	25.14±3.04
p-value	–	0.000^{a} 0.624^{x}	0.000^{b} -	0.000^{c} 0.763^{y}	0.000^{d} -	0.000^{e} 0.789^{z}	0.000^{f} -

Table-II: *Comparison of serum creatinine level (mg/dl) among STZ induced diabetic groups (Group III, Group V, Group VII) and control group (Group II) of rats*

	Group II n=8 Mean±SD	Group III n=7 Mean±SD	Group V n=8 Mean±SD	Group VII n=8 Mean±SD
Serum creatinine (mg/dl)	0.54±0.06	0.90±0.15	1.82±0.09	2.35±0.16
p-value	0.003[a]	0.000[b]	0.000[c]	0.000[x]0.000[y]

Table-III: *Comparison of serum creatinine level among STZ induced diabetic groups and concomitantly desloratadine treated groups*

	Group III n=7 Mean±SD	Group IV n=8 Mean±SD	Group V n=8 Mean±SD	Group VI n=8 Mean±SD	Group VII n=8 Mean±SD	Group VIII n=7 Mean±SD
Serum creatinine(mg/dl)	0.90±0.15	0.73±0.05	1.82±0.09	1.62±0.14	2.35±0.15	2.06±0.16
p-value	-	0.022[x]	-	0.005[y]	-	0.003[z]

Table-IV: *Comparison of renal tissue homogenate MDA and GSH concentration among STZ induced diabetic groups and control group of rats*

Variables	Group II n=8 Mean±SD	Group III n=7 Mean±SD	Group V n=8 Mean±SD	Group VII n=8 Mean±SD
MDA conc.(µmol/L)	0.84±0.11	1.83±0.34	2.82±0.31	4.06±0.21
p-value	0.001[a]	0.000[b]	0.000[c]	0.000[x]0.000[y]
GSH conc.(mg/ml)	3.33±0.18	2.23±0.20	1.60±0.15	1.16±0.16
p-value	0.001[a]	0.000[b]	0.000[c]	0.000[x]0.000[y]

Table-V: *Comparison of MDA and GSH concentration (µmol/L) among STZ induced diabetic groups and concomitantly desloratadine treated groups*

Variables	III n=7 Mean±SD	IV n=8 Mean±SD	V n=8 Mean±SD	VI n=8 Mean±SD	VII n=8 Mean±SD	VIII n=7 Mean±SD
MDA conc.(µmol/L)	1.83 ± 0.34	1.43±0.22	2.82±0.31	2.47±0.19	4.06±0.21	3.77±0.17
p value	-	0.025[x]	-	0.021[y]	-	0.011[z]
GSH conc.(mg/ml)	2.23±0.20	2.66±0.22	1.60±0.15	1.96±0.10	1.16±0.16	1.56±0.28
p value	-	0.002[x]	-	0.001[y]	-	0.008[z]

Table-VI: *Histopathology of renal tissue with qualitative changes and arbitrary score*

Variables	Group I n=8 Mean±SD	Group II n=8 Mean±SD	Group III n=7 Mean±SD	Group IV n=8 Mean±SD	Group V n=8 Mean±SD	Group VI n=8 Mean±SD	Group VII n=8 Mean±SD	Group VIII n=7 Mean±SD
Glomerular hypertrophy Mean±SD	0.00±0.00	0.00±0.00	0.86±0.38	0.38±0.52	2.38±0.52	1.00±0.54	3.13±0.35	1.29±0.49
p^Avalue	-	-	0.009[a]	0.058[b]	0.000[c]	0.001[d]	0.000[e]	0.000[f]
Mononuclear cell infiltration Mean±SD	0.00±0.00	0.00±0.00	0.43±0.54	0.00±0.00	0.75±0.46	0.25±0.46	1.5±0.54	0.57±0.53
p^B value	-	-	0.078[a]	0.078[b]	0.003[c]	0.049[d]	0.000[e]	0.005[f]
Podocyte injury Mean±SD	0.00±0.00	0.00±0.00	0.00±0.00	0.00±0.00	0.25±0.46	0.00±0.00	0.88±0.64	0.29±0.49
p^C value	-	-	-	-	0.170[c]	0.170[d]	0.006[e]	0.065[f]
Glomerular basement membrane thickening Mean±SD	0.63±0.52	1.88±0.84	0.71±0.49	0.00±0.00	0.00±0.00	0.43±0.54	0.00±0.00	1.13±0.64
p^D value	-	-	0.078[a]	0.078[b]	0.002[c]	0.110[d]	0.000[e]	0.007[f]
Mesangial cell expansion Mean±SD	0.00±0.00	0.00±0.00	0.71±0.49	0.13±0.35	1.13±0.64	0.50±0.54	1.88±0.35	0.86±0.38
p^E value	-	-	0.008[a]	0.023[b]	0.002[c]	0.053[d]	0.000[e]	0.001[f]
Nodular sclerosis Mean±SD	0.00±0.00	0.00±0.00	0.00±0.00	0.00±0.00	0.00±0.00	0.00± 0.00	0.25±0.46	0.00± 0.00
p^F value	-	-	-	-	-	-	0.170[e]	0.170[f]

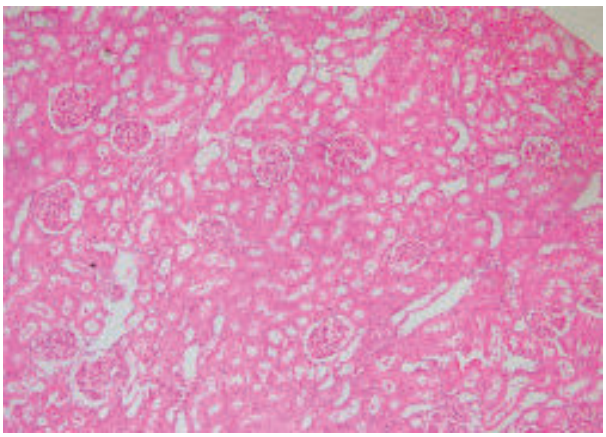

Fig. 1: *Histopathology slide showing normal histoarchitecture with well-defined glomeruli, intact renal tubules, and unremarkable interstitium (H&E stain; ×100).*

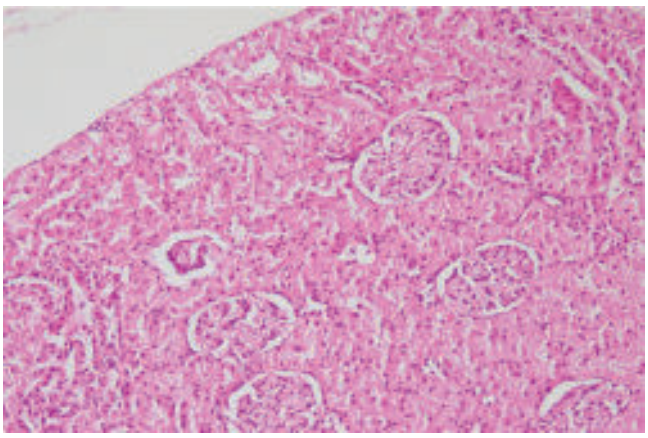

Fig. 2: *Histopathology slide showing mild glomerular hypertrophy and early mesangial expansion (H&E stain; ×100).*

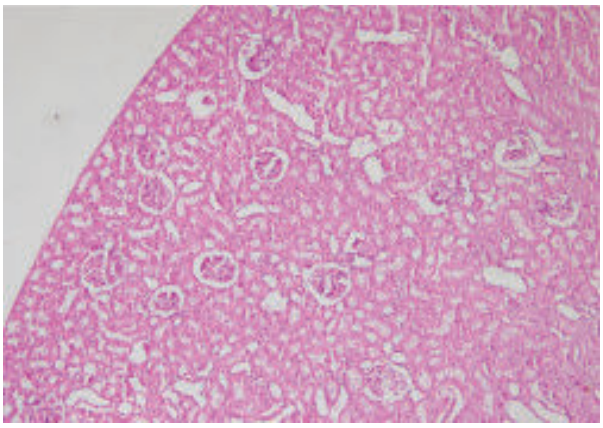

Fig. 3: *Histopathology slide showing mild amelioration of glomerular hypertrophy, with near-normal mesangial appearance and preserved tubular structure (H&E stain; ×100).*

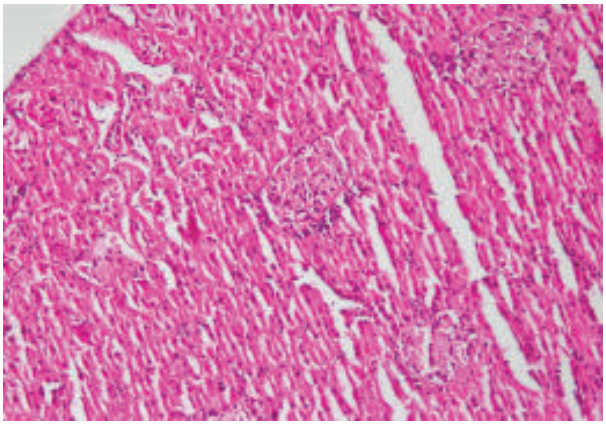

Fig. 6: *Histopathology slide showing severe mesangial expansion, podocyte loss and vacuolization, mononuclear cell infiltration, and pronounced glomerular basement membrane thickening (H&E stain; ×100).*

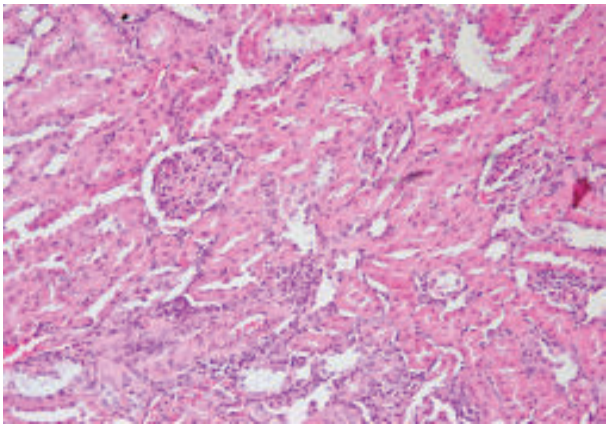

Fig. 4: *Histopathology slide showing glomerular hypertrophy, marked mesangial expansion, prominent mononuclear cell infiltration, and glomerular basement membrane thickening (H&E stain; ×100).*

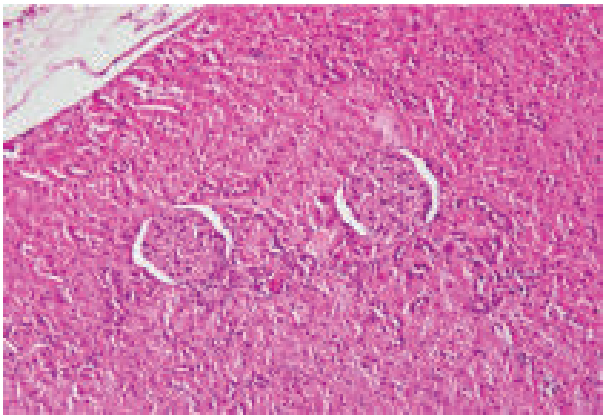

Fig. 7: *Histopathology slide showing notable amelioration of mesangial expansion, improved podocyte morphology, and decreased mononuclear cell infiltration (H&E stain; ×100).*

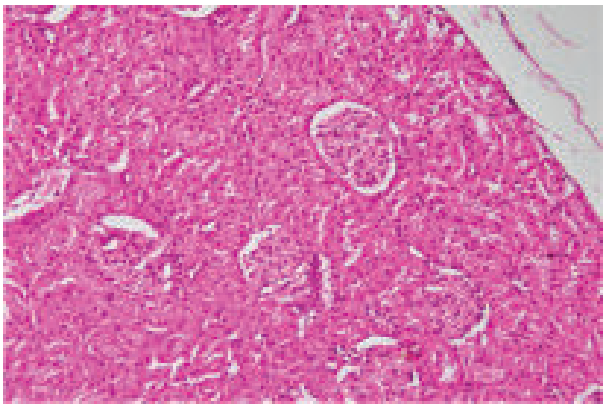

Fig. 5: *Histopathology slide showing amelioration of glomerular hypertrophy, reduced mesangial expansion, and decreased mononuclear cell infiltration (H&E stain; ×100).*

DISCUSSION

To our knowledge, this is the first study to investigate the potential renoprotective effects of desloratadine in a rat model of streptozotocin (STZ)-induced diabetic nephropathy (DN). The present findings demonstrate that desloratadine exerts beneficial effects across multiple stages of DN progression, possibly through its antioxidant mechanisms.STZ-induced diabetes in rats resulted in significant renal functional impairment, as evidenced by elevated serum creatinine levels. This aligns with previous reports indicating kidney dysfunction as a hallmark of DN. Treatment with desloratadine significantly attenuated serum creatinine elevation when administered both at the early stage of diabetes and at established DN stages, suggesting a protective

effect on renal function irrespective of treatment onset. While creatinine levels in treated groups remained higher than in normal controls, the consistent reduction observed supports a functional renoprotective role. These results are consistent with earlier studies where H1 receptor antagonists such as levocetirizine, cetirizine, and rupatadine also conferred renal protection in diabetic rats.[9,11] Oxidative stress is another central contributor to DN pathophysiology. Diabetic control rats showed significantly elevated malondialdehyde (MDA) levels and depleted reduced glutathione (GSH) content, indicating increased lipid peroxidation and compromised antioxidant defense. Desloratadine treatment significantly lowered MDA levels and restored GSH content, irrespective of treatment timing. These findings suggest that desloratadine mitigates oxidative damage, possibly via direct antioxidant activity or by reducing reactive oxygen species through inflammatory pathway modulation. Previous in vivo and in vitro studies support the antioxidant effects of desloratadine,[14,18] reinforcing its potential as a therapeutic agent in oxidative stress-related renal damage.Histopathological examination of renal tissue revealed structural abnormalities in diabetic control rats, which worsened with disease progression. Desloratadine-treated groups showed non-significant improvements in early intervention phases but demonstrated significant structural preservation when treatment was initiatedat the DN stage and continued for a longer duration. These histological improvements likely reflect the antioxidative actions of desloratadine. Notably, the renoprotective histological changes are consistent with findings from previous studies using other H1 receptor antagonists[9,11] and from desloratadine's effects in other organ systems subjected to ischemia-reperfusion injury.[15,16] Collectively, the current data provide compelling evidence that desloratadine confers renoprotection in STZ-induced DN, regardless of the stage at which treatment is initiated. Its capacity to modulate oxidative stress pathways may underlie these beneficial effects. However, it should be noted that while desloratadine ameliorated renal damage, it did not fully restore all parameters to baseline, suggesting a partial protective effect that might be enhanced through combination therapies or higher dosing strategies.

CONCLUSION

Our findings suggest that desloratadine holds promise as a novel therapeutic candidate for diabetic nephropathy. Its renoprotective effects, observed across various stages of disease progression, appear to be mediated through attenuation of oxidative stress. Further mechanistic studies and clinical investigations are needed to validate these preclinical findings and to explore desloratadine's potential as a pharmacological intervention for diabetic nephropathy.

Conflict of Interest: None declared.

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Ethical Approval: Ethical approval was obtained from the Institutional Review Board (IRB) of Bangabandhu Sheikh Mujib Medical University, Dhaka, Bangladesh (Ref: BSMMU/2022/3780). All animal procedures adhered to the standard ethical guidelines on animal welfare and care and use of laboratory animals for research.[19,20]

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