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Protective efficacy of ethanolic *Moringa oleifera* leaf extract against aluminum chloride-induced toxicity in male rats

H. T. Mahdi

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Correspondence Author
H. T. Mahdi
E-mail:
hayder.t@uokerbala.edu.iq

College of Veterinary Medicine, University of Kerbala,
Karbala, 56001, Iraq

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Aluminum (Al) is the third most abundant element in the Earth crust and constitutes about 8 % of the total mineral components. Aluminum chloride ($AlCl_3$) is widely spread in the environment and has no known biological function. Chronic exposure to aluminum chloride ($AlCl_3$) induces systemic toxicity, primarily through oxidative stress, neuroinflammation, hepatotoxicity, and dyslipidemia. Conversely, ethanolic leaf extract of *Moringa oleifera* (MOE), which is also characterized by a high concentration of polyphenols and flavonoid, has proven to possess effective antioxidant properties and is a promising systemic protector. The current study is aimed at determining the protective effect of the therapeutic dose of ethanolic Moringa extract against the toxicity caused by aluminum chloride ($AlCl_3$) in albino male rats (*Rattus rattus*). Forty albino males were divided into four groups and put through a successive 28 days exposure regimen. Orally gavage fed with aluminum chloride at the level of 100 mg/kg/body weight induced toxicity. Ethanolic Moringa extract at therapeutic dose of 300 mg/kg/body weight was applied on the protection group 1 hour following the administration of aluminum chloride. The main indicators were serum hepatic enzymes (ALT, AST, ALP), lipid status (TC, TG, LDL, HDL), oxidative-stress-responses (SOD, NO, MDA) and renal functioning (urea, creatinine). Aluminum chloride was given and prior to this there was notable hepatic enzyme activity (ALT, AST, and ALP) and dyslipidemia, manifested by increased TC, TG, and LDL, and by an equivalent decrease in HDL. Moringa extract (300 mg/kg) was found to greatly reduce these systemic toxic manifestations. In cerebral tissue, the extract provided neuroprotection through increasing antioxidant capacity – as indicated by higher levels of SOD activity and by eliminating nitrative stress. The ethanolic extract of *Moringa oleifera* leaves gives extensive and powerful protection against both systemic and neurotoxic effects caused by 100 mg/kg of aluminum chloride when administered at a 300 mg/kg dose. This protective effect can be primarily explained by its high antioxidant activity and by its capability to regulate the action of inflammatory and lipid-regulating activities, which highlights its possible use as a prophylaxis in nutrition.

Keywords: *Moringa oleifera*, aluminum chloride, hepatotoxicity, nephrotoxicity, antioxidant enzymes, oxidative stress, lipid profile.

Протективні властивості спиртового екстракту листя *Moringa olifera* за умови експериментального токсикозу щурів хлоридом алюмінію

Х. Т. Махді

Коледж ветеринарної медицини, Університет Кербела,
провінція Кербела, Ірак

Алюміній (Al) є третім за поширеністю елементом у земній корі та становить близько 8 % від загальної кількості мінеральних компонентів. Хлорид алюмінію ($AlCl_3$) є достатньо поширеним у навколишньому середовищі. Одночасно цей елемент не виконує в живому організмі ніяких біологічно-важливих функцій. Тривалий вплив хлориду алюмінію викликає системну токсичність, в першу чергу, через появу: оксидативного стресу, хронічних запальних процесів у нервовій системі, гепатотоксичності та порушення обміну ліпідів. Поряд з цим, спиртовий екстракт листя *Moringa oleifera*, який у своєму складі характеризується високою концентрацією поліфенолів і флавоноїдів, має ефективні антиоксидантні властивості і є перспективним системним протектором. Поточне дослідження спрямоване на визначення протективного ефекту терапевтичної дози спиртового екстракту листя *M. oleifera* за умови експериментального токсикозу щурів, викликаного хлоридом алюмінію. Дослідження проводилися на білих лабораторних щурах, з яких за принципом аналогів було сформовано 4 групи (1 контрольна та 3 дослідних). Моніторинг протективних властивостей спиртового екстракту листя *M. oleifera* здійснювали за показниками активності печінкових ферментів, ліпідного профілю, за оцінкою показників оксидативного стресу та функціонуванням нирок. Визначено, що введення хлориду алюмінію щурам третьої дослідної групи призводило до вірогідного ($P<0,05$) підвищення активності печінкових ферментів (АсАТ, АлАТ та ЛФ), концентрації загального холестерину, тригліцеридів, ліпопротеїдів низької щільності на фоні зниження ($P<0,05$) ліпопротеїдів високої щільності. Застосування щурам третьої дослідної групи $AlCl_3$ у поєднанні з екстрактом листя *M. oleifera* знижувало його токсичний вплив. Зафіксовано вірогідне зниження ($P<0,05$) активності АсАТ, АлАТ, ЛФ, вмісту загального холестерину, тригліцеридів, ліпопротеїдів низької щільності на фоні підвищення ($P<0,05$) вмісту ліпопротеїдів високої щільності. Застосування *M. oleifera* проявляло нейропротективні властивості, про що свідчили вищі ($P<0,05$) рівні активності СОД на фоні значного зниження ($P<0,05$) оксиду азоту (NO) порівняно з аналогічними показниками у тварин третьої дослідної групи. Спиртовий екстракт листя *M. oleifera* забезпечує потужний та широкий захист від системних та нейротоксичних ефектів, викликаних дією на організм $AlCl_3$, що вказує на можливість його використання як профілактичної харчової добавки.

Ключові слова: *Moringa oleifera*, хлорид алюмінію, гепатотоксичність, нефротоксичність, антиоксидантні ферменти, оксидативний стрес, ліпідний профіль.

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Introduction

The third most abundant element in the Earth crust is aluminum (Al3+), which is widely used in packaging food, purification of water, pharmaceutical formulas, and kitchenwares [1]. Even though it is widespread, aluminum has no physiological role in biological systems and is classified as a non-essential element [2]. The long-term effects of aluminum are associated with a continuum of health effects, primarily due to neurotoxicity in the form of cognitive impairment and neurodegenerative diseases, including Alzheimer's disease [3].

The neurotoxic effects of aluminum are mediated through multiple mechanisms, including the generation of reactive oxygen species (ROS), induction of neuro-inflammation, promotion of amyloid-beta aggregation, and hyperphosphorylation of tau proteins [4]. After cellular absorption, aluminum is able to cross the blood brain barrier, accumulate in neural tissue and cause neuronal damage and apoptosis [5]. Besides the central nervous system, systemic toxicity as evidenced by hepatic dysfunction (significant increase in blood serum of ALT, AST, and ALP), Renal dysfunction is manifested by increased serum urea and creatinine levels and dyslipidemia (increased total cholesterol (TC), triglycerides (TG), low-density lipoprotein (LDL), and a decrease in high-density lipoprotein (HDL)) as a result of aluminum exposure [6].

Moringa oleifera, also known as the drumstick or horseradish tree is native to India though it grows in various areas of tropical and subtropical regions [7]. All the components of the plant such as leaves, seeds and pods have been used in traditional medicine because of the nutritional and medicinal properties [8]. Leaves, especially, are a good source of vitamins A, C and E, a vast array of phytochemicals including polyphenols (such as quercetin, kaempferol), flavonoids (e.g., rutin), and glucosinolates, thus giving them strong antioxidative and anti-inflammatory effects [9].

Mechanisms of aluminum-induced toxicity. Exposure to aluminum is observed during the intake of contaminated food, water, antacids and leaching through cooking ware [10]. Aluminum that is ingested is transported to critical body organs like the brain, liver, and bones where it causes toxicity along various mechanistic pathways [11]. The primary action is the production of reactive oxygen species (ROS), which inactivate natural antioxidants, such as superoxide dismutase, and glutathione, and increase lipid peroxidation, oxidation of proteins, and damage DNA [12]. The final result of this oxidative condition is the stimulation of microglia and astrocytes and the release of pro-inflammatory cytokines, including tumor necrosis factor-alpha (TNF-alpha) and interleukin-6 (IL-6) [13]. Also, aluminum enhances amyloid-beta deposition and tau protein hyperphosphorylation, characteristic of the Alzheimer disease [14]. Memory deficits are aggravated due to cholinergic transmission disruption by the increased acetylcholinesterase (AChE) activity [15]. Hepatocyte deposition of aluminum leads to liver injury in the form of elevated serum alanine aminotransferase (ALT), aspartate aminotransferase (AST) and alkaline phosphatase (ALP) activities, systemically. Lipid metabolism is also impaired due to aluminum, increasing serum levels of TC, TG, and LDL and decreasing HDL levels [16, 17].

Therapeutic potential of Moringa oleifera. Leaves of *Moringa oleifera* are an important source of nutrition and contain high bioactive molecules. Rich in vitamins C, E, and A, they act like direct scavengers for free radicals [18]. Polyphenolic compounds like chlorogenic acid, quercetin, gallic acid, and flavonols like isoquercetin and rutin constitute the primary basis for *Moringa oleifera* antioxidant properties, supported by in vitro experiments demonstrating radical scavenger activity versus DPPH and ABTS similar to model antioxidants [19]. Experiments on animal models suggest that water extracts of *Moringa oleifera* increase the levels of glutathione (GSH) and total antioxidant capacity (TAC) in multiple tissues [20]. *Moringa oleifera* anti-inflammatory effect is evidenced by suppression of TNF-α and IL-6 generation in LPS-stimulated macrophages, most likely through the modulation of NF-κB and MAPK signaling pathways. These features render *Moringa oleifera* a promising means to counteract aluminum-induced oxidative and inflammatory damage. Preclinical testing evidence of *Moringa oleifera* extract (MOE) protection against aluminum toxicity [21].

Moringa oleifera protective effect against $AlCl_3$-induced toxicity. Ekong et al. (2017) exposed Wistar rats to $AlCl_3$ (100 mg/kg of body weight) for 28 days, and MOE (300 mg/kg of body weight) was given an hour after $AlCl_3$. MOE treatment decreased temporal cortical neuronal degeneration significantly, enhanced superoxide dismutase (SOD) activity, and lowered cholesterol peroxidation in brain homogenates [22]. Gouda et al. (2018) extended the duration of exposure to 60 days and found that MOE corrected $AlCl_3$ evoked cardiotoxicity, restoring cardiac histopathology as well as antioxidant markers [15]. Hindawy et al. (2024) conducted a comprehensive 28-day research in chronic Wistar rats and validated that MOE (300 mg/kg of body weight) co-treatment with $AlCl_3$ (100 mg/kg of body weight) reinstated cerebral SOD and nitric oxide (NO) homeostasis, suppressed TNF-α and IL-6 expression, and preserved cortical neuronal structure, as evidenced through hematoxylin and eosin staining [20].

The aim of the study

The current study aims to determine the protective effect of a therapeutic dose of ethanolic Moringa extract against the toxicity caused by aluminum chloride ($AlCl_3$) in albino male rats (*Rattus rattus*).

Materials and methods

Preparation of the ethanolic extraction of Moringa oleifera

Leaves were washed well with distilled water to eliminate surface contaminants and dust, and air-dried in a well-flowing shaded area at room temperature (25±2°C) for 21 days to avoid photodecomposition of bioactive molecules [24]. After complete drying, the dried leaves were milled into a fine, uniform powder using a

high-speed hammer mill and sieved on a 0.5 mm mesh screen to ensure uniform particle size. The powder was stored in amber glass bottles sealed against oxidation and microbial contamination at 4°C until extraction. For extraction of ethanolic, dry leaf powder (100 g) was macerated in 1,000 ml of 100 % pure ethanol (Sigma-Aldrich, Germany) in a glass jar covered with a lid. The mixture was shaken at room temperature (25°C) every day for 72 hours to facilitate the dissolution of polar and semi-polar phytochemicals [25].

Animals of the study

This research was conducted at the University of Kerbala, College of Veterinary Medicine, in the animal house of the Physiology Department. A total of forty male albino rats (*Rattus rattus*) of mean weight 200±20 g and aged from 8 to 10 weeks were used. The animals were kept for two weeks before the experiment for acclimatization. Ten animals were kept in each of the 15 x 35 x 50 cm individual plastic cages. Ad libitum feeding was provided with a standard pellet diet, and free access to water was given. The animals were kept under controlled temperature (22–25°C) and lighting (14L: 10D) conditions.

Experimental design

The experimental animals were divided into four groups, each consisting of 10 male rats as follows:

Group I (Control). Treated orally by gavage once daily with 0.5 ml of distilled water.

Group II (Moringa). Treated orally by gavage once daily with ethanolic *Moringa oleifera* extract in a dose of 300 mg/kg of body weight.

Group III ($AlCl_3$). Treated orally by gavage once daily with aluminum chloride ($AlCl_3 \cdot 6H_2O$) in a dose of 100 mg/kg of body weight.

Group IV (Moringa + $AlCl_3$). Treated orally by gavage with Moringa extract (300 mg/kg) and aluminum chloride (100 mg/kg) once a day.

All the treatments were given by oral gavage with a stainless-steel feeding needle for four weeks in succession. After the treatment period (28 days), the animals were kept overnight (12 hours) fasted with free water access. Blood was taken by cardiac puncture under light anesthesia, and the blood serum was separated and kept at -80°C till biochemical analysis.

Biochemical assays

Serum lipid profile

Kits were used to assess the serum levels of total cholesterol (TC), high-density lipoprotein (HDL), and triglycerides (TG) (Agappe Diagnostic LTD, Kerala, India). LDL was calculated according to Schettler & Nüssel, [26]. The atherogenic index (TC/HDL) and LDL/HDL ratio were also calculated (Leimieux et al., 2001) [27].

Liver enzymes

An aspartate aminotransferase test kit was used to measure AST activity in the serum (Agappe diagnostic, India code 683-562), An Alanine aminotransferase test kit was used to measure in serum (Agappe diagnostic, India, Clin. Chem, and an Alkaline phosphatase test kit was used to measure in serum (Agappe diagnostic, India).

Serum bio-oxidative markers:

- Malondialdehyde (MDA): It is easy to use the MDA Microplate Assay Kit to detect MDA in a range of samples. Thiobarbituric acid (TBA) reacts with MDA in the sample to form the MDA-TBA adduct. You may readily measure the MDA-TBA adduct using a colorimeter (λ= 532 nm). The results were expressed in nmol/ml.

- Superoxide dismutase (SOD): Microplate Assay Kit. Light with a wavelength of 560 nm or longer is absorbed by NBT-diformazan. SODs diminish the levels of superoxide ions, which in turn reduces the rate at which NBT-diformazan is formed. NBT-diformazan decrease is a good indicator of SOD activity in the experimental samples.

To assess the amount of NO, it is a usual practice to test the total NO2-/NO3- ratio. At 550 nm, a colorimetric readout may be used to determine the reaction products. According to Kavsaket et al. [28], the values were expressed in µmol/l.

Kidney function tests

Serum creatinine and urea levels were determined by the use of specialized kit (Agappe diagnostics, India).

- Creatinine: In order to detect the blood creatinine levels, the researchers used a specialized kit (Agappe diagnostics, India). When creatinine is combined with picrate in alkaline media, a colorful complex is formed. In order to estimate the quantity of creatinine in the specimen, a spectrophotometer may be used to measure the absorbance at 500 nm.

- Urea: The following reaction is used to determine urea through enzymatic means according to the Agappe diagnostic India kit protocol (Kassirer, 1971) [29].

Statistical analysis

The values were expressed as mean ± standard deviation (SD). One-way analysis of variance (ANOVA) was performed for the analysis of significant differences between the groups, and follow-up the least significant difference (LSD) post-hoc test for multiple comparisons using SPSS version 26 (IBM Corp., Armonk, NY, USA). The statistical significance was determined with a P-value < 0.05.

Results and discussion

The protective effect of the ethanolic extract of *Moringa oleifera* against aluminum chloride-induced toxicity is indicated through the various biochemical parameters. From the obtained results, it can be shown that aluminum chloride exposure initiated serious systemic disturbances, which were effectively reversed through the co-treatment with Moringa extract.

Effect of the treatments on liver enzyme activity

Table 1 shows the effect of different treatments on liver enzymes (AST, ALT, ALP). Group III (exposed to aluminum chloride only) showed a drastic and statistically significant rise ($P<0.05$) in the activity of all the enzymes compared to the control group.

Table 1
The effects of different treatments on liver enzyme activity in male rats, Mean ± SD, (n=10)

Groups	Parameters		
	AST (U/L)	ALT (U/L)	ALP (U/L)
Group I. (Control)	85.7±9.84 [c]	55.4±8.14 [c]	162.4±9.90 [c]
Group II. (Moringa oleifera)	78.3±8.52 [c]	48.1±6.17 [c]	158.5±10.72 [c]
Group III. (Aluminum)	170.6±11.64 [a]	110.9±12.15 [a]	261.7±13.73 [a]
Group IV. (Moringa + Aluminum)	110.4±10.47 [b]	75.7±6.23 [b]	187.8±11.5 [b]
LSD values	9.23	7.73	10.47

Note: means followed by different letters in the same column indicate a statistically significant difference (P<0.05) between the values.

AST level increased from 85.7 U/L for the control group to 170.6 U/L in the aluminum group (a rise of 99 %). Similarly, the level of ALT increased from 55.4 U/L to 110.9 U/L and the level of ALP increased from 162.4 U/L to 261.7 U/L. These increases depict the extent of the liver cell damage.

Conversely, the co-treatment with Moringa extract and aluminum chloride (Group IV) yielded a significant decrease (P<0.05) in the levels of these enzymes compared to those with the aluminum group alone. The value of AST decreased to 110.4 U/L, the value of ALT – to 75.7 U/L, and the value of ALP – to 187.8 U/L. The significant decrease is the proof of the hepato-protective effect of Moringa extract.

Group II (solely Moringa) had enzyme levels as in the control group, and this indicated the safety of the extract on the liver functioning at the given dose.

Effect of the treatments on kidney function indicators

The effect of the treatments on the kidney functioning markers (urea and creatinine) is shown in ***Table 2***. Aluminum chloride treatment (Group III) caused a significant (P<0.05) rise in serum urea and creatinine compared to the other treatments.

Table 2
The effects of different treatments on kidney functioning in male rats, Mean ± SD, (n=10)

Groups	Parameters	
	Urea (mg/dl)	Creatinine (mg/dl)
Group I. (Control)	17.85±1.23 [c]	0.54±0.02 [c]
Group II. (Moringa oleifera)	16.6±1.52 [c]	0.56±0.06 [c]
Group III. (Aluminum)	28.25±2.37 [a]	0.96±0.04 [a]
Group IV. (Moringa + Aluminum)	19.1±2.85 [b]	0.71±0.01 [b]
LSD values	2.93	0.21

Note: means followed by different letters in the same column indicate a statistically significant difference (P<0.05) between the values.

The urea level increased from 7.85 mg/dl in the control group to 28.25 mg/dl, and the creatinine level increased from 0.54 mg/dl to 0.96 mg/dl, showing a pathological effect on renal function.

Co-treatment group (Aluminum + Moringa) exhibited outstanding protection and healing (P<0.05), wherein urea and creatinine were reduced to 19.1 and 0.71 mg/dl, respectively. They are both far more similar to the indicators of the control group. This demonstrates the possibilities of Moringa extract to prevent the kidneys toxicity against aluminum.

Effect of the treatments on oxidative stress biomarkers

Table 3 shows the oxidative stress condition in the various experimental groups. Group III, or the aluminum group, had significantly increased (P<0.05) malondialdehyde (MDA), a marker for lipid peroxidation, with the level reading 10.8 nmol/ml, compared to the level of the control group, which was 4.1 nmol/ml. Also, the level of nitric oxide (NO) markedly increased to 15.7 µmol/mg, while superoxide dismutase (SOD) activity markedly dropped to 8.1 U/mg. In all, these data indicate an oxidative stress critical state.

Table 3
The effects of different treatments on oxidative activity in male rats, Mean ± SD, (n=10)

Groups	Parameters		
	NO (µmol/mg)	SOD (U/mg)	MDA (nmol/ml)
Group I. (Control)	5.2±0.5 [c]	15.2±1.80 [c]	4.1±0.6 [c]
Group II. (Moringa oleifera)	5.6±0.9 [c]	21.5±2.2 [a]	3.4±0.5 [c]
Group III. (Aluminum)	15.7±1.4 [a]	8.1±1.53 [d]	10.8±1.9 [a]
Group IV. (Moringa + Aluminum)	7.5±0.3 [b]	17.8±2.01 [b]	5.2±0.8 [b]
LSD values	1.37	1.64	0.84

Note: means followed by different letters in the same column indicate a statistically significant difference (P<0.05) between the values.

The Moringa extract treatment (Group IV) reversed these changes very effectively. MDA value dropped to 5.2 nmol/ml, and NO concentration was lowered to 7.5 µmol/mg. It is important that SOD enzyme activity was also restored impressively to 17.8 U/mg, nearing the control group level. Moreover, the Moringa-alone group revealed the highest level of SOD activity of 21.5 U/mg, confirming its strong antioxidant-protection activity.

Effect of the treatments on lipid profile markers

Table 4 shows the impact on the serum lipid profile markers. Aluminum chloride (Group 3) exposure caused severe dyslipidemia as seen by significant statistically significant increase (P<0.05) in total cholesterol (TC) to 180.6 mg/dl, triglycerides (TG) to 161.3 mg/dl, and low-density lipoprotein cholesterol (LDL-C) to 120.2 mg/dl. At the same time, there was a significant decrease in the high-density lipoprotein cholesterol (HDL-C), or the good cholesterol, which was 35.1 mg/dl.

Table 4

The effects of different treatments on lipid profile activity in male rats, Mean ± SD, (n=10)

Groups	Parameters			
	Total cholesterol (TC) (mg/dl)	Triglycerides (TG) (mg/dl)	LDL-C (mg/dl)	HDL-C (mg/dl)
Group I. (Control)	110.3±12.4[c]	85.4±10.1 [c]	45.4±7.1 [c]	55.2±6.3 [b]
Group II. (Moringa oleifera)	110.7±9.2 [c]	78.1±8.7 [c]	41.5±6.2 [c]	65.6±7.1 [a]
Group III. (Aluminum)	180.6±18.4 [a]	161.3±19.5 [a]	120.2±17.73 [a]	35.1±5.4 [c]
Group IV. (Moringa + Aluminum)	135.4±11.7 [b]	112.7±13.2 [b]	75.8±10.1[b]	54.6±6.8 [b]
LSD values	13.24	12.96	10.23	5.48

Note: means followed by different letters in the same column indicate a statistically significant difference (P<0.05) between the values.

These complications were greatly relieved by co-treatment with Moringa extract (Group 4). The TC, TG, and LDL-C concentrations decreased significantly, and the HDL-C was 54.6 mg/dl, which is not statistically different from the control. These findings highlight the ability of Moringa to regulate lipid metabolism and prevent lipidemia caused by aluminum. Especially a higher concentration of HDL-C was observed in the Moringa-only group (65.6 mg/dl), which indicates a possible cardiovascular effect.

The results of this study strongly confirm the multiple protective efficacy of the ethanolic extract of *Moringa oleifera* leaves against systemic neurotoxicity induced by aluminum chloride in rats. The multiple protective mechanism by which the extract demonstrates its action, including hepatoprotection, nephroprotection, oxidative stress modulation, and dyslipidemia regulation, makes it an effective means as a protective dietary supplement.

The present study has shown that the treatment with aluminum chloride leads to acute nephrotoxicity and hepatotoxicity, as evidenced by the significant rise in the liver enzymes (AST, ALT, ALP) and kidney function tests (urea and creatinine). These findings are consistent with previous observations that aluminum is accumulated in the liver and kidneys and leads to cell damage and the release of these markers into the circulatory system. The significant decrease of these markers in the Moringa + aluminum group indicates the extract's ability to maintain the integrity of hepatocyte and renal cell membranes. Moringa's high phenolic and flavonoid content, such as quercetin and kaempferol, may be accountable for this protection because of their potency as antioxidants and anti-inflammatory agents, which reduce cellular damage caused by aluminum-induced oxidative stress. This observation is in agreement with [23] on the normalization of serum liver enzymes in rats fed on Moringa extract after being treated with aluminum.

Aluminum toxicity involves oxidative stress mechanism. We found that aluminum induced severe complication of the oxidation state manifested by elevated lipid peroxidation (MDA), elevated nitrative stress (NO), and loss of endogenous antioxidant protection (SOD). The effectiveness of Moringa extract in normalizing the SOD activity to the high level and decreasing MDA and NO to lower levels is beyond doubt the evidence of its antioxidant power. SOD enzyme is one of the first lines of defense mechanism against free oxygen radicals and when its work is stimulated, the destructive mechanisms that cause cell damage are diminished. The findings agree with [22] in which it was found that Moringa extract replenished SOD activity and decreased lipid peroxidation in the exposed rats' brain tissue due to exposure to aluminum. Moreover, polyphenols, being active constituents in Moringa, are direct free radical scavengers, as well as they can regulate cellular signaling pathways, including the Nrf2/ARE pathway which is a regulator of antioxidant gene expression. Other studies attribute the reversal of the neuroinflammation being a crucial component of aluminum-induced neurodegenerative diseases to the action of Moringa in inhibiting t he synthesis of the inflammatory cytokines of TNF-alpha and IL-6.

Dyslipidemia caused by aluminum was considered in the current study, where there were significant increases in TC, TG and LDL-C and a decrease in HDL-C. The raised risk of cardiovascular disease is observed in this pattern of lipid disturbance. The ability of the Moringa extract to normalize these parameters, especially to raise the level of good cholesterol called HDL-C, signifies an added benefit on top of neuroprotection [30]. This is explained by the fact that Moringa is able to stimulate the liver, as it is one of the key organs when it comes to lipid metabolism. It is also possible that the compounds in Moringa affect the major enzymes involved in cholesterol synthesis and absorption. This confirms earlier experiments that have demonstrated that Moringa could potentially have hypolipidemic activity, preventing metabolic syndrome.

Conclusions

The current research has concluded that ethanolic extract of *Moringa oleifera* leaves at 300mg/kg of body weight is effective and provides general protection against systemic and neurotoxicity of aluminum chloride (100 mg/kg) in male albino rats. Its protective power lies in its superior antioxidant, anti-inflammatory and anti-apoptotic effect, which has been achieved by the preservation of liver functions, lipid balance and neuronal integrity. Though these findings place Moringa extract on the frontlines as a potential protective supplement, clinical studies are critical as far as referring these findings down to human health.

Conflict of interest

The author state that there is no conflict of interest.

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ORCID

H. T. Mahdi https://orcid.org/0000-0002-1288-8591