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Assessment of the Renoprotective Potential of Melatonin Against Sodium Arsenite-Induced Nephrotoxicity in Male Rabbits: A Morphological and Biochemical Study

Ferdows. A. Alokshy[1]* & Fayrouz. A. Khaled[2]

[1]*Chemistry Department, Faculty of Science, AljufraUniversity, Hoon-Libya*

[2]Chemistry Department, Faculty of Science, Omar Al-Mokhtar University, El -Beida-Libya.

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Abstract:

Background: Chronic exposure to arsenic is a global health concern, frequently leading to severe organ damage, particularly nephrotoxicity. This study aimed to evaluate the potential renoprotective effects of Melatonin (Me) against Sodium Arsenite (Sa)-induced renal injury in male rabbits over a 12-week period. **Methods:** Rabbits were divided into four groups: Control (Con), Melatonin-only (Me), Sodium Arsenite (Sa), and a combination group (Me + Sa). Evaluations included gross morphological analysis of renal tissue and measurement of serum biochemical markers, specifically Urea and Creatinine levels. **Results:** Morphological analysis revealed that the Sa group suffered profound pathological changes, including significant renal atrophy, discoloration, and irregular surface textures, consistent with ROS-driven oxidative stress and tubular necrosis. In contrast, the Me + Sa group showed a remarkable recovery, with kidneys maintaining a structure and color similar to the control group. Biochemical data supported these findings: the Sa group exhibited significantly elevated levels of Serum Urea (47.88 ±1.395 mg/dl) and Creatinine (1.22 ±0.095 mg/dl) compared to the control ($p < 0.05$). Co-administration of Melatonin in the Me + Sa group significantly attenuated these elevations, bringing Urea (41.72 ±2.012 mg/dl) and Creatinine (0.87 ±0.076 mg/dl) levels back toward baseline values. Notably, Melatonin alone showed no adverse effects and even slightly improved renal markers compared to the control. **Conclusion:** The results demonstrate that chronic Sodium Arsenite exposure induces significant nephrotoxicity and structural degradation. However, Melatonin effectively mitigates these damages, likely due to its potent antioxidant properties. This suggests that Melatonin may serve as a promising therapeutic agent in preventing arsenic-induced renal dysfunction.

Keywords: Melatonin, Sodium Arsenite, Nephrotoxicity, Renoprotective Effect, Oxidative Stress, Renal Biochemical Markers.

Introduction

Arsenic contamination of groundwater is a major global public health crisis, affecting millions of people across various geographical regions[1]. Among its different forms, Sodium Arsenite (Sa) is recognized as one of the most toxic inorganic arsenic compounds. Chronic exposure to Sa occurs primarily through contaminated drinking water and industrial runoff, leading to multi-organ failure[2]. The kidney is a primary target of arsenic toxicity due to its role in the filtration and excretion of heavy metals, which results in the accumulation of arsenic within the renal cortex and tubular epithelium [3]. The pathogenesis of arsenic-induced nephrotoxicity is primarily driven by the induction of oxidative stress. Sodium Arsenite promotes the excessive generation of Reactive Oxygen Species (ROS), such as superoxide anions and hydroxyl radicals[4]. This surge in ROS overwhelms the endogenous antioxidant defense system, leading to lipid peroxidation of cellular membranes, protein denaturation, and DNA damage. These molecular disruptions eventually manifest as pathological morphological changes, including tubular necrosis, atrophy, and significant impairment of renal function, characterized by elevated serum levels of urea and creatinine [5]. In recent years, research has shifted toward identifying potent antioxidants that can mitigate heavy metal-induced damage [6-12]. Melatonin (Me), a hormone secreted by the pineal gland, has emerged as a powerful candidate. Beyond its role in regulating circadian rhythms, Melatonin is a highly effective broad-spectrum antioxidant and free radical scavenger. Its amphiphilic nature allows it to cross all biological membranes, protecting intracellular components from oxidative damage. Furthermore, Melatonin stimulates the activity of several antioxidant enzymes, such as superoxide dismutase and glutathione peroxidase, thereby enhancing the body's natural defense against toxic insults [13]. While the toxic effects of arsenic are well-documented, there is a growing need to evaluate the efficacy of Melatonin in recovering structural and functional renal integrity following chronic exposure[14]. Therefore, this study was designed to investigate the renoprotective potential of Melatonin against Sodium Arsenite-induced toxicity in male rabbits, specifically focusing on gross morphological alterations and biochemical markers of kidney function, including serum urea and creatinine levels.

***Corresponding Author**
Ferdows. A. Alokshy*
Chemistry Department, Faculty of Science, AljufraUniversity, Hoon-Libya.
Correspondence: aferdows872@gmail.com

Materials and Methods

Adult male rabbits (approximate weight 1.5–2.0 kg) were obtained for this study. The animals were housed in a controlled environment with a 12-hour light/dark cycle and provided with a standard laboratory diet and water *ad libitum*. All animals were acclimatized for one week prior to the commencement of the experiment to ensure physiological stability. Sodium Arsenite ($NaAsO_2$): Obtained in high purity and dissolved in distilled water for administration. Melatonin: Purchased from specialized chemical suppliers, prepared according to the required dosage. Biochemical Kits: Commercial diagnostic kits were used for the quantitative determination of Serum Urea and Creatinine. The rabbits were randomly assigned into four experimental groups (n = 5per group) for a duration of 12 weeks: Control Group (Con): Received a standard diet and distilled water without any treatment. Melatonin Group (Me): Administered Melatonin alone to evaluate its safety and baseline antioxidant effects. Sodium Arsenite Group (Sa): Administered Sodium Arsenite to induce chronic nephrotoxicity. Combined Group (Me + Sa): Co-administered Melatonin and Sodium Arsenite to evaluate the renoprotective potential of Melatonin. At the end of the 12-week experimental period, blood samples were collected from the marginal ear vein of each rabbit. The blood was allowed to clot at room temperature and then centrifuged at 3000 rpm for 15 minutes to separate the serum. Serum samples were stored at 20^C until biochemical analysis. After blood collection, the rabbits were humanely sacrificed. The kidneys were immediately excised, weighed, and examined for gross morphological changes (color, texture, and size). For histopathological analysis: Kidney tissues were fixed in 10% neutral buffered formalin. Tissues were dehydrated, cleared, and embedded in paraffin wax. Sections of 5 $m thickness were cut and stained with Hematoxylin and Eosin (H&E). The slides were examined under a light microscope to assess structural damage, such as tubular necrosis and hyperchromatic nuclei. The functional integrity of the kidneys was assessed by measuring: Serum Urea: Determined using the urease-enzymatic method. Serum Creatinine: Determined using the Jaffe reaction (colorimetric method). Data were expressed as Mean ± Standard Error (SE). Statistical significance was determined using one-way analysis of variance (ANOVA) followed by appropriate post-hoc tests to compare differences between groups. A p-value of less than 0.05 ($p < 0.05$) was considered statistically significant.

Results

Morphological Analysis of Renal Tissue

Control Group (Con): The renal specimens in the control group exhibit a normal physiological morphology. The kidneys maintain a characteristic mahogany color, a smooth capsular surface, and a healthy bean-shaped structure, indicating intact homeostatic conditions and the absence of oxidative stress. Melatonin Group (Me): The kidneys in the Melatonin-only group appear identical to the control group. This confirms that Melatonin, at the administered dosage, is biocompatible and does not induce any adverse morphological alterations, serving primarily as an antioxidant baseline. Sodium Arsenite Group (Sa): There is a profound pathological transformation in the Sa-treated group. The specimens show significant atrophy, discoloration (paleness), and irregular surface texture. These changes are indicative of chronic arsenic toxicity, likely driven by: Oxidative Stress: Generation of Reactive Oxygen Species (ROS) leading to lipid peroxidation of renal membranes. Nephrotoxicity: Induced apoptosis and necrosis of the tubular epithelium, resulting in the visible shrinkage and structural degradation observed. Combined Group (Me + Sa): The "Me + Sa" group demonstrates a significant morphological recovery compared to the Sa group. The kidneys appear fuller, with a color and texture much closer to the control group. This suggests a powerful renoprotective effect of Melatonin.

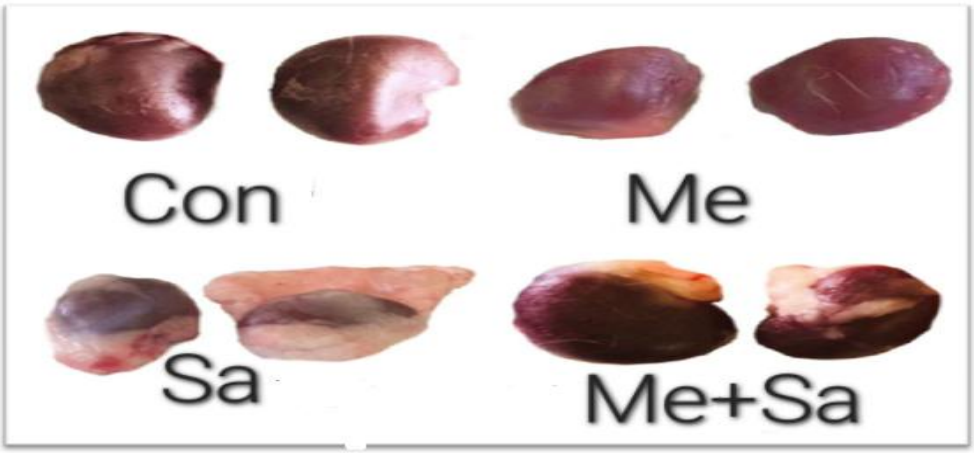

Figure 1: Representative photographs of the gross morphological changes in rabbit kidneys following 12 weeks of treatment with Sodium Arsenite (Sa), Melatonin (Me), or their combination.

Renal Toxicity Induced by Sodium Arsenite and the Protective Role of Melatonin in Male Rabbits

The data in Table 1 demonstrate that sodium arsenite (Sa) induced a marked nephrotoxic effect in male rabbits, as evidenced by a significant elevation in serum urea and creatinine levels compared with the control group ($p < 0.05$). These findings indicate impaired renal filtration and reduced glomerular function following chronic arsenic exposure. In contrast, melatonin (Me) administration alone significantly reduced serum urea and maintained creatinine levels near the control values, reflecting its potential renoprotective and antioxidant properties. Notably, co-administration of melatonin with sodium arsenite (Me+Sa) significantly attenuated the arsenic-induced increases in urea and creatinine, suggesting a protective modulatory effect of melatonin against arsenic-mediated renal dysfunction. Regarding kidney weight, no statistically significant differences were observed among the experimental groups, indicating that the functional and biochemical alterations in renal markers occurred prior to or independently of gross morphological changes. The relative kidney weight data (Figure 2) further support this observation, showing minimal variation between groups at the end of the 12-week exposure period. The time-course analysis of serum urea (Figure 3) revealed a progressive increase in the Sa-treated group over the experimental duration, reflecting cumulative renal impairment with prolonged arsenic exposure. Conversely, melatonin treatment moderated this rise when co-administered with sodium arsenite, maintaining urea levels closer to physiological ranges. A similar trend was observed for serum creatinine (Figure 4), where chronic arsenic exposure resulted in sustained elevations over time, while melatonin co-treatment significantly reduced creatinine accumulation, indicating preservation of renal excretory capacity. Overall, these results provide strong biochemical evidence that melatonin exerts a protective effect against arsenic-induced nephrotoxicity, likely mediated through its well-documented antioxidant, anti-inflammatory, and mitochondrial-protective mechanisms.

Table 1: Effect of Sodium Arsenite, Melatonin, and their combination on kidney weight and serum biochemical markers (Urea and Creatinine) in male rabbits.

Enzyme	***Experimental groups***			
	CON	**Me**	**Sa**	**Me+Sa**
Kidney(g)	11.374 ± 0.868^{a}	13.284 ± 0.647^{a}	11.994 ± 1.144^{a}	11.578 ± 0.914^{a}
Urea (mg/dl)	40.73 ± 0.916^{b}	33.56 ± 1.514^{c}	47.88 ± 1.395^{a}	41.72 ± 2.012^{bc}
Creatinine (mg/dl)	0.71 ± 0.021^{bc}	0.054 ± 0.033^{c}	1.22 ± 0.095^{a}	0.87 ± 0.076^{b}

Data are expressed as mean ± SE of 5 rabbit. Within each row, means with different superscript (a, b, c or d) were significantly different at $p<0.05$. Where means superscripts with the same letters mean that there is no significant difference ($p>0.05$).

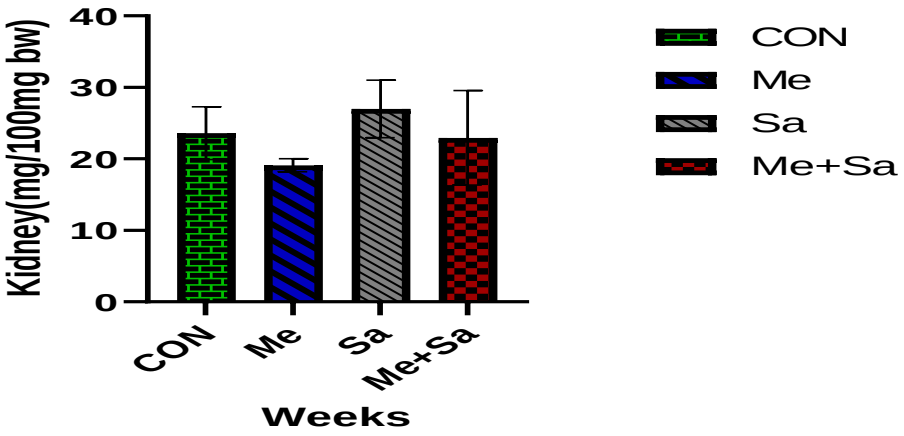

Figure 2: Relative kidney weight changes (mg/100mg bw) across experimental groups at the end of the 12-week period.

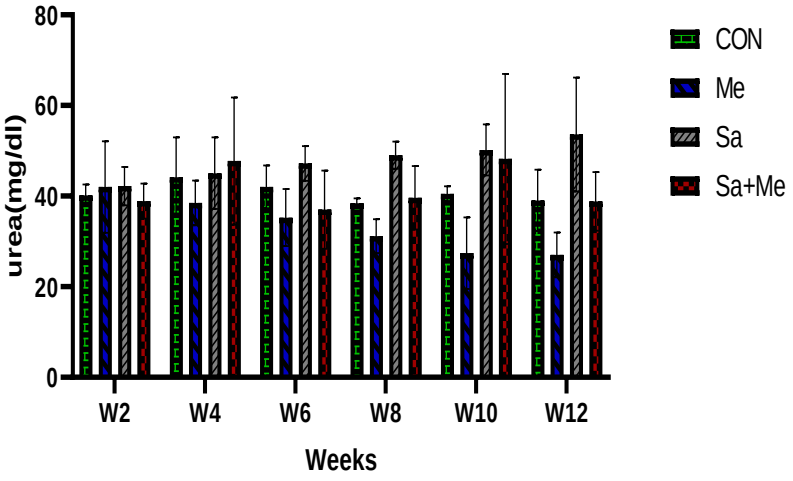

Figure 3: Time-course effect of Sodium Arsenite and Melatonin on Serum Urea levels in male rabbits over 12 weeks.

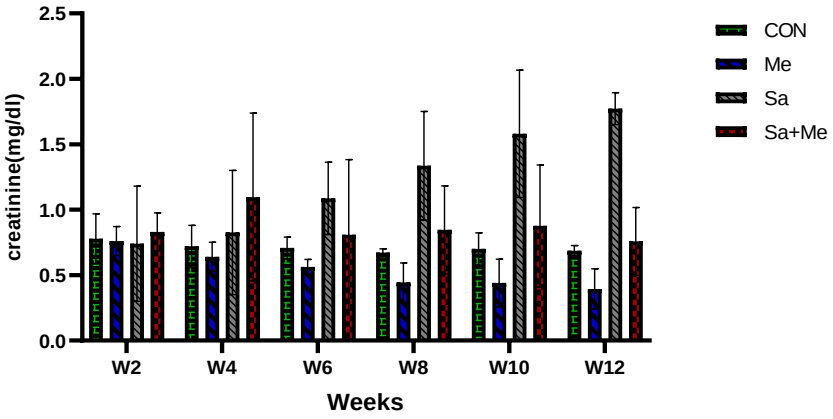

Figure 4: Impact of chronic Arsenic exposure and Melatonin co-administration on Serum Creatinine concentrations.

Histopathological Results Commentary (Kidney Tissue):

The histological examination of renal tissue (Figure 4) clearly demonstrates that chronic exposure to sodium arsenite induced pronounced structural damage in the kidney, particularly affecting the proximal convoluted tubules. The arsenic-treated group exhibited marked tubular epithelial disorganization, cellular crowding with hyperchromatic nuclei, increased abnormal mitotic figures, tubular lumen distortion, and evident desquamation of epithelial cells, all of which are characteristic features of toxicant-induced nephrotoxicity and tubular degeneration. These pathological alterations reflect severe cellular stress, impaired tubular integrity, and disrupted regenerative balance within renal tissue. In contrast, renal sections obtained from rabbits treated with melatonin alone displayed largely preserved renal architecture with near-normal tubular morphology, indicating the absence of adverse structural effects and supporting the safety of melatonin at the administered dose. Importantly, co-administration of melatonin with sodium arsenite markedly ameliorated the arsenic-induced histopathological lesions, with renal tissue showing substantial preservation of tubular structure and reduced epithelial damage compared with the arsenic-only group. This protective effect is consistent with the biochemical improvements observed in renal function markers and highlights the role of melatonin in stabilizing cellular membranes, limiting oxidative damage, and modulating apoptosis and aberrant cell proliferation within renal tubules. Overall, the histopathological findings provide strong morphological evidence that melatonin confers significant protection against arsenic-induced renal injury, reinforcing its potential therapeutic value in mitigating heavy metal–associated nephrotoxicity.

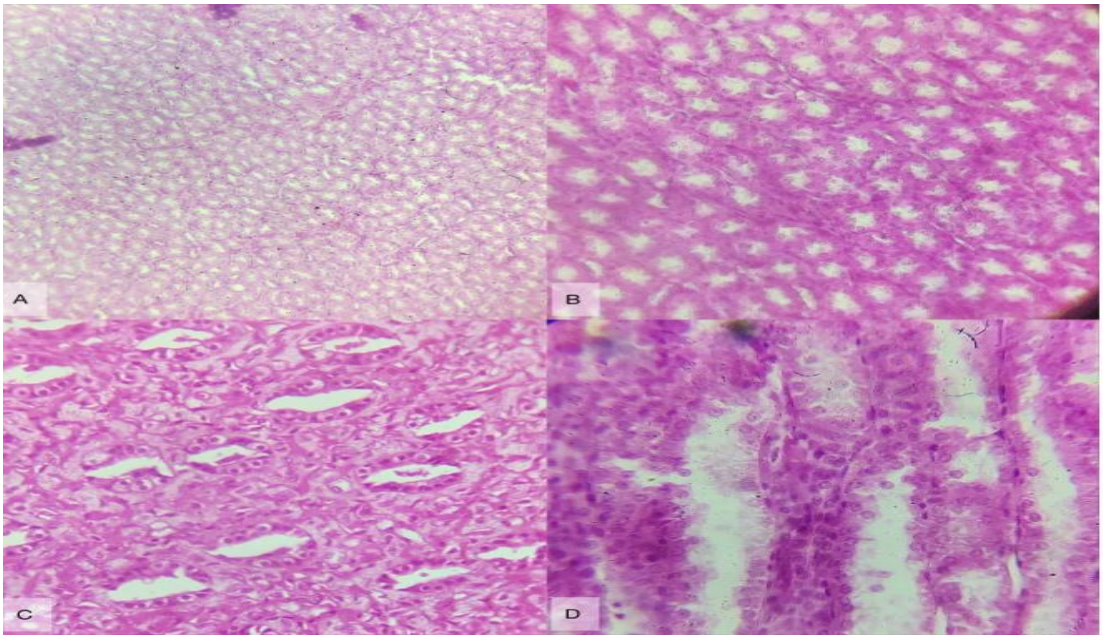

Figure4: Histopathological sections of rabbit renal tissue (H&E stain) illustrating the protective effect of Melatonin against Arsenite-induced structural damage.

Haematoxyline and Eosin stained kidney tissue. A. A slide taken from Melatonin group showing normal kidney tissue (magnification x20). B. A slide taken from Arsenic/Melatonin group showing normal kidney tissue (magnification x20). C. A cross sectioned slide taken from Arsenic group (magnification x40). D. A longitudinal-sectioned slide from Arsenic group (magnification x40). Both A and B showing abnormal proximal convoluted tubules (PCT) with crowded cells having hyperchromatic nuclei and showing many abnormal mitotic figures. The lumen of the of PCTs is not uniformly rounded and contains many desquamated cells.

Discussion

The chronic exposure to Sodium Arsenite (Sa) induces severe nephrotoxicity in male rabbits, characterized by a significant elevation in serum Urea and Creatinine levels, which indicates a marked decline in glomerular filtration and tubular function. This renal impairment is driven by Sa-induced oxidative stress and the excessive generation of reactive oxygen species (ROS), leading to the observed morphological atrophy, discoloration, and histopathological damage such as tubular necrosis and hyperchromatic nuclei [15-20]. However, the co-administration of Melatonin (Me) effectively mitigates these toxic effects, restoring biochemical markers toward baseline values and preserving the structural integrity of the renal tissue[21-30]. This renoprotection is attributed to Melatonin's dual role as a potent direct free radical scavenger and an indirect antioxidant that bolsters endogenous defense mechanisms [31-40]. Ultimately, the study concludes that Melatonin serves as a powerful protective agent against arsenic-induced structural and functional degradation, suggesting its potential therapeutic utility in preventing heavy metal-induced organ failure[40-43].

In conclusion, chronic sodium arsenite exposure induces significant nephrotoxicity and structural renal damage driven by oxidative stress. However, melatonin effectively mitigates these effects and restores kidney function, proving to be a potent renoprotective agent against arsenic-induced toxicity.

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