RESEARCH ARTICLE

Mitigating Arsenite-Induced Clinico-Pathological and Ultrastructural Alterations in Wistar Rats using *Withania somnifera* Root Extract

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Abstract

Sodium arsenite induces marked haematological and pathological alterations in multiple organs, including the liver, stomach, kidneys, brain, testes, and skin, with chronic exposure known to predispose individuals to skin carcinogenesis. The present study evaluated the protective efficacy of *Withania somnifera* (Ashwagandha) root extract against arsenic-induced toxicity. 24 Wistar rats of 150-200 gm were randomly divided into 4 equal groups. Rats in Group A served as the untreated control, in Group B received sodium arsenite at a dose of 4 mg/kg b. wt. orally for 45 days. Group C was administered an aqueous root extract of *W. somnifera* @ 200 mg/kg body weight concurrently with sodium arsenite for the same duration. Group D received only aqueous root extract of *W. somnifera* @ 200 mg/kg body weight. Affected rats exhibited macrocytic hypochromic anaemia and an elevated leukocyte count and oxidative stress. Major visceral organs of toxicity group showed degeneration, necrosis, and leukocytic infiltration. Ultrastructural examination of hepatic tissue revealed indistinct nuclear membranes, electron-dense mitochondria, and disrupted, discontinuous rough endoplasmic reticulum (RER). *W. somnifera* root extract reduced the haemato-biochemical changes and oxidative stress. It also exhibited protection of 16.95% and 19.49% in gross and histopathological lesions, and restoration of nuclear integrity and RER architecture. In conclusion, *W. somnifera* root extract mitigated sodium arsenite–induced toxicity in Wistar rats by reducing oxidative stress and preserving cellular structure, thereby demonstrating its potential as a protective agent against arsenic toxicity.

Key words: Anaemia, Arsenic, Haematological, Hepatotoxicity, Lipid peroxidation, Nephrotoxicity.

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Introduction

Arsenic (As) is a naturally occurring metalloid that contaminates groundwater through leaching, erosion, and weathering of arsenic-bearing rocks. High groundwater arsenic levels have been reported in parts of Asia and the western United States (Podgorski and Berg, 2020). In India, natural arsenic contamination occurs widely across central and eastern regions (Sankhla *et al.,* 2018). Arsenic is also used in wood preservatives, pesticides, herbicides, semiconductors, pharmaceuticals, and glass, making anthropogenic exposure significant (Bhattacharya *et al.,* 2011). Dietary intake arises from fish, shellfish, meat, poultry, dairy products, and cereals, with seafood containing primarily organic arsenic forms (De Gieter *et al.,* 2002). Rice, an efficient arsenic bio-accumulator, accumulates higher levels than other cereals due to long-term soil persistence (Chowdhury *et al.,* 2018). Animals are exposed through contaminated feed, water, and arsenic-based pesticides such as dipping solutions. Common signs of arsenic poisoning include skin lesions, weakness, anorexia, nausea, vomiting, edema, pigmentation changes (arsenical melanosis), hepatomegaly, respiratory disorders, and increased risk of skin cancer (Sandhu and Brar, 2008).

Extracts from several medicinal plants, such as *Phyllanthus fraternus*, *Terminalia arjuna*, *Mentha piperita*, *Hibiscus sabdariffa*, *Withania somnifera*, *Pteris longifolia*, and *Bauhinia variegate,* have shown antioxidant, hepatoprotective, nephroprotective, and genoprotective activities through modulation of lipid peroxidation and free radical scavenging (Bhattacharya, 2017; Susan *et al.,* 2019). *Withania somnifera* (Ashwagandha), distributed across Africa to India (Purohit and Vyas, 2004), possesses rejuvenating, anti-ageing, immunomodulatory, and health-promoting properties (Sheela *et al.,* 2014). Its active constituents include withanolides, withaferins, and

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various alkaloids and saponins (Mishra and Singh, 2000). It exhibits dose-dependent hepatoprotection against heavy metal and environmental toxicants (Bhattacharya *et al.,* 2000) and reduces reactive oxygen species while regulating apoptosis (Dar *et al.,* 2015). Considering these properties, we investigated the protective efficacy of *Withania somnifera* (WS) root extract against experimentally induced sub-acute arsenic toxicity in rats.

Materials and Methods

The experimental protocol was approved by the Institutional Animal Ethics Committee (IAEC), as per Letter No. IAEC/RES/03/01-10/8/2023. Twenty-four rats of 150-200 gm were reared in the College Animal House under strict hygienic and environmental conditions in accordance with the guidelines of the CCSEA. They were given access to pelletized feed and water *ad libitum*. The rats were randomly divided into four groups (A, B, C & D) with 6 rats in each group. Each rat was observed individually and body weight was recorded weekly.

The treatment received by various experimental groups was as follows. Group A- untreated rats, served as control. Rats in Group B (SA) - received sodium arsenite @ 4 mg/kg body weight orally, daily for 45 days, while those in Group C (SA+WS) - received sodium arsenite @ 4 mg/kg body weight + *Withania somnifera* root extract @ 200 mg/kg body weight orally, daily for 45 days. Rats in Group D (WS) - received only *Withania somnifera* root extract supplementation @ 200 mg/kg body weight orally, daily for 45 days.

Hematolo-Biochemical Parameters

On the 46th day of the study, blood samples were collected from the retro-orbital sinus of each rat across all groups, followed by sacrifice through atlanto-occipital dislocation under isoflurane anesthesia. Blood was collected in sterile, dry vials containing EDTA and detailed haematological analysis was performed using a haematology analyzer (Mindray, Modal No. RM-303-03, Sr No. 3903). The blood samples collected without anticoagulant were centrifuged at x 1000 g for 15 min at 4ºC and serum stored in deep freezer was analysed for the biochemical test like alanine transaminase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), gamma-glutamyltransferase (GGT), total protein, albumin, creatinine, and blood urea nitrogen (BUN) by reagents kit (Aspen Laboratories Pvt. Ltd.) using the bio-chemistry auto-analyzer (CAT No. BGS-246, Biogen).

Oxidative Stress

Lipid peroxidation was assessed by measuring malondialdehyde (MDA) levels using the thiobarbituric acid-reactive substances (TBARS) test (Fernanda *et al.,* 2005). Superoxide dismutase (SOD) activity was estimated following the method described by Madesh and Balasubramanian (1998).

Pathological Studies

On the 46th day, all rats from each group were sacrificed using inhalation anesthesia with isoflurane followed by decapitation. A thorough necropsy examination was performed on all rats, and any gross lesions observed were recorded. All the vital organs (brain, heart, lung, liver, spleen, stomach, intestine, kidney and testes) were collected in 10% neutral buffered formalin for histopathological examination. The formalin-fixed tissue was washed and processed for paraffin embedding technique. The sections were subsequently stained using standard technique of the hematoxylin and eosin (H&E) staining method. Lesion scoring of gross and histopathological changes was done and further percent protective effect due to *W. Somnifera* root extract supplementation in sub-acute sodium arsenite toxicity was calculated (Witter, 1982).

Ultrastructural Studies

Ultra-structural changes in liver tissue were assessed by fixing samples in Karnovsky's fluid for 24 h at 4°C, followed by washing in 0.1 M PBS (pH 7.4). Tissues were post-fixed in 1% osmium tetroxide for 1 h at 4°C, washed, and dehydrated through graded acetone. After clearing in toluene, samples were infiltrated and embedded in Araldite CY212 resin. Ultrathin sections (70-80 nm) were cut using an ultramicrotome, mounted on copper grids, stained with uranyl acetate and lead citrate, and examined under a TECNAI 200 kV TEM (FEI).

Results and Discussion

Body Weight and Clinical Findings

Across all intervals, no statistically significant differences in body weight were observed among the groups. However, arsenic-treated rats (Group B) showed a gradual reduction in body weight (Day 7, 14, and 46), likely due to reduced feed and water intake and gastrointestinal dysfunction, as confirmed by pathological lesions in the stomach and intestine. Similar observations were reported in earlier studies (Bhattacharya and Haldar, 2012; Messarah *et al.,* 2012). Groups A and D did not show any clinical signs throughout the experiment. Clinical signs in Group B (arsenite-treated rats) included alopecia, dullness, anorexia, abnormal gait, circling, depression, and nervous signs after 35 days. These signs corroborated previous findings in arsenic toxicity (Bhattacharya and Haldar, 2012; Messarah *et al.,* 2012). Group C (arsenic + *W. somnifera*) exhibited similar signs, but lacked neurological symptoms, supported by histopathology showing preserved neuronal integrity, indicating neuroprotective effects of *Withania somnifera*.

Haematological Alterations

Haematological changes are presented in Table 1. The haematological parameters of groups A, C and D did not

Table 1: Mean (± SE) values of hematological parameters of different experimental groups (n=6)

Groups	RBC parameters						WBC parameters				Platelet parameters				
	RBC (10^{12}/L)	Hb (g/dL)	HCT (%)	MCV (fL)	MCH (pg)	MCHC (g/dL)	TLC (10^9/L)	LYM (10^9/L)	MID (10^9/L)	NEUT (10^9/L)	PLT (10^9/L)	MPV (fL)	PDW (%)	PCT (%)	P-LCR (%)
A	5.96^{a} ±0.36	12.25^{a} ±0.69	44.56^{a} ±2.72	74.85^{a} ±0.50	20.53^{a} ±0.18	27.46^{b} ±0.18	7.68^{ab} ±043	4.65^{ab} ±0.37	0.14^{a} ±0.01	2.89^{ab} ±0.31	376.50^{a} ±28.21	8.40^{a} ±0.17	11.05^{ab} ±0.93	0.31^{ab} ±0.02	18.05^{a} ±1.62
B	5.65^{a} ±0.11	11.73^{a} ±0.24	46.31^{a} ±0.94	82.03^{b} ±1.65	20.70^{a} ±0.31	25.33^{a} ±0.82	9.73^{b} ±1.76	5.75^{b} ±1.08	0.20^{a} ±0.04	3.78^{ab} ±0.66	366.16^{a} ±28.52	9.00^{b} ±0.18	12.93^{b} ±0.64	0.32^{ab} ±0.02	22.93^{b} ±1.87
C	6.28^{a} ±0.22	12.86^{a} ±0.50	45.60^{a} ±1.73	72.61^{a} ±0.72	20.41^{a} ±0.29	28.15^{b} ±0.31	11.05^{b} ±1.30	6.45^{b} ±0.76	0.23^{a} ±0.06	4.37^{b} ±0.53	346.83^{a} ±19.09	8.03^{a} ±0.11	9.76^{a} ±0.35	0.27^{a} ±0.01	14.61^{a} ±0.84
D	5.90^{a} ±0.10	12.11^{a} ±0.25	44.38^{a} ±0.77	75.38^{a} ±1.35	20.48^{a} ±0.11	27.26^{b} ±0.54	5.80^{a} ±0.68	3.21^{a} ±0.36	0.18^{a} ±0.04	2.73^{a} ±0.34	415^{a} ±30.52	8.35^{a} ±0.14	11.41^{ab} ±0.51	0.36^{b} ±0.03	16.63^{a} ±1.14

Groups: A=control, B=Arsenic, C= Arsenic + WS, D= WS. Values with different superscripts within the same column differ significantly between groups at $p<0.05$.

Table 2: Mean (± SE) values of serum biochemical parameters across different experimental groups (n=6)

Group	Biochemical parameters							
	ALT (U/L)	AST(U/L)	ALP (U/L)	GGT (U/L)	TP (g/dL)	Albumin (g/dL)	BUN (mg/dL)	Creatinine (mg/dL)
A	$76.19^{a} \pm 5.14$	$132.79^{a} \pm 7.56$	$265.39^{a} \pm 16.45$	$19.38^{b} \pm 0.23$	$8.51^{b} \pm 0.56$	$3.53^{ab} \pm 0.49$	$31.90^{c} \pm 2.99$	$0.85^{a} \pm 0.02$
B	$140.26^{c} \pm 4.41$	$173.38^{c} \pm 1.47$	$417.19^{b} \pm 42.61$	$23.04^{d} \pm 0.35$	$6.13^{a} \pm 0.52$	$2.73^{a} \pm 0.13$	$32.56^{c} \pm 2.23$	$4.45^{c} \pm 0.37$
C	$116.31^{b} \pm 4.97$	$156.82^{b} \pm 2.66$	$470.89^{b} \pm 32.61$	$21.45^{c} \pm 0.29$	$7.48^{ab} \pm 0.64$	$4.05^{bc} \pm 0.14$	$23.48^{b} \pm 1.55$	$3.62^{b} \pm 0.25$
D	$83.97^{a} \pm 5.03$	$119.90^{a} \pm 4.67$	$226.12^{a} \pm 8.82$	$17.06^{a} \pm 0.30$	$8.10^{b} \pm 0.37$	$4.56^{c} \pm 0.37$	$15.82^{a} \pm 0.20$	$0.56^{a} \pm 0.05$

Groups: A=control, B=Arsenic, C= Arsenic + WS, D= WS. Values with different superscripts within the same column differ significantly between groups at $p<0.05$.

differ significantly. Arsenic-treated rats (Group B) showed significantly elevated MCV and reduced MCHC, indicating macrocytic hypochromic anaemia, which aligned with earlier studies showing arsenic-induced impairment of erythropoiesis and haemoglobin synthesis (Messarah *et al.*, 2012). Blood smear evaluation confirmed basophilic stippling. Group C, receiving arsenic + *W. somnifera*, showed no such changes, suggesting protection against oxidative injury to bone marrow and hepatocytes.

Total WBC count was significantly increased in Groups B and C compared to group D, consistent with previous reports attributing leukocytosis to inflammatory responses induced by toxicants (Tandan *et al.*, 2012). Elevated lymphocyte and neutrophil counts were recorded in groups B and C compared to group D which reflect immune activation. The slightly higher values in Group C may indicate the immunostimulatory action of *W. somnifera*. Platelet indices revealed elevated MPV, PDW, and P-LCR in group B, indicating platelet anisocytosis, likely due to toxic suppression of bone marrow activity (Saran *et al.*, 2022). The PCT (%) in Group D (*W. somnifera*) was significantly higher as compared to all other groups suggesting stimulation of platelet production by the extract.

Biochemical Changes

Biochemical changes are presented in Table 2. The groups A and D revealed normal serum enzyme levels. Arsenic exposure produced significant increase in ALT, AST, ALP, and GGT levels in Groups B and C, representing compromised liver integrity. These results aligned with established reports that arsenic induces hepatic injury through free radical generation and oxidative stress (Sharma *et al.*, 2009; Chattopadhyay *et al.*, 2011; Forkan *et al.*, 2016). Total protein levels were significantly reduced in Group B, whereas Group C maintained near-normal values, demonstrating the hepatoprotective action of *W. somnifera*. Albumin level of group D was significantly higher than group A, whereas, it was significantly lower in Group B compared to C, again indicating reduced hepatic

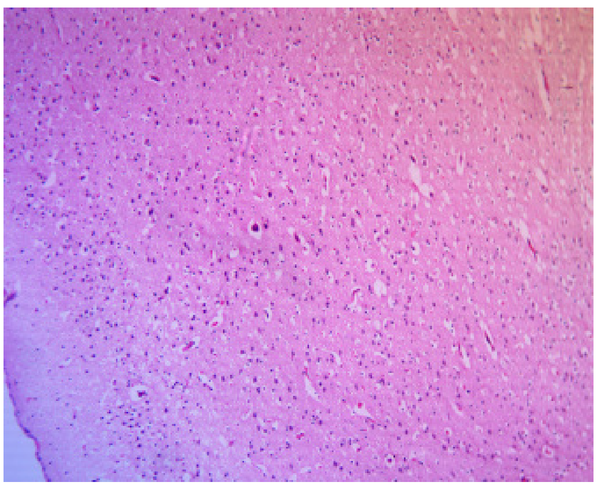

Fig. 1: Brain showing cerebral cortex having vacuolation in neuropil, increase in microglial cells, dilated capillaries and neuronophagia (Group B, 100X H&E)

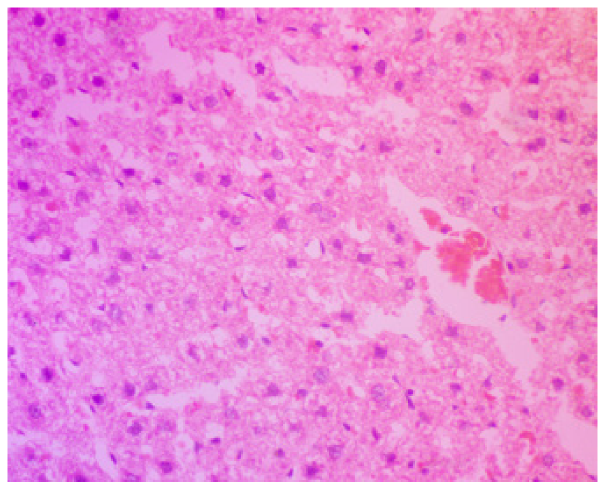

Fig. 2: Liver showing fatty change in hepatocytes with signet ring appearance, congested vessel and coagulative necrosis of hepatocytes (Group B, 400X H&E)

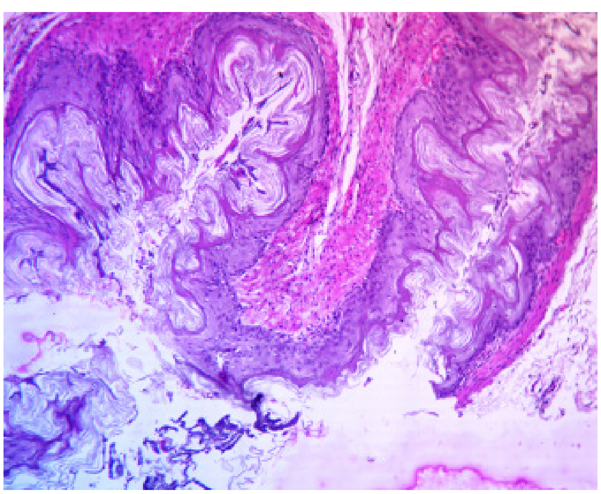

Fig. 3: Stomach showing hyper-keratinization of the epithelium (Group B, 100X H&E)

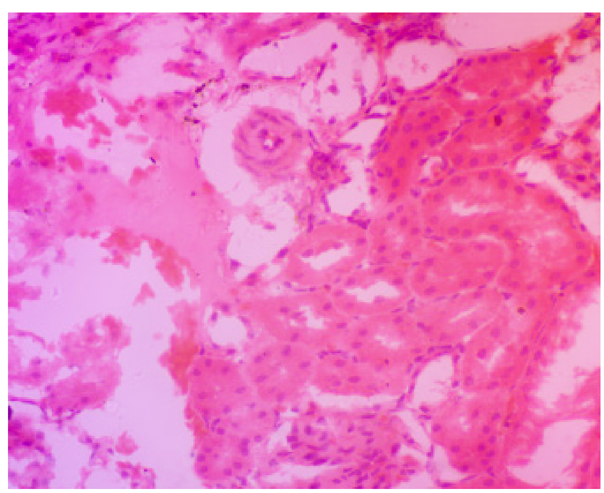

Fig. 4: Kidney showing edema, tubular necrosis, and thickened vessel wall (Group B, 400X H&E)

damage in the *W. somnifera*-supplemented group (Tandan *et al.,* 2012). The BUN values were lower in group C and D as compared to group A and B. Creatinine levels were elevated in Groups B and C, suggesting renal damage; however, Group C exhibited significantly lower BUN levels compared to B, consistent with earlier findings that arsenic affects renal function through tubular injury (Messarah *et al.,* 2012) and *W. somnifera* has reduced the renal damage.

Oxidative Stress Markers

Arsenic toxicity significantly increased hepatic MDA levels in Groups B and C ($p<0.05$) as compared to control and Group D. Moreover, Group B had significantly higher MDA levels compared to group C. Correspondingly, SOD activity significantly decreased in arsenic-treated rats. These findings agreed with the established mechanism of arsenic-induced ROS generation, which leads to lipid peroxidation and

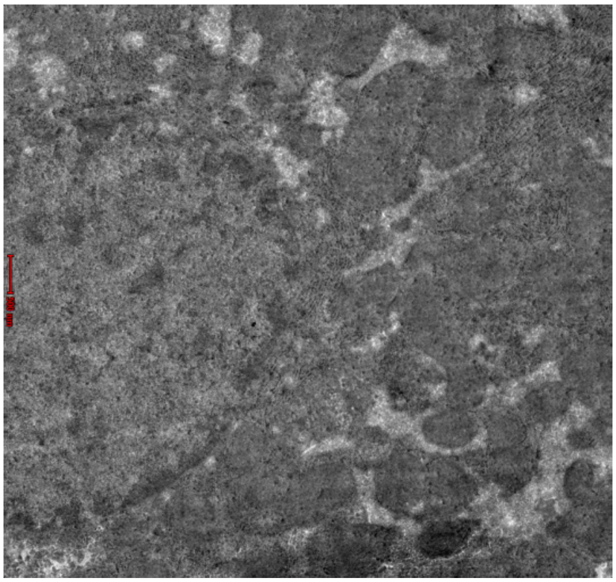

Fig. 5: Ultrastructure of liver showing electron dens round mitochondria and moderately dilated spaces, Nucleus indistinct nuclear membrane, dilated nuclear pores with Few remnants of RER at left top corner. (Group B)

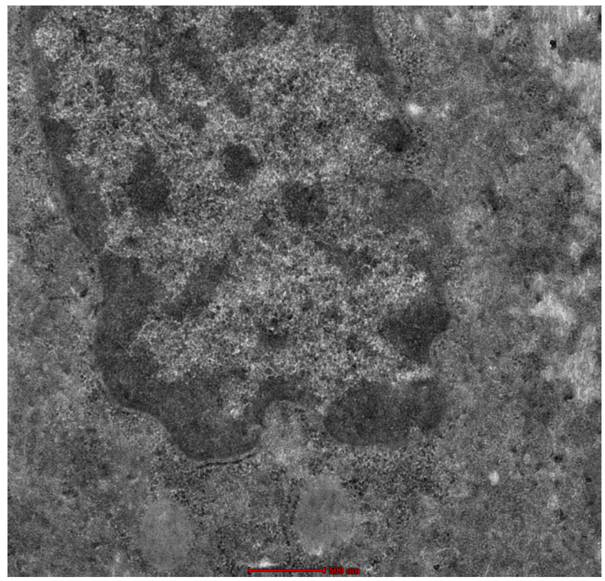

Fig. 6: Ultrastructure of liver showing pyknotic nucleus, indistinct nuclear membrane, margination of chromatin and irregular indentation of nuclear membrane. (Group B)

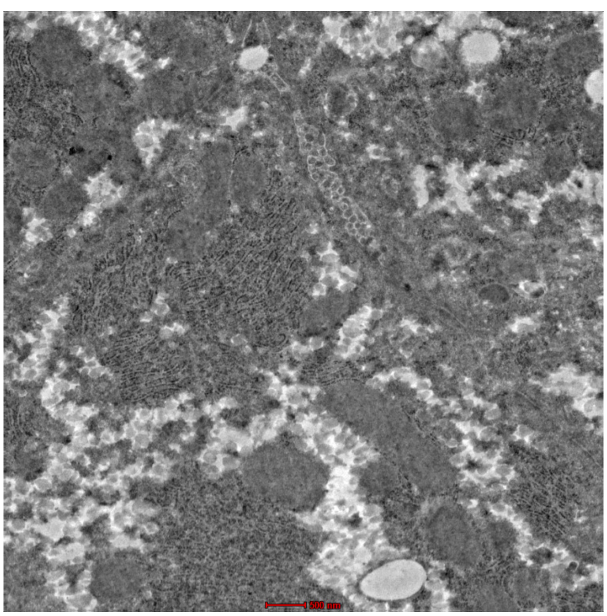

Fig. 7: Ultrastructure of liver showing restoration of RER with few aggregates of electron dense bodies. (Group C)

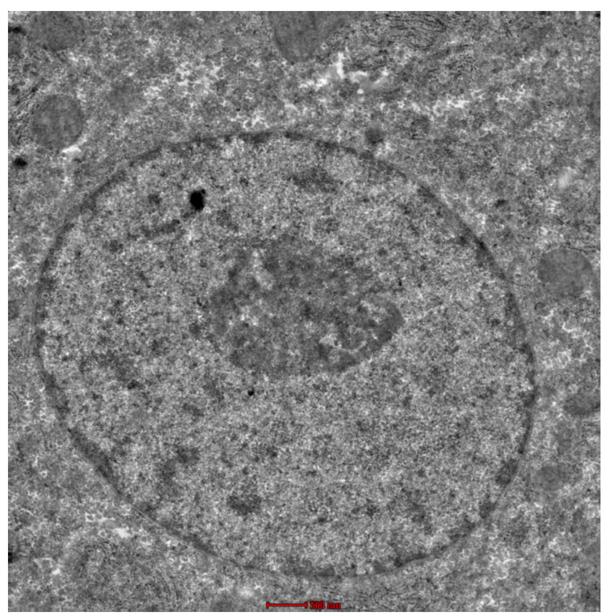

Fig. 8: Ultrastructure of liver showing normal nucleus with nuclear membranes, centrally placed distinct nucleolus and round mitochondria with mild evidence of matrix and crystae and gyrae. (Group C)

enzyme inhibition (Sharma *et al.,* 2009; Singh *et al.,* 2013). Group C demonstrated improved antioxidant status, with SOD values comparable to controls, reflecting the potent antioxidative property of *W. somnifera*, also reported by earlier researchers (Sharma *et al.,* 2009; Chattopadhyay *et al.,* 2011; Forkan *et al.,* 2016). Moreover, group D (WS) showed highest SOD levels compared to all other groups.

Gross and Histopathological Findings

No gross and histopatholgical changes were observed in Group A and D. Gross pathological changes in arsenic-treated rats (Group B) included congestion, enlargement, edema, and haemorrhage in multiple organs. These lesions agreed with previous findings in arsenic toxicity studies (Satapathy *et al.,* 2021). Histopathologically, brain of Group B exhibited vacuolation, microgliosis, dilated capillaries, and neuronophagia (Fig. 1). Group C showed only mild vascular congestion, confirming a neuroprotective effect. Liver of Group B showed portal vein congestion, edema, ballooning degeneration, neutrophil infiltration, pyknotic nuclei, fatty change, and coagulative necrosis (Fig. 2). Group C exhibited only mild degenerative changes, aligning with prior reports showing oxidative stress as a key driver of arsenic hepatotoxicity (Sharma *et al.,* 2009; Chattopadhyay *et al.,* 2011; Forkan *et al.,* 2016).

Stomach and intestine of Group B rats showed severe congestion, mucosal hyperplasia, leucocyte infiltration, villous damage, and mucosal necrosis (Fig. 3), while rats of Group C exhibited mild lesions, similar to previous observations by Biswas *et al.* (2000). Kidney of Group B displayed constricted glomeruli, swollen tubules, haemorrhages, tubular necrosis, eosinophilic casts, and sloughing of tubular cells (Fig. 4). Group C showed only moderate swelling and mild necrosis. These findings match reports linking arsenic to increased glomerular permeability and tubular degeneration (Forkan *et al.,* 2016).

Severe degenerative and inflammatory lesions were seen in Group B across organs (Heart, Lungs, Spleen, Testis), similar to earlier findings (Biswas *et al.,* 2000; Forkan *et al.,* 2016). Group C consistently showed milder pathology. Overall, *W. somnifera* provided 19.49% histopathological and 16.95% gross protective effect.

Ultrastructural Changes

Transmission electron microscopy of Group B (arsenic-treated) rats revealed indistinct nuclear membrane, dilated nuclear pores, granular nucleoplasm, electron-dense mitochondria, disrupted RER, pyknotic nuclei, chromatin margination, and swollen nuclei (Fig. 5, 6). Similar ultrastructural damage has been reported by Fowler *et al.* (1977).

Ultrastructure of Group C rats (arsenic + *W. somnifera*) showed largely restored nuclear membranes, normal nucleoli, reorganization of RER, and partial improvement in mitochondrial structure (Fig. 7, 8). Some areas exhibited mild apoptotic changes and fat body accumulation, but overall injury was significantly reduced, confirming the ameliorative effect of *W. somnifera*.

Conclusion

The finding of the present study revealed that the sub-acute arsenic exposure of Wistar rats @ 4 mg/kg b. wt. for 45 days produced multisystem toxicity marked by haematological, biochemical, oxidative, histopathological, and ultrastructural alterations. Simultaneous supplementation of *Withania somnifera* root extract (200 mg/kg b.wt.) along with arsenite significantly mitigated these changes by enhancing antioxidant defenses, reducing oxidative stress, and protecting tissues from structural and functional damage. The findings in control healthy group and that received *Withania somnifera* root extract (200 mg/kg b.wt.) alone did not show major alterations in the parameters studied.

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