

The Protective Effect of Eggplant Fruits Against Neurotoxicity Induced by Aluminum Chloride in Experimental Rats

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Abstract

The current study was performed to evaluate the protective effect of eggplant fruits against neurotoxicity induced by Aluminum chloride in rats. Thirty-six adult male albino rats with "*Sprague Dawley*" strain weighing (150±10 g.) were used and split into two major groups, the first group (6 rats) fed on a basal diet and kept as a negative control.

The second group of 30 rats received 17 mg/kg b.w. from ALCL3 was dissolved in saline and given by gavage daily (for 28 days) and divided into five subgroups: Sup group (1): was fed on basal diet (and served as positive control). Sup group (2, 3): were fed on basal diet + (5&10%) white eggplant dried fruits. Sup group (4, 5): were fed on basal diet + (5&10%) of purple eggplant dried fruits for 28 days. Body weight gain (BWG), Feed intake (F.I.), feed efficiency ratio (FER), another relative brain weight were computed at the finish of the experiment. Assessment of brain tissues was analyzed for antioxidant/enzymes markers, biochemical analysis were done to determine a phenolic compound in white and purple

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eggplant fruits by LC-MS in addition to brain tissue , also histopathology of the brain was assessed. The results revealed that white and purple eggplant dried fruits improved the biological evaluation, brain functions, antioxidant enzyme activity, and brain histopathology compared to the positive group and the best result was for purple eggplant . In conclusion, administering white and purple eggplant dried fruits can lower the impact of aluminum chloride on the brain.

Keywords: white eggplant, purple eggplant, Aluminium chloride, brain functions, antioxidant enzymes.

Introduction

The brain is a large interactive processor capable of processing sensory inputs, storing information for short or long periods, and expressing the mental output by language, mimicry, or behavior(***Salleet al.,2006***).

Aluminum(Al) is the third most abundant element in the earth's crust. Various aluminum compounds are produced and used for different purposes, such as water treatment, papermaking, fire retardant, fillers, food additives, colors, and pharmaceuticals. Aluminum metal, mainly in the form of alloys with other metals, has many uses, including in consumer appliances, food packaging, and cookware(***European Food Safety Authority(EFSA), 2008***).

Al has shown neurotoxicity in patients undergoing dialysis and thereby chronically exposed parenterally to high aluminum concentrations. It has been suggested that Al is implicated in the etiology of Alzheimer's disease and associated with other neurodegenerative diseases in humans. However, these

hypotheses remain controversial. Based on the available scientific data, the Panel does not consider exposure to aluminum via food to constitute a risk for developing Alzheimer's disease (**EFSA, 2008**). Al has been shown to accumulate in all regions of the rat brain following chronic exposure, with maximum levels in the hippocampus, which is the site of memory and learning. Al contributes to dialysis encephalopathy, and Al-induced oxidative stress and neurotoxicity are considered to be pathological factors for Alzheimer's disease. Various studies have indicated neurochemical, neuropathological, and neurobehavioral changes following Al exposure (**Kumar and Gill, 2014**). Its routine use has resulted in excessive human exposure and, due to the potential neurotoxic effects, has attained a huge interest in recent years (**Dey and Singh, 2022**). Al is a metal that promotes oxidative damage leading to neuronal death in different brain regions with behavior, cognition, and memory deficits. (**Camposet al., 2022**).

Polyphenols have important biological activities; they are powerful anti-oxidants that inhibit the production of free radicals, thus limiting the risk of developing oxidative stress-induced degenerative disorders such as ischemia, Parkinson's disease, or Alzheimer's disease (**Basli et al .,2012**).several mechanisms, underlying the potency of polyphenols to improve neurological health, including their interaction with neuronal and glial signaling pathways, decreasing neurotoxins-mediated neural damage and loss or neuroinflammation, diminishing reactive oxygen species (ROS) production, and attenuating the accumulation of neuropathological markers, such as amyloid-b (Ab) and Tau protein,providing new protective and therapeutic strategies for preventing or delaying neurocognitive impairment in brain disorders (**Bensalem et al., 2015**).

Eggplant fruits contain different classes of phenolic phytochemicals (flavonols, phenolic acids, and anthocyanins) that can benefit human health. The color, size, and shape of eggplant fruits vary with the cultivar type, and differences in phenolic profile have also been observed (**Singh et al., 2017**).

Acetylcholin (Ach) is a well-known animal neurotransmitter presented in eggplant (**Horiuchi et al., 2003**). Ach is the first substance proven to be a neurotransmitter and has an important role in enhancing sensory perception when we wake up and sustaining attention (**Jones, 2005**). The neurons synthesizing monoamines or acetylcholine reside in the brainstem and basal forebrain (**Cools and Arnsten, 2022**). Therefore, the goal of the current investigation was to assess the neuroprotective efficacy of purple and white eggplant on aluminum chloride-induced brain toxicity in experimental rats.

Materials and Methods

Materials

- 1- Eggplant fruits (*Solanum melongena L*) were obtained from a local supermarket in Tanta, Egypt. The study samples are fully equipped with the peel.
- 2- Corn oil, Wheat bran, Sucrose, and Corn starch were purchased from the local market. Casein, vitamins, minerals, Choline bitartrate, and L-cysteine were obtained from the Cairo Company for Chemical Trading, Cairo, Egypt.
- 3- Aluminum chloride anhydrous ($ALCL_3$) was supplied by Sigma chemical company.
- 4- Thirty-six male albino rats (*Sprague Dawley strain*) were obtained from the Laboratory Animal Colony, Helwan, Cairo – Egypt, weighing approximately 150 ± 10 g.

Methods

Preparation of the raw materials

Purple and white eggplants were washed with tap water, chopped into small pieces and dipped in 0.5% (W/V) citric acid solution for 10 min (*Alkarkhiet al., 2011*) then ,sun-dried for 10 days. The dried materials were separated and reduced into powder as far as possible and stored in the refrigerator at 4°C until use,according to(*Nwannaet al.,2019*).

Phenolic compounds of eggplant fruit

The polyphenolic compounds of fruit extract were fractionated and identified for phenolic compounds and flavonoid compounds by LC-MS, according to the method described by (*Tanget al., 2022*).

Diet Preparationand experimental design

The basal diet of the rats was prepared following the laboratory animal diet guidelines as previously described according to(*Reeves et al., 1993*). Treatment diets were prepared by incorporating 50 g & 100g of white and purple eggplant powder into each kilogram of food to get a concentration of 5%&10%.

Experimental design

Thirty-six adult male albino rats *Sprague Dawley* strain weighing (150±10g) were housed in well-aerated cages under a hygienic condition and were fed on a basal diet, according to *Reeves et al., (1993)*, for one week for adaptation. After this week, the rats were divided into two main groups: The first group (6 rats) was fed on a basal diet and served as a negative control. The second group (30 rats) received 17 mg/kg b.w.

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ALCL₃ solute in saline and given by gavage daily (for 28 days) to induce toxicity according to **Ahmed et al., (2015)** and divided into five subgroups as follows : Sup group (1): was fed on basal diet + received 17 mg/kg b.w. ALCL₃ (and served as positive control). Supgroup (2): were fed on basal diet + (5%) white eggplant dried fruits+ received 17 mg/kg b.w. ALCL₃. Sup group (3) were fed on basal diet + (10%) white eggplant dried fruits+ received 17 mg/kg b.w. ALCL₃. Sup group (4): were fed on basal diet + (5%) of purple eggplant dried fruits + received 17 mg/kg b.w. ALCL₃. Sup group (5): were fed on basal diet + (10%) of purple eggplant dried fruits+ received 17 mg/kg b.w. ALCL₃. (**Fajriana et al., 2017**). Body weight and feed intake were checked once a week. Ultimately, animals were weighed, fasted overnight, and sacrificed under light anesthesia. 36 rats

Biological evaluation

At the end of the experiment, feed intake, body weight gain, relative organs weight, and feed efficiency ratio were calculated according to **Chapman et al., (1959)**.

Preparation of brain homogenates and biochemical analysis

The cerebrum and Cerebellum were separated and cleaned with an ice-cold saline solution. Brain parts were homogenized in a proportion of 1:10 (W/V) ice-cold KCL buffer (1, 15%; pH 7, 2). The homogenate was centrifuged at 10.000×g for 10 minutes at four °C to obtain post mitochondrial supernatant (PMS), which was used for the quantification of gamma-aminobutyric acid (GABA) according to (**Lasley et al., 1984**), acetylcholine esterase (AChE) was estimated according to (**Carageorgio et al., 2005**), dopamine (DA) and serotonin (S.T.) were determined according to (**Sasa and Blank, 1977**).

Assessment of antioxidant activities and lipid peroxidation in the brain tissues:

Antioxidant indications were assessed, such as superoxide dismutase (SOD), according to **Nandi and Chatterjee**, (1988). Glutathione peroxidase (GPX), according to **Hadwan and Ahmed**, (2021) Catalas (CAT) according to **Soto et al.**, (2011). Malondialdehyde (MDA), according to **Giera et al.**, (2012), and Tumor necrosis factor - α (α -TNF), according to **Acharya et al.**, (1996).

Histopathological examinations

The brains were fixed in situ through saline perfusion through the aorta as centers for 3 min followed by 4% formaldehyde for 10 min and immersion in 4% formaldehyde for 24 h and then removed from the skulls by a non-traumatic technique (resection of bone structures at the skull base, followed by a midline incision from the foramen magnum to the nose). Immersion fixed in 4% formaldehyde for more than 24 h. Whole coronal sections of the brains were dehydrated and embedded in paraffin, and sectioned at 5 μ m with a microtome. Samples of other organs were frozen or fixed for possible future analysis, according to **Grafströmet et al.**, (2008).

Statistical analysis:

Results are expressed as mean \pm standard deviation (S.D.). Differences between means in different groups were tested for significance using a one-way analysis of variance (ANOVA) followed by Duncan's test, and the P value of 0.05 or less was considered significant .using SPSS (version 20) according to **(Sendcor and Cochran, 1979)**.

Results and Discussion

Phenolic compounds of Purple and white eggplant by LC-MS analysis

Purple eggplant was analyzed for its phenolic compounds. The results are shown in Table 1; purple eggplant recorded higher content of Apigenin (isomers Galangin or Genistein) 2-Hexoses-120, M-Kaempferol+2H (440 m/e) While the lowest compounds are Coumaric Acid-H₂O and Hydroxybenzoic acid. White eggplant was analyzed for its phenolic compounds. The obtained results are shown in Table 2 white eggplant recorded higher content of Apigenin (isomers Galangin or Genistein) 2-Hexoses -120 Caffeic-2 Hexoses - 90 - H and Caffeic-Pentose – Hexose – 60-H. In comparison the lowest compounds are Caffeic-Hexose – 90-H and Caffeic – Pentose – 60-H.

The present study was supported by **Mohamed et al., (2019)** who found that purple-colored eggplant had higher total phenolic and total flavonoid content than the white a green colored eggplant, pale green eggplant, and long green eggplant. **Colak et al., (2022)** cleared that the most common anthocyanin structure in peel in eggplant fruits is delphinidin-3-(p-coumaroyl-rutinoside)-5-glucoside, known as nasunin, while the main phenolic acid in the flesh is chlorogenic acid (CGA) together with its hydroxycinnamic acid conjugates (chlorogenic acid isomers, is chlorogenic acid isomers, amide conjugates, unidentified caffeic acids conjugates, and acetylated chlorogenic acid isomers) varying from 75 to 94% of total phenolic content in a wide range of eggplant.

Biological evaluation

Feed intake (F.I.), body weight gain % (BWG), feed efficiency ratio (FER), and relative brain weight are shown in (Table

3) showed a significant decrease in the positive control group compared to the negative group. However, the other treated groups have revealed a significant increase in all of them compared with the positive control group. The best results were found in the purple eggplant 10% group in F.I., BWG, and FER; the superior results for relative brain weight were recorded in groups treated with purple eggplant 5, 10% and white eggplant 10%. The findings of the ongoing work are in line with *Han et al., (2013)* and *Abbas et al., (2022)* indicated that the final body weight value and brain weight were significantly lower in the AlCl₃ group compared to the normal control group because chronic exposure to AlCl₃ in rats induced significant body weight loss this is consistent with previous reports which documented impairment of normal metabolism by aluminum. The weight loss was mediated through protein and lipid oxidation, inhibiting glycolysis and the Krebs cycle.

Brain functions

The data in Table 4 indicated that the mean values of AChE in the positive control group were significantly higher than in the negative control group. In the treated group, they were significantly decreased ($P < 0.05$) compared to the positive control. The purple eggplant (10%) group shows the best findings. The results revealed that the mean value of serum gamma-aminobutyric acid, serotonin, and dopamine in the aluminum chloride group (G+) was significantly decreased compared with the normal group (G-). In contrast, all treated groups recorded a significant increase compared to the positive group.

The highest level of GABA, serotonin, and DA was found in the group treated with purple eggplant (10%). These findings agree with *Kaizer et al., (2005)* found that increased AChE activity following AlCl₃ administration may be due to the direct neurotoxic effect of toxic metal on the cholinergic system or could be due to

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disruption of plasma membrane resulting from increased lipid peroxidation (LPO). **Kaizer et al ., (2008)** and **Aljarari and Bawazir, (2019)** demonstrated that the oral administration of AlCl₃ significantly increased AchE activities of the cerebral cortex and hippocampus homogenate compared to the control group.

It has been demonstrated that the increase in AchE activity after Al exposure was due to the allosteric reaction between Al and the enzyme molecule's peripheral anionic location, resulting in changes in the secondary structure and enzyme activity. In this respect, **Liaquat et al., (2019)** reported that to be involved in increased AChE activity in the brain is apoptotic neuronal loss following Al intoxication that later results in cognitive dysfunction. Increased AChE activity may result in cognitive dysfunction due to increased degradation and low availability of ACh in the synapse.

Temitayo et al.,(2020) found that AChE interferes with choline uptake/release at presynaptic terminals, resulting in an alteration of neurotransmission evidenced by substantial neocortical deficits in the enzyme responsible for the synthesis of acetylcholine (ACh), choline acetyltransferase (ChAT). We observed elevated levels of AChE in AlCl₃-treated groups. The ongoing findings are in harmony with **Obadeet al., (2018)**, who revealed that AChE inhibitory activity of all the solvent fractions of *S. dasyphyllum* might be a result of various phytochemicals present in the plants, such as alkaloids, flavonoids, coumarin, and steroids (unpublished), that have been reported to possess anticholinesterase activity. *S. dasyphyllum* can be classified among anticholinesterase plants in treating AChE-related neurological disorders.

The ongoing study's findings agree with **Abbas et al., (2019)**, reporting that the rats administered AICI₃ showed a significant decrease in the neurotransmitters concentration of dopamine in brain tissue because it alters the membrane polarization that allowed the entrance of calcium ions, resulting in vesicular fusion and releasing noradrenalin. It is pertinent to mention that the direct precursor of noradrenalin is dopamine, occurring predominantly in the neurotransmitter vesicle. **Aljarariand Bawazir (2019)** supported the present outcomes.

They demonstrated that the oral administration of AICI significantly decreased dopamine (DA) activities of the cerebral cortex and hippocampus homogenate compared to the normal control group. **Elreedyet al., (2022)** concluded that the group treated with AICI₃ significantly decreased in Dopamine (DA) and acetylcholinesterase) AChE level in serum. **Abbas et al., (2022)** indicated that brain dopamine and serotonin levels were significantly reduced in the AICI₃ group compared to the normal control group because AICI₃ increased depression-like behavior and helplessness. Depression is strongly associated with hypofunction of the central serotonergic system and a reduced brain serotonin level.

This finding is emphasized by **Junget al.,(2011) and Li et al.,(2017)** suggested that the levels of dopamine were more prominently and significantly elevated in the Eggplant (*Solanum melongena* L(E.E.) +6-hydroxydopamine hydrochloride (6-OHDA) receiving animals than the group as compared to the 6-OHDA lesioned animals indicated improved dopaminergic neurons' functional viability. The probable mechanism of E.E.'s neurorescue and neuroprotective effects could be the principal and significant amount of polyphenols and flavonoids, which are considered strong anti-oxidants.

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Gonçalves and Silva (2007) and *Aljarari and Bawazir, (2019)* cleared that in the cortex and hippocampus homogenate of the AICI₃ group. GABA was significantly decreased compared to the control group. The decrease in GABA level might be due to the increase in its catabolism through enzymatic activities and selective loss of GABAergic neurons due to exposure to AICI₃. *Horie et al., (2013)* mentioned that mental stress recovery effects are demonstrated after the oral ingestion of γ -amino butyric acid (GABA). The contents of GABA in fruit were compared among the eight varieties of eggplant (*Solanum melongena*).

Antioxidant enzymes (CAT, SOD, and GP_x), Lipid peroxidation parameter (MDA), and tumor necrosis factor (α -TNF) in brain tissue.

Table 5 shows that compared to the control (-ve), the activities of catalase (CAT), superoxide dismutase (SOD), and glutathione peroxidase (GPX) considerably decreased in positive control (ALCL₃), but they elevated in the treated groups. Purple eggplant (10%) had the best CAT, SOD, and GPX results. Further, the same table showed that the mean values of MDA were significantly higher in the ALCL₃ positive than in negative group, whereas their values were significantly lower in the other groups. At the same time, Purple eggplant (10%) showed the best improvements for MDA. The data in Table 6 showed that the treated groups mean TNF- α values were significantly higher than those of the normal group. Compared to the positive control rats, every parameter in the different remediations decreased significantly ($P < 0.05$). Purple eggplant (10%) gave the best improved result. These findings agree with *Wang et al., (2017)* found that the activities of SOD, CAT and GPx in primary hippocampal neuronal cells showed a significant decrease responding to the AICI₃ exposure.

However, the administration of CGA (chlorogenic acid) remarkably enhanced the activities of the SOD, CAT, and GPx. While the exposure to AlCl₃ resulted in an obvious increase in MDA level. Administration of CGA (50 µM) significantly decreased the level of MDA in the hippocampus compared with the Al treatment alone. In this respect, **Mohamed** and **Abd El-Moneim, (2017)** showed that rats administered AlCl₃ showed significant decreases (P<0.05) in SOD and CAT in brain tissue as compared to the control group.

The significant decrease in CAT and SOD determined in the brain of rats treated with AlCl₃ could be attributed to the direct interaction of Al with free radicals scavenging enzymes. These enzymes represent the first line of defense against oxygen free radicals, and the decrease in their levels may reflect the induction of oxidative stress in brain tissue. The ongoing findings are in harmony with **Liaquat et al., (2019)** revealed significantly impaired activities of antioxidant enzymes SOD and GPx in test rats compared to the control. In CAT, comparable results were obtained in AlCl₃ intoxicated rats compared to control rats. Brain MDA levels further confirmed the pro-oxidant nature of Al.

MDA is considered a biomarker of oxidative stress. A significant increase in brain MDA levels in AlCl₃ intoxicated was observed compared to control rats. These findings are emphasized by **Adelakun et al., (2020)** indicated that the CAT, SOD, and GPx levels in the *Solanum melongena* (S.M.) group were increased (p < 0.05) compared to a positive control group. MDA decreased in S.M. + HgCl₂ group compared to the HgCl₂-only group because S.M. is rich in antioxidant constituents such as phenol, chlorogenic acid, anthocyanin, ascorbic acid, and flavonoids. So S.M. boosted the enzymatic antioxidants to effectively scavenge the free radicals preventing lipid peroxidation and reducing HgCl₂ toxicity. The

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findings of the ongoing work are in line with **Hao et al., (2015)**, told that chlorogenic acid (C.A.) can modulate cadmium (Cd)-induced oxidative brain damage.

The intragastric administration of C.A. reversed the toxic effect of Cd and elicited a significant restoration of the levels of antioxidant enzymes SOD, CAT, and GPx. However, treatment with C.A. significantly diminished the degree of elevation of the MDA level in the brain compared to that in the rats treated with Cd alone. The results align with those of **Milnerowicz et al., (2015)** reported that exposure to metals such as aluminum increased TNF- α . Upregulation of these cytokines stimulates the recruitment of leukocytes, amplifying the inflammatory response by releasing more pro-inflammatory cytokines.

Maksoud et al., (2020) revealed that administration of AlCl₃ to normal rats exhibited a significant increase in brain TNF- α compared with the control group due to the accumulation of abnormal protein aggregates, like Ab-42 and free radicals (NO, ROS and RNS), that trigger cellular stress and neuroinflammation by activation of the brain's innate. The current findings align with **Lee et al ., (2018)** observed that eggplant has many polyphenols such as p-Coumaric acid (p-CA), which can reduce inflammation-related molecular changes such as TNF- α demonstrate the potential of p-CA as an alternative for treatment-resistant depression.

Histological Results

Microscopic pictures of H&E-stained cerebral sections from the control group show normal neurons in the cortex and hippocampal pyramidal layer. Neurons in cerebellar grey matter consisting of molecular "m", Purkinje "P", and granular "g" layers appear normal in the control group. Meanwhile, cerebral sections

from the ALCL₃ group show marked shrinkage and degeneration of neurons (black arrows) in the cortex, shrinkage (black arrows), and nuclear pyknosis (red arrows) of neurons in the hippocampal pyramidal layer. Neurons in the cerebellar Purkinje layer have dark eosinophilic cytoplasm indicating degeneration (black arrows) with marked loss of neurons (blue arrows) in the ALCL₃ group. Microscopic pictures of H&E stained cerebral sections from the treated group with 5% white eggplant show mild shrinkage and degeneration of neurons (black arrows) in the cortex, mild shrinkage (black arrows), and nuclear pyknosis (red arrows) of neurons in hippocampal pyramidal layer. Neurons in the cerebellar Purkinje layer from the treated group with 5% white eggplant show mild degeneration (black arrow) and mild loss of neurons (blue arrows).

Cerebral sections from the treated group with 10% white eggplant show milder shrinkage of neurons (black arrows) in the cortex and very few pyknotic nuclei of neurons in the hippocampal pyramidal layer (red arrows). Neurons in the cerebellar Purkinje layer from the treated group with 10% white eggplant appear normal. Microscopic pictures of H&E stained cerebral sections from the treated group with 5% Purple eggplant show very mild shrinkage and degeneration of a few neurons (black arrows) in the cortex, mild shrinkage (black arrows), and nuclear pyknosis (red arrows) of neurons in hippocampal pyramidal layer. Neurons in the cerebellar Purkinje layer from the treated group with 5% Purple eggplant show very mild degeneration (black arrow). Cerebral sections from the treated group with 10% Purple eggplant show mild shrinkage of neurons (black arrows) in the cortex and very few pyknotic nuclei of neurons in the hippocampal pyramidal layer (red arrows).

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Neurons in the cerebellar Purkinje layer from the treated group with 10% Purple eggplant appear normal. **Sumathiet al., (2015)** observed that the Control Transverse section of the brain's cerebral cortex showed normal histo-architecture. However, the AI-induced Transverse section of the brain's cerebral cortex shows diffused gliosis and pericellular edema. Control Transverse section of Cerebellum showing normal histo-architecture. However, the AI-induced Transverse section of the Cerebellum shows disruption in the Purkinjee cells layer. The Control Transverse section of the brain's hippocampus shows normal histo architecture. However, the AI-induced transverse section of the brain's hippocampus shows a high level of pyramidal cell degeneration with marked cell distortion.

Liaquatet al.,(2019) indicated that in the cerebral cortex of the control group, the neurons were tightly arranged, nuclear membrane and nuclei were cleared and lightly stained with a circular shape as compared to AICI3 injected rats in which the nuclear membrane was indistinguishable, the cytoplasm of AICI3 intoxicated neurons were shrunken and dark stained. The hippocampus Control group exhibited normal morphology with no damaged hippocampus neurons, whereas in AICI3-intoxicated rats, neurodegeneration was clearly observed with the irregular nucleus and shrunken cytoplasm. Vacuolated cytoplasm (V.C.) was also observed in AICI3-injected rats. **Al-Hazmi et al.,(2021)** observed that A Photomicrograph of brain cerebral cortex of male albino Wistar rats of the control group showed the normal histological structure of the cerebral cortex, and the cell bodies of the nerve cells embody a prominent dense nucleus encircled by granular cytoplasm. However The impact of AI exposure on the cerebellar cortex resulted in cellular disorganization, irregular cell body shape, eccentric nuclei, focal gliosis, congested cerebral vessels, and cellular deterioration.

Conclusion

The present study that Purple and white eggplant may have a protective effect against ALCL₃induced neurotoxicity. It successfully alleviated oxidative stress and neuroinflammation through its viability as an antioxidant and anti-inflammatory. Therefore, eggplant fruit administration could be useful in preventing and reversing the pathogenesis of neurodegenerative disorders.

Table (1):

phenolic compounds in Purple eggplant mass/ charge (m/e) .

Phenolic Compounds	Results (m/e)
Hydroxybenzoic acid	138
Coumaric Acid-H ₂ O	146
Apigenin(isomers Galangin or Genistein) - 2Hexoses-120	474
M-Kaempherol+2H	440
Ester of quininic and caffeicacids+H	355

Table (2):

phenolic compounds in white eggplant mass/ charge (m/e) .

Phenolic Compounds	Results (m/e)
Caffeic-2 Hexoses-90-H	413
Caffeic-Hexose-90-H	251
Caffeic-Pentose-60-H	251
Caffeic-Pentose-Hexose-60-H	413
Apigenin(isomers Galangin or Genistein) - 2Hexoses-120	474
M of ester of either Ferulic-Ferulic or p- coumaric-Synaptic	370

Table (3):

Protective Effect of white and purple eggplant powder on feed intake, body weight gain %, feed efficiency ratio and relative brain weight in rats with neurotoxicity (mean \pm S.D.)

Parameters Groups	feed intake (g)	, body weight gain (%)	feed efficiency ratio	Relative brain weight gm %
Control -ve	513 \pm 1.87 ^a	41.83 \pm 3.67 ^a	0.105 \pm 0.005 ^a	1.71 \pm 0.05 ^a
Control +ve (ALCL ₃)	385 \pm 2.4 ^f	12.69 \pm 1.80 ^f	0.043 \pm 0.004 ^f	1.44 \pm 0.02 ^d
ALCL ₃ +White eggplant 5%	449 \pm 1.41 ^e	19.19 \pm 1.79 ^e	0.056 \pm 0.005 ^e	1.52 \pm 0.06 ^c
ALCL ₃ +White eggplant	499 \pm 10.41 ^c	28.81 \pm 1.74 ^c	0.078 \pm 0.004 ^c	1.62 \pm 0.04 ^b
ALCL ₃ +Purple eggplant 5%	465 \pm 1.41 ^d	24.05 \pm 2.84 ^d	0.072 \pm 0.006 ^d	1.61 \pm 0.05 ^b
ALCL ₃ +Purple eggplant 10%	505 \pm 1.41 ^b	35.07 \pm 1.58 ^b	0.093 \pm 0.004 ^b	1.63 \pm 0.02 ^b

Means with different letters (in the same column are significant at (p \leq 0.05).

Table (4):

Protective Effect of white and purple eggplant powder on (GABA), (AChE), serotonin, and (DA) in rats brain with neurotoxicity (mean \pm S.D)

Parameters Groups	ACHE picograms (pg) per milligram (mg) Pg/mg	GABA picograms (pg) per milligram (mg) Pg/mg	Serotonin Nanograms (ng) per milligrams (mg) ng/mg	Dopamin Nanograms (ng) per milligrams (mg) ng/mg
Control -ve	126.05 \pm 0.18 ^f	1174.33 \pm 20.16 ^a	170.58 \pm 5.38 ^a	7.96 \pm 2.1 ^a
Control +ve (ALCL ₃)	322.8 \pm 5.5 ^a	71.71 \pm 5.22 ^f	30.7 \pm 2.3 ^f	0.33 \pm 0.05 ^f
ALCL ₃ +White eggplant 5%	295.06 \pm 4.31 ^b	271.83 \pm 8.56 ^d	64.98 \pm 6.27 ^e	2.43 \pm 0.08 ^e
ALCL ₃ +White eggplant 10%	226.05 \pm 5.3 ^c	704.61 \pm 10.52 ^c	113.75 \pm 8.18 ^d	5.67 \pm 0.4 ^c
ALCL ₃ +Purple eggplant 5%	181.15 \pm 7.24 ^d	203.75 \pm 8.33 ^e	118.08 \pm 7.23 ^c	3.76 \pm 0.6 ^d
ALCL ₃ +Purple eggplant 10%	168.05 \pm 5.28 ^e	815 \pm 9.46 ^b	145.03 \pm 5.18 ^b	7.15 \pm 1.4 ^b

Means with different letters (in the same column are significant at (p \leq 0.05) .

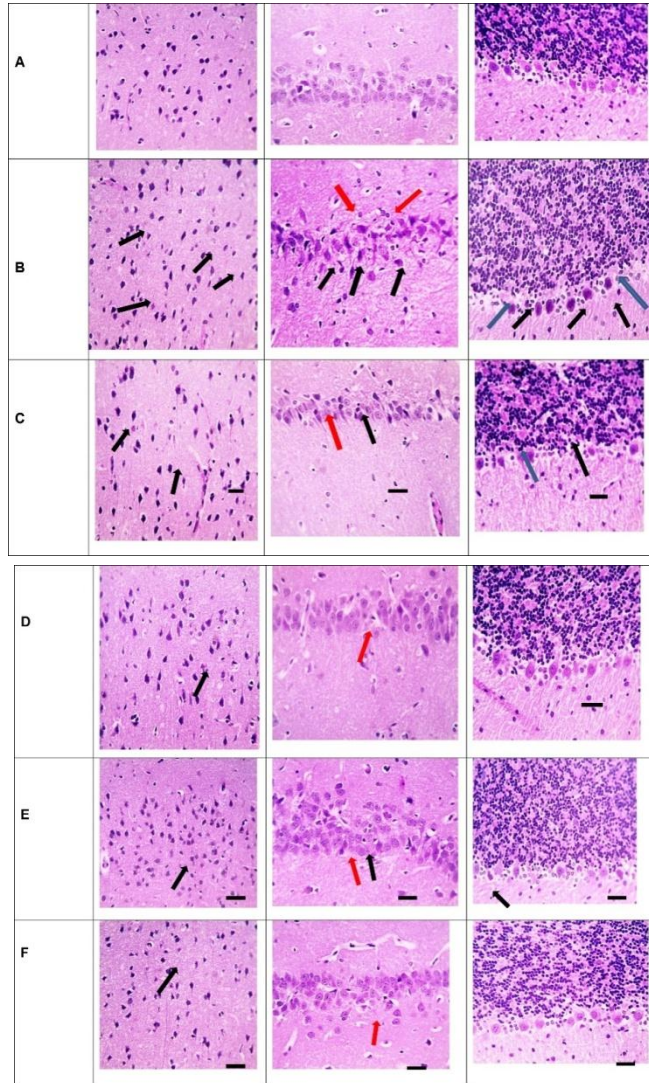
Table (5):

Protective effect of white and purple eggplant powder on Antioxidant indicators CAT, SOD, GP_x, MDA, and TNF- α in brain tissues
(mean \pm S.D.)

Parameters Groups	CAT Nanograms (ng) per milligrams (mg) ng/mg	SOD units per milligram (U/mg)	GP _x units per milligram (U/mg)	MDA nanomoles (nmol) per litre nmol/mg)	TNF- α Nanograms (ng) per milligrams (mg) (pg/mg)
Control -ve	12.27 \pm 0.98 ^a	210.5 \pm 3.18 ^a	211.83 \pm 2.11a	0.46 \pm 0.01 ^f	41.83 \pm 2.68 ^f
Control +ve (ALCL ₃)	0.85 \pm 0.07 ^f	25.5 \pm 2.04 ^f	30 \pm 1.78 ^f	10.2 \pm 2.01 ^a	397 \pm 7.78 ^a
ALCL ₃ +White eggplant 5%	2.69 \pm 0.38 ^e	80.66 \pm 2.73 ^e	48 \pm 2.61 ^e	4.033 \pm 1.01 ^b	262.25 \pm 8.67 ^b
ALCL ₃ +White eggplant 10%	3.84 \pm 0.33 ^d	144.5 \pm 3.37 ^c	78.5 \pm 3.34 ^d	2.56 \pm 1.10 ^c	168.16 \pm 6.12 ^c
ALCL ₃ +Purple eggplant 5%	5.69 \pm 0.33 ^c	163.16 \pm 3.58d	129.33 \pm 4.36c	1.273 \pm 0.10 ^d	166 \pm 9.89 ^d
ALCL ₃ +Purple eggplant 10%	9.03 \pm 0.18 ^b	178.5 \pm 2.23 ^b	159.5 \pm 5.34 ^b	0.85 \pm 0.01 ^e	75.66 \pm 4.12 ^e

Means with different letters (in the same column are significant at ($p \leq 0.05$).

Histological Results



Microscopic images of hematoxylin and eosin (H& Ex 400) stained brain sections showing (A) Normal (-ve) Control group. (B) ALCL₃ (+ve) Control group. (C) ALCL₃ + White eggplant 5% group. (D) ALCL₃ + White eggplant 10% group. (E) ALCL₃ + Purple eggplant 5% group. (F) ALCL₃ + Purple eggplant 10% group.

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التأثير الوقائي لثمار الباذنجان ضد السمية العصبية المحدثه بكلوريد
الألومونيوم في فئران التجارب

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الملخص العربي

أجريت الدراسة الحالية لتقييم التأثير الوقائي لثمار الباذنجان ضد السمية العصبية التي يسببها كلوريد الألومونيوم في الفئران. تم استخدام ستة وثلاثون من ذكور الفئران تتراوح أوزانهم بين (150 ± 10 جم) قسمت إلي مجموعتين رئيسيتين. المجموعة الأولى (6جرذان) تغذت علي الغذاء القياسي كمجموعة ضابطه سالبه المجموعة الثانية (30 جرد)، و المجموعة الثانية المكونة من 30 فأر تم إعطائهم كلوريد الألومونيوم بنسبة 17 ملجم / كجم من وزن الجسم مذابه في محلول محلي عن طريق الفم يوميا لمدة 28 يوم . وتم تقسيم هذه المجموعة إلي خمس مجموعات فرعية . مجموعه (1) تتغذى علي الغذاء القياسي +كلوريد الألومونيوم بنسبة 17 ملجم / كجم من وزن الجسم كمجموعة ضابطة موجبة . مجموعة(2) تغذت علي الغذاء القياسي بالإضافة إلي ثمار الباذنجان البيضاء المجففة بنسبة (5%) +كلوريد الألومونيوم بنسبة 17 ملجم / كجم من وزن الجسم . مجموعة (3) تغذت علي الغذاء القياسي بالإضافة إلي ثمار الباذنجان البيضاء المجففة بنسبة (10%) + كلوريد الألومونيوم بنسبة 17 ملجم / كجم من وزن الجسم . مجموعة (4) تغذت علي الغذاء القياسي بالإضافة إلي ثمار الباذنجان البنفسجي المجففة بنسبة (5%) . مجموعة (5) تغذت علي الغذاء القياسي بالإضافة إلي ثمار الباذنجان البنفسجي المجففة بنسبة (10%) لمدة 28 يوم. في نهاية التجربة تم حساب المآخوذ الغذائي، الزيادة المكتسبة في الوزن، معدل كفاءة الغذاء و الوزن النسبي للمخ . كما تم تقدير مضادات الأكسدة والعوامل المؤكسدة في أنسجة المخ و التحليل الكيمائية في أنسجة المخ كما أجري الفحص الهستوباثولوجي لأنسجة المخ وتم تقييم المركبات الفينولية في ثمار الباذنجان الأبيض والبنفسجي بواسطة LC-MS. أظهرت النتائج أن ثمار الباذنجان الأبيض والبنفسجي المجففة أدت إلى تحسين في التقييم البيولوجي ووظائف المخ والإنزيمات المضادة للأكسدو الفحص الهستوباثولوجي لأنسجة المخ مقارنة بالمجموعة الضابطة الموجبة ، وسجلت المجموعت المعالجة بالباذنجان البنفسجي أفضل نتيجة ،لذلك يمكن أن نستنتج أن تناول الفواكه المجففة من الباذنجان الأبيض والبنفسجي يمكن أن تقلل من تأثير كلوريد الألومونيوم الضارة على المخ

الكلمات المفتاحية: الباذنجان الأبيض ، الباذنجان البنفسجي ، كلوريد الألومونيوم ، ووظائف المخ ، الإنزيمات المضادة للأكسدة.