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The Histopathological and Immunohistochemical Effects of Lead Chloride on the cerebrum of Japanese Quail (*Coturnix coturnix japonica*)

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Abstract

Objective

Lead toxicity is a major environmental health concern with well-documented neurotoxic effects across vertebrate species. The Japanese quail (*Coturnix coturnix japonica*) is a valuable avian model for studying heavy metal toxicity due to its sensitivity to environmental contaminants and relevance to wildlife and poultry health. This research investigates the histopathological effects of lead chloride ($PbCl_2$) on the quail cerebrum and evaluates immunohistochemical exchanges by calculating glial fibrillary acidic protein (GFAP) expression.

Materials and methods

Thirty quails of both sexes were randomly divided into three groups of ten birds each. The control group received distilled water for 60 days. The first experimental group was administered $PbCl_2$ at 25 mg/kg body weight daily for 30 days, and the second experimental group received $PbCl_2$ at 50 mg/kg body weight daily for 30 days. Birds were euthanized at 15, 30, and 60 days from the start of the experiment for histopathological and immunohistochemical analysis.

Results

Histological examination of the cerebrum revealed multiple lesions across all time points. Widespread congestion was observed in most cerebral cortex layers, exclusively the inner pyramidal layer. Degeneration of glial and neuronal cells was noted in the outer granular layer, accompanied by glial cell clustering. Hypertrophy of pyramidal cells was observed in the outer

pyramidal layer. Immunohistochemical analysis of GFAP expression in the first experimental group showed a strongly positive reaction (11-25 cells stained) at 15 days, a weakly positive reaction (1-3 cells stained) at 30 days, and a weakly positive reaction at 60 days. In the second experimental group, GFAP expression was very strongly positive (>25 cells stained) at 15 days, strongly positive (11-25 cells stained) at 30 days, and strongly positive at 60 days.

Conclusions

Lead chloride exposure induces severe histopathological damage in the quail cerebrum, described by neuronal degeneration, glial activation, and inflammatory responses. Immunohistochemical and histopathological results elucidate the cellular mechanisms of lead-induced neurotoxicity. These results enhance understanding of-heavy metal toxicity in avian species and support the applying of Japanese quail as a sentinel species for environmental lead contamination investigations.

Keywords: Cerebrum, GFAP, immunohistochemistry, Japanese quail, lead chloride

Paper Type: Research Paper.

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Introduction

Lead is one of the earliest heavy metals utilized by humans and continues to be applied in large quantities. The smelting of lead has been practiced for thousands of years, and lead stays one of the most meaningful heavy metal pollutants due to its multiple sources, ease of transmission, and numerous health effects (Mansour, 2019). Lead is a persistent inorganic pollutant that affects all body systems, leading to neurological, hematological, immunological, reproductive, and behavioral disorders in exposed animals (Durkalec et al., 2022). The

accumulation of heavy metals in birds depends on numerous factors, containing dietary status, genetic diversity, age, sex, size of the birds, and feather types, as these environmental pollutants are non-biodegradable and accumulate at higher levels of the food chain (Habbo, 2021). Lead creates numerous histological, physiological, and biochemical abnormalities in humans and animals exposed to lead for extended periods (Adepoju et al., 2025).

Numerous investigations have demonstrated that elevated levels of lead toxicity can lead to serious health problems in humans, in addition to causing severe environmental pollution. Lead toxicity can be traced to sources like food, soil, air, drinking water, industrial emissions, electronic waste, industrial processes, herbal products, smoking, traditional medicines, cosmetics, household sources, paints, gasoline in pipes, toys, storage batteries, ceramics, and plumbing pipes (Munir et al., 2021). Lead exerts harmful effects on both the central and peripheral nervous systems. The developing brain is exclusively sensitive to lead, even at minimal concentrations, which can alter the trajectory of neurodevelopment, causing irreversible effects on motor, emotional, and cognitive traits that persist into later stages of adulthood in humans and animals (Ahmad et al., 2020). The central nervous system is the most affected by lead, as it contributes to neuronal damage by inducing oxidative stress through improved production of reactive oxygen species (ROS). Lead alters cellular biology by disrupting numerous metabolic pathways and numerous physiological processes, affecting critical neurobiological mechanisms involved in brain development (Adli et al., 2020). Livestock production in general and domestic chicken production in particular play a vital socio-economic role for people living in low-income countries of Africa and Asia (Mohamadinejad et al., 2024; Mohammadabadi et al., 2025). Domestic chickens are widely distributed avian species around the world due to their short generation interval and adaptability in a wide range of agroecological conditions (Mohammadifar & Mohammadabadi, 2017; Khabiri et al., 2025). Domestic chickens prepare high-quality protein and income for poor rural households and are the most widely kept livestock species globally (Mohammadabadi et al., 2010; Mohammadifar & Mohammadabadi, 2018; Mohammadabadi et al., 2024). This is attributed to valuable traits like disease resistance, adaptation to harsh environments, and the capability to utilize poor-quality feeds (Shahdadnejad et al., 2016; Khabiri et al., 2023). The Japanese quail (*Coturnix coturnix japonica*) is one of the most prominent small-sized domesticated bird species belonging to the Phasianidae family. It has a lifespan of 2–3 years and is primarily raised for meat and egg production, exclusively in resource-limited rural areas (Al-Badiri, 2017). Different species of quail are distinguished by their unique productive and reproductive characteristics (Elkhaiat et al., 2023). Japanese quail (*Coturnix coturnix japonica*) is an optimal species for heavy metal toxicity investigations, exclusively for neurological research, for different key reasons. The toxicity outcomes in Japanese quail are translatable to

human health because their physiological and metabolic pathways share similarities with mammals, and their brain architecture and maturation patterns are close to mammalian systems, offering insights into lead neurotoxicity (Hosseini et al., 2017). Furthermore, Japanese quail have a short incubation period of two weeks and reach sexual maturity in 6–8 weeks, permiting investigations of developmental stages when the nervous system is most vulnerable to lead toxicity (González-Redondo et al., 2023). Their size prepares sufficient brain tissue for histopathological and immunohistochemical investigations while staying cost-effective for large-scale investigations. The brain size is large enough to permit analysis of lead-induced exchanges (Bialas et al., 2022). Birds are highly sensitive to heavy metal toxicity, making Japanese quail a suitable species for detecting metal toxic effects (Liang et al., 2016). The current investigation aims to investigate the effect of lead chloride ($PbCl_2$) on the histological structure of the cerebrum in Japanese quail and to identify exchanges in glial fibrillary acidic protein (GFAP) in the cerebrum resulting from exposure to $PbCl_2$.

Materials and methods

Study birds: The investigation was managed on Japanese quail (*Coturnix coturnix japonica*), aged 6–8 weeks and weighing 200–250 g, achieved from the Department of Animal Production, College of Agriculture and Forestry, University of Mosul. The birds were housed in wooden cages designed for hygiene, with regular cleaning and sterilization, and prepared continuous access to feed. They were maintained under standard hygienic conditions with a 12-hour photoperiod, a temperature of 25°C, and a relative humidity of 45–50%. The birds had ad libitum access to water and commercial food pellets (Zorab et al., 2021).

Dosing strategy: The doses were selected based on a prior experiment determining the LD50 (Al-Kshab & Fathi, 2021). Lead chloride ($PbCl_2$) was administered at 25 mg/kg and 50 mg/kg body weight per day, dissolved in 2 mL of distilled water, and delivered orally via gavage needle.

Experimental design: Thirty Japanese quail of both sexes were randomly divided into three groups of ten birds each. The control group received distilled water orally for 60 consecutive days. The first experimental group was administered $PbCl_2$ at 25 mg/kg body weight daily for 30 days. The second experimental group received $PbCl_2$ at 50 mg/kg body weight daily for 30 days. Three birds from each group were euthanized at each time point (15, 30, and 60 days) from the start of the experiment.

Preparation of histological sections: The birds were euthanized following anesthesia with chloroform. The skull was carefully opened, and the brain was extracted applying fine forceps. Histological sections were prepared applying standard methods (Althanoon & Merkhan, 2023).

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Tissue sections were stained with Delafield's Haematoxylin and Eosin (H&E) and examined applying a digital camera attached to an optical microscope for imaging and analysis.

GFAP immunohistochemistry: Sections were stained for glial fibrillary acidic protein (GFAP) applying a primary antibody (Luijerink et al., 2020) based on the following protocol: paraffin was removed from sections in xylene, followed by rehydration through a graded series of ethanol washes. Antigen retrieval was carried out applying a 10% Tris-EDTA buffer solution for 14 minutes in a microwave. Sections were cooled for 20 minutes in a 3% phosphate-buffered saline (PBS) solution containing water and methanol, then blocked with 10% normal horse serum (NHS) for 30 minutes. Sections were incubated overnight with the primary antibody against GFAP, diluted to the appropriate concentration in 1% NHS. The following day, sections were incubated at room temperature for one hour with a purified secondary antibody against mouse/rabbit, followed by a one-hour incubation with an avidin-biotin peroxidase complex. Slides were counterstained with hematoxylin, dehydrated through a graded series of ethanol, cleared in xylene, and mounted for analysis.

Scoring of GFAP expression intensity: The intensity of GFAP expression per microscopic field was scored based on the number of stained cells, as outlined in Table 1.

Table 1. Scoring system for GFAP staining intensity based on the number of stained cells

Scoring system	Negative	Weakly positive (+)	Slightly Positive (++)	Strongly positive (+++)	Very Strong positive (++++)
Number of cells positive per slide field	0	1-3	4-10	11-25	>25

Results and Discussion

Histopathological results-normal structure of the cerebrum: Histological examination of the cerebrum in the control group of quails revealed a structure consisting of two main components.

Pallium (cerebral cortex): This region, comprising the gray matter, is externally surrounded by the pia mater and organized into different layers. The superficial fibromolecular layer contains nerve fibers oriented parallel to the pia mater (Figure 1A). The outer granular layer includes capillaries and small glial cells (Figures 1A, 1B, 1C). The outer pyramidal layer contains spherical

glial cells and capillaries (Figures 1A, 1B, 1C). The inner granular layer is described by stellate neurons, axons branching from neurons of preceding layers, and small glial cells (Figures 1A, 1C, 1D). The inner pyramidal layer, adjacent to the cerebral medulla, consists of large pyramidal cells, which are larger than neurons in previous layers, along with capillary blood vessels and glial cells (Figures 1A, 1D).

Cerebral medulla: This region comprises three types of fibers: association fibers, which connect different cortical areas within a single hemisphere; commissural fibers, which link the two hemispheres in cortical regions; and projection fibers, which connect internal cerebral structures (Figures 1A, 1D).

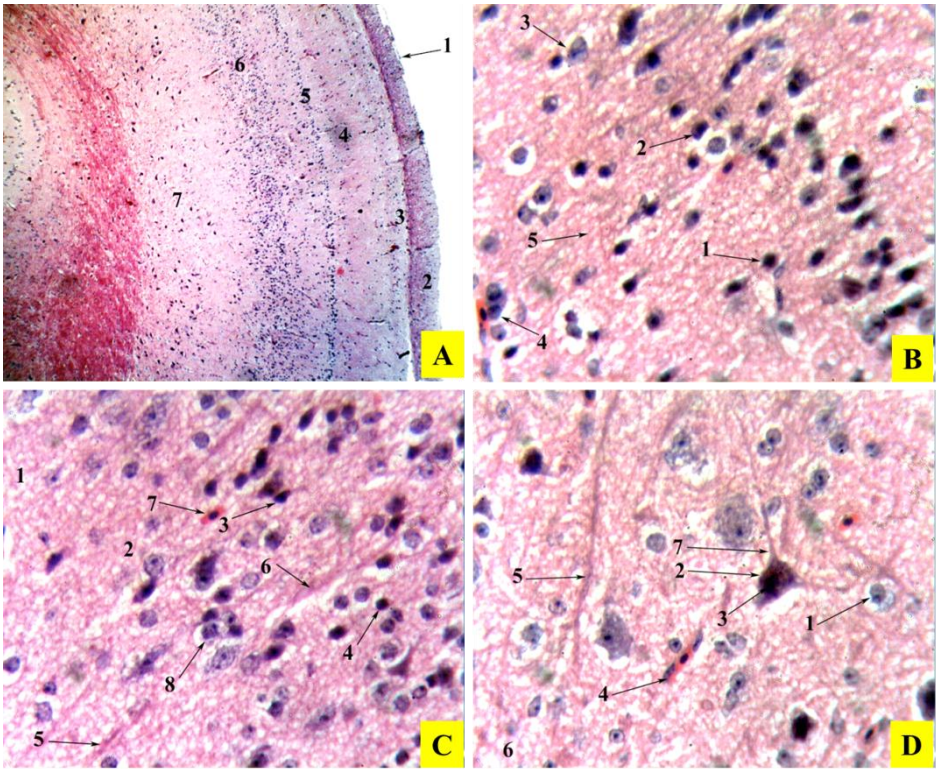

Figure 1. Histological sections of the quail cerebrum (control group). (A) Section showing: 1. Pia mater, 2. Fibromolecular layer, 3. Outer granular layer, 4. Outer pyramidal layer, 5. Inner granular layer, 6. Inner pyramidal layer, 7. White matter (100×, H&E stain). (B) Section showing: 1. Globular neurons, 2. Glial cells, 3. Astrocytes, 4. Blood vessel, 5. Nerve fibers (400×, H&E stain). (C) Section showing: 1. Outer granular layer, 2. Outer pyramidal layer, 3. Pyramidal cells, 4. Glial cells, 5. Nerve fibers, 6. Axons, 7. Blood vessel, 8. Astrocytes (400×, H&E stain). (D) Section showing: 1. Astrocytes, 2. Pyramidal cells, 3. Nucleus, 4. Blood vessel, 5. Nerve fibers, 6. Medulla, 7. Nerve axons (400×, H&E stain)

The cerebral structure observed in quails is consistent with that reported in other avian species, like the barn owl (Abd-Alrahman, 2017; Abid and Al-Bakri, 2016). The cerebrum plays a critical role in regulating voluntary behavior in birds, which are described by a relatively large cerebrum compared to reptiles and similarly sized vertebrates. Notable variations in cerebrum size exist among bird species, likely accompanied by differences in processing speed and efficiency of information within the cerebrum (Höglund et al., 2020).

Histopathological effects of $PbCl_2$ after 15 days: In the first experimental group (25 mg/kg $PbCl_2$), widespread vascular congestion was observed in most layers of the cerebral cortex, exclusively in the inner pyramidal layer. Disintegration of nerve fibers was noted in the fibromolecular and outer granular layers. Additionally, degeneration and clustering of glial cells were observed in the outer pyramidal layer (Figure 2A). In the second experimental group (50 mg/kg $PbCl_2$), separation of the pia mater from the cerebral cortex was evident, along with severe congestion of the pia mater's blood vessels and an improve in vessel size. Congestion of capillary blood vessels was observed across all cortical layers, most prominently in the outer pyramidal layer. Degeneration of glial and nerve cells, along with glial cell clustering, was noted in the outer granular layer. Necrosis of some glial cells was observed in the outer pyramidal layer (Figure 2B). These results are consistent with an investigation on female mice, which reported degeneration and necrosis of brain structures, along with a reduction in granule and molecular layer cells following $PbCl_2$ exposure (Al-Khafaf et al., 2021).

Histopathological effects of $PbCl_2$ after 30 days: In the first experimental group, separation of the pia mater from the fibromolecular layer of the cerebral cortex was observed. Degeneration of glial cells and an improve in nerve fiber density were noted in the fibromolecular layer. Congestion of capillaries was evident in most cortical layers, and vacuoles appeared among neural tissue components in the inner granular layer (Figure 2C). In the second experimental group, vascular congestion was observed in the outer pyramidal layer, with separation of blood vessels from surrounding tissue. Degeneration of glial cells was noted in this layer, along with vacuolation and atrophy of glial cells in the inner granular layer. Degeneration and necrosis of glial cells were observed in the inner pyramidal layer (Figure 2D). These results align with an investigation on rats exposed to lead, a heavy metal that accumulates in the brain, causing structural damage (Maiti et al., 2017). Similar results were reported in the cerebellar cortex of rats exposed to high lead doses, which exhibited exchanges in pyramidal and cortical cells (Highab et al., 2018). Comparable degenerative exchanges were observed in the brain of *Gambusia affinis* fish treated with $PbCl_2$ (Al-Kshab and Taha, 2021).

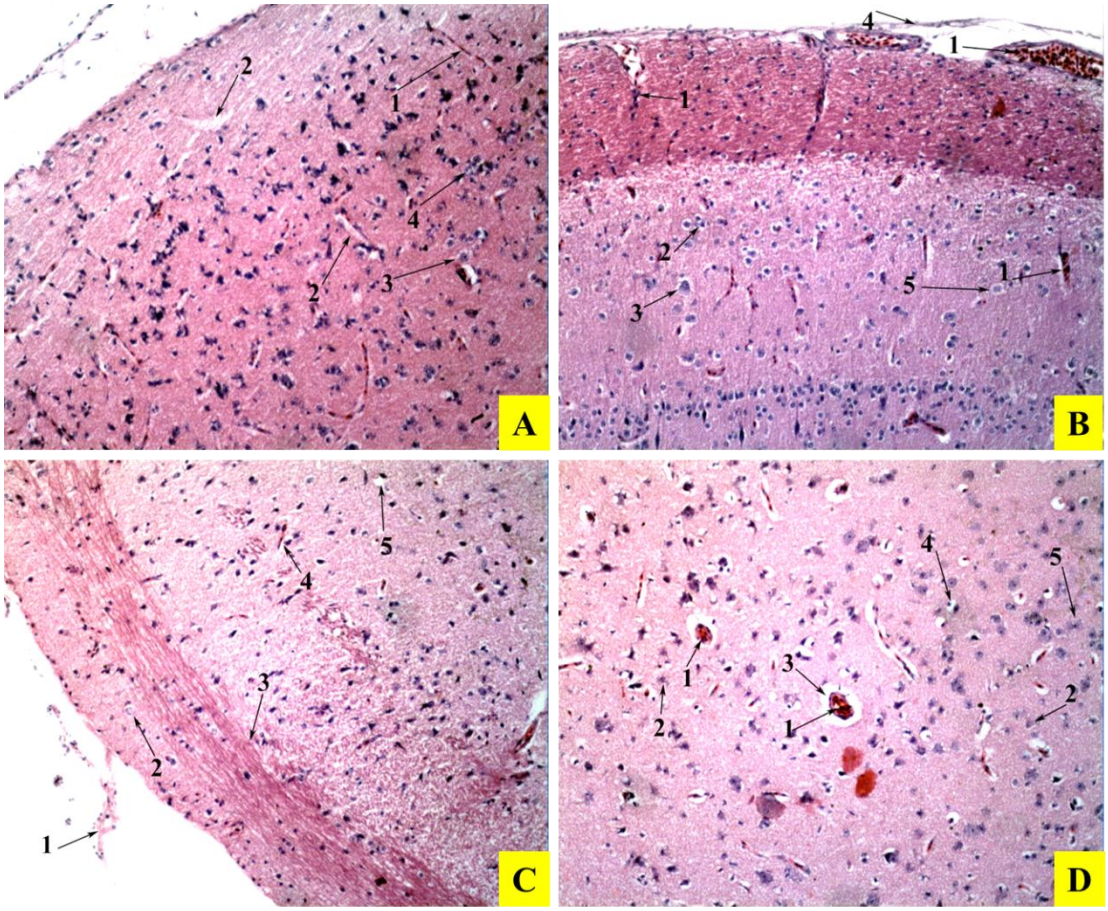

Figure 2. Histological sections of the quail cerebrum treated with $PbCl_2$. (A) First experimental group (25 mg/kg, 15 days) showing: 1. Congestion, 2. Disintegration of nerve fibers, 3. Degeneration, 4. Aggregation of glial cells (100×, H&E stain). (B) Second experimental group (50 mg/kg, 15 days) showing: 1. Congestion of blood vessels, 2. Aggregation of glial cells, 3. Degeneration, 4. Separation of the pia mater, 5. Necrosis of glial cells (100×, H&E stain). (C) First experimental group (25 mg/kg, 30 days) showing: 1. Separation of the pia mater, 2. Degeneration of glial cells, 3. Improved density of fibers, 4. Congestion, 5. Vacuolation (100×, H&E stain). (D) Second experimental group (50 mg/kg, 30 days) showing: 1. Congestion of blood vessels, 2. Degeneration, 3. Separation of the vascular surroundings, 4. Vacuolation, 5. Necrosis (400×, H&E stain)

Histopathological effects of $PbCl_2$ after 60 days: In the first experimental group, congestion of capillary blood vessels was observed across the cerebral cortex layers, accompanied by mild hemorrhage in the inner pyramidal layer. Disintegration of nerve fibers was noted in the inner granular layer, inner pyramidal layer, and medulla. Degeneration of glial and pyramidal cells, along with vacuolation, was observed in the inner pyramidal layer (Figure 3A). In the

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second experimental group, congestion of capillary blood vessels persisted across the cerebral cortex layers, with degeneration of glial cells in most layers. Hypertrophy of some pyramidal cells was noted in the outer pyramidal layer, along with clustering of glial cells in the outer granular layer. Vacuolation was observed in certain areas of the cerebral cortex (Figure 3B).

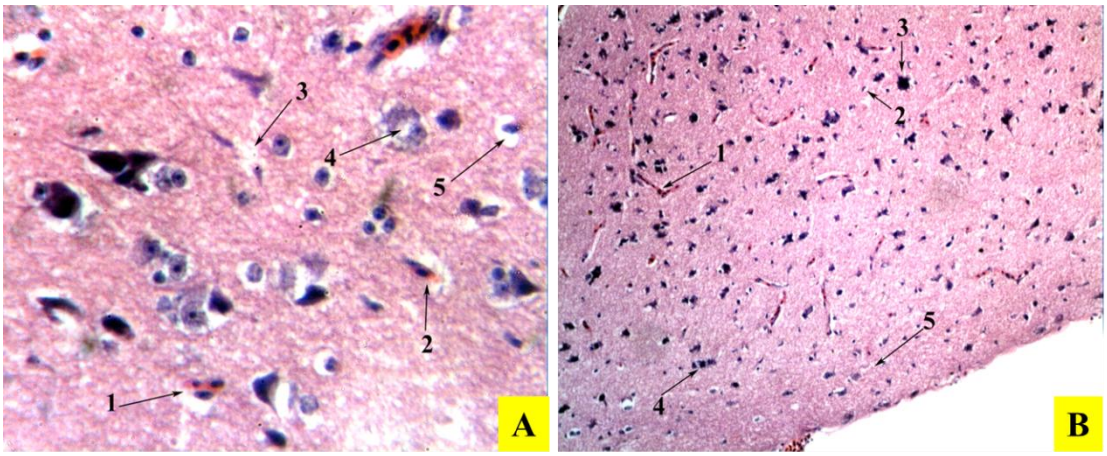

Figure 3. Histological sections of the quail cerebrum treated with $PbCl_2$ after 60 days. (A) First experimental group (25 mg/kg) showing: 1. Congestion, 2. Hemorrhage, 3. Disintegration, 4. Degeneration, 5. Vacuolation (400×, H&E stain). (B) Second experimental group (50 mg/kg) showing: 1. Congestion of blood vessels, 2. Degeneration, 3. Hypertrophy of astrocytes, 4. Aggregation of glial cells, 5. Vacuolation (400×, H&E stain)

These results are consistent with an investigation on quails exposed to lead, which reported microdamage in numerous brain regions (Zhang et al., 2024). Similar alterations in brain structure and cellular degeneration were observed in mice exposed to both low and high doses of lead (Hegazy et al., 2023). Additionally, an investigation on lead-treated mice reported neuronal degeneration and disruption of normal cerebral layering (Sidhu and Nehru, 2004).

Immunohistochemical results: In the control group, immunohistochemical staining for glial fibrillary acidic protein (GFAP) showed no response, denoting a negative reaction in cerebral tissue (Figure 4).

The immunohistochemical expression of GFAP in the cerebrum of the first experimental group revealed a strongly positive reaction (11-25 stained cells) in both neuronal axons and the cytoplasm of glial cells after 15 days from the onset of the experiment, denoting a widespread response in the brain tissue (Figure 5A). The staining appeared as brown-golden fibers within the cytoplasm. However, the reaction was weakly positive at 30 days (Figure 5B) and stayed weakly positive at 60 days (Figure 5C).

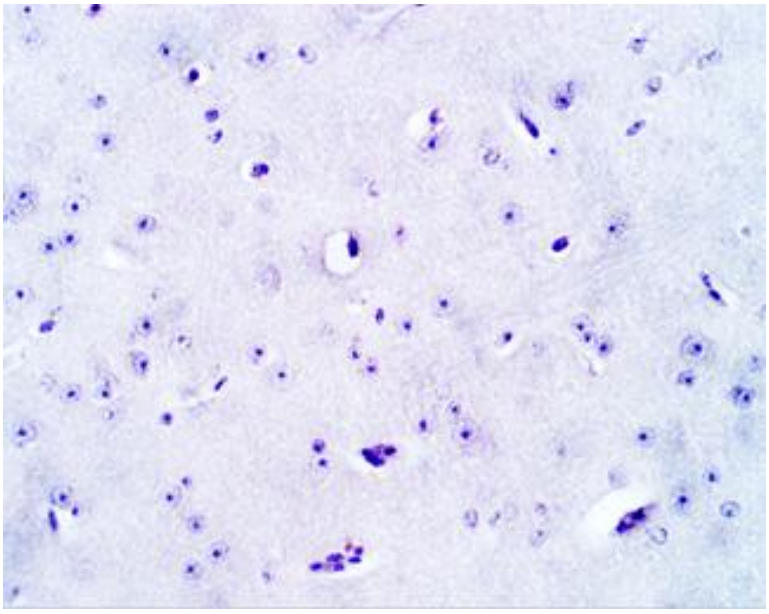

Figure 4. Histological section of the quail cerebrum (control group) showing a negative immunohistochemical reaction for GFAP (400×).

These results are consistent with an investigation on mice treated with lead acetate, which reported improved GFAP expression related to lead-induced neurotoxicity (Li et al., 2015). In rabbits exposed to lead, GFAP was concentrated in astrocytes, showing a meaningful improve from 15 days to 3 months (Amer and Azaghloul, 2015). Lead-induced neuroinflammation may elevate GFAP levels, impacting astrocyte function and contributing to neuronal damage and blood-brain barrier impairment (Farag et al., 2024; Goel and Aschner, 2021; Parithathvi et al., 2024). In the second experimental group (50 mg/kg $PbCl_2$), a highly positive and widespread GFAP reaction was observed after 15 days, appearing as brown-golden staining in neurons and glial cells (Figure 6A). The reaction stayed highly positive after 30 days, though less prevalent (Figure 6B), and showed a strong positive response after 60 days (Figure 6C). The results of this investigation are consistent with previous results in mice treated with lead, where neuronal and glial alterations in the brain were observed, along with an improved expression of glial fibrillary acidic protein (GFAP) in lead-exposed groups. Immunohistochemical analysis revealed GFAP expression accompanied by astrocyte hypertrophy and maturation following lead exposure (Harry et al., 1996). Similarly, the current results align with those reported by Sansar et al. (2011), who demonstrated immunological and histological exchanges in glial and dopaminergic cells in male mice exposed to lead. Their investigation reported enhanced GFAP immunoreactivity, alterations in cortical glial cells, a reduction in reactive neuronal immune processes, and impaired behavioral performance.

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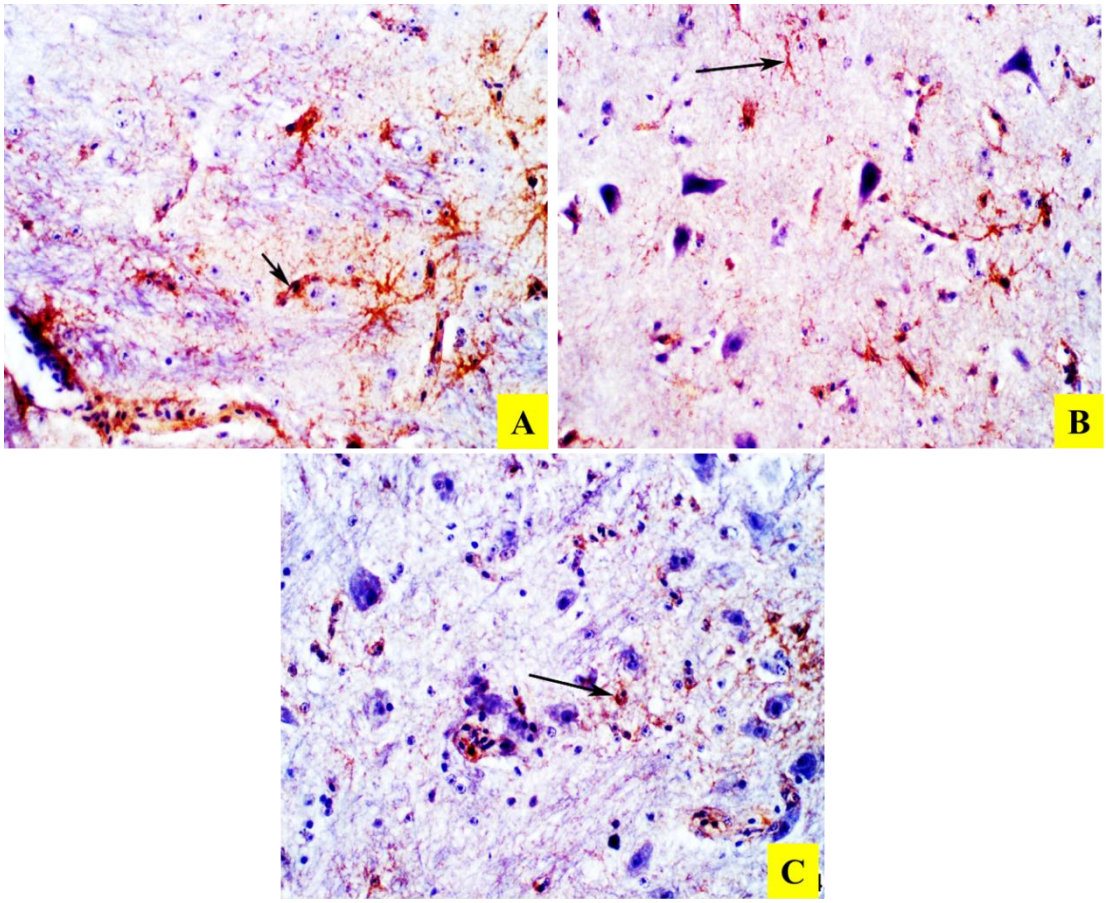

Figure 5. Histological sections of the quail cerebrum (first experimental group) showing GFAP immunohistochemical reactions. (A) Strong positive reaction (black arrow) appearing as brown-golden fibers in cerebral tissue after 15 days (400×). (B) Weak positive reaction (black arrow) appearing as brown-golden fibers in cerebral tissue after 30 days (400×). (C) Weak positive reaction (black arrow) appearing as brown-golden fibers in cerebral tissue after 60 days (400×)

The effects of lead are believed to result from its interference with astrocytic function, which affects GFAP expression through multiple mechanisms. Lead may alter GFAP gene expression, leading to exchanges in protein levels within astrocytes, or it may disrupt metabolic processes, indirectly influencing GFAP expression. Furthermore, lead-induced oxidative stress in astrocytes may result in protein damage, containing damage to GFAP (Oria et al., 2022; Amalia, 2021; Parithathvi et al., 2024).

Conclusions: This investigation concludes that exposure to lead chloride induces meaningful histological damage in the cerebral tissue of quails, with implications that may extend to humans and other animals. These effects also impact key proteins, notably leading to improved

expression of glial fibrillary acidic protein (GFAP), which reflects underlying immunological disturbances and suggests dysfunction within the cerebral tissue.

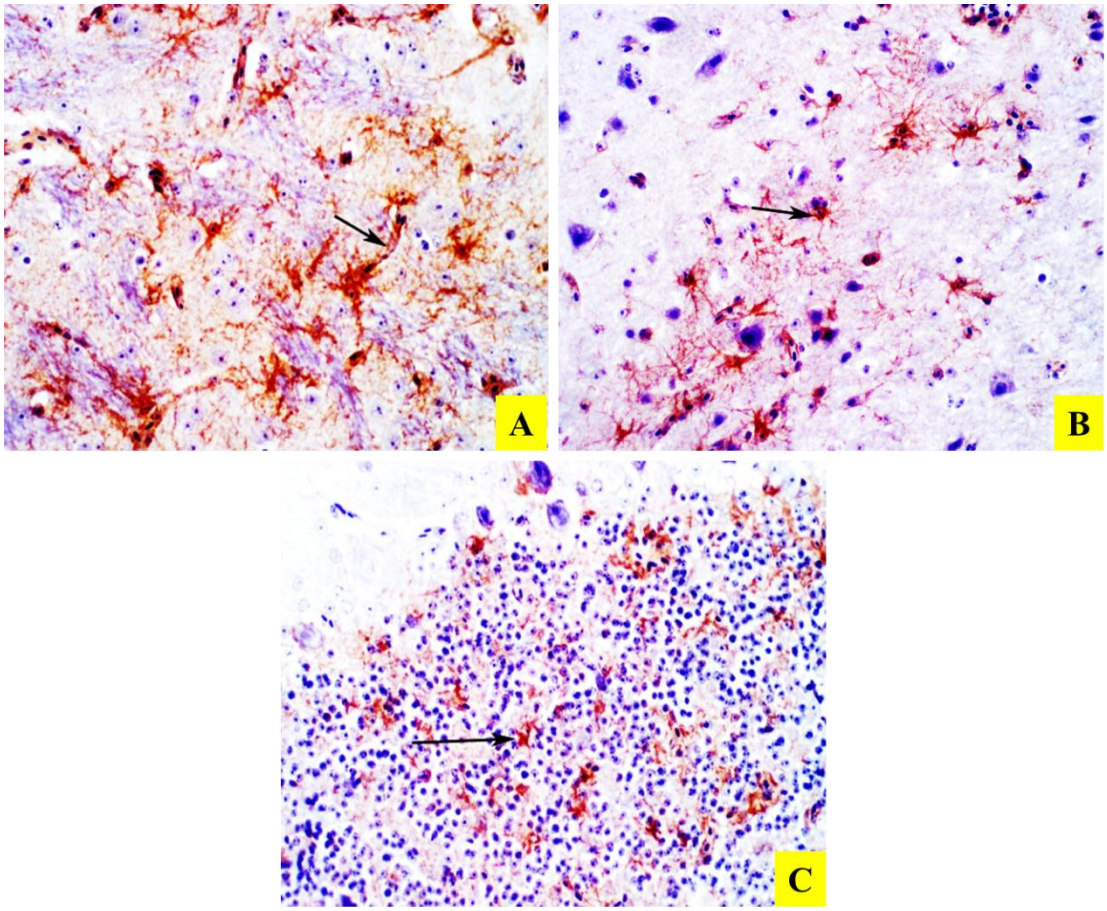

Figure 6. Histological sections of the quail cerebrum (second experimental group) showing GFAP immunohistochemical reactions. (A) Very strong positive reaction (black arrow) appearing as brown-golden fibers in cerebral tissue after 15 days (400×). (B) Strong positive reaction (black arrow) appearing as brown-golden fibers in cerebral tissue after 30 days (400×). (C) Strong positive reaction (black arrow) appearing as brown-golden fibers in cerebral tissue after 60 days (400×)
(X400)

Author contributions

Conceptualization: AMT; Data Collection: MIM; Formal Analysis: MIM; Investigation: MIM; Methodology: MIM; Project Supervision: AMT; Software: AMT; Resources: MIM and AMT; Validation: AMT; Visualization: AMT; Writing – Original Draft: MIM and AMT;

Writing-Review & Editing: AMT. All authors contributed equally to the conceptualization and preparation of both the original and revised drafts of the manuscript.

Data availability statement

The data supporting the results of this investigation are available from the corresponding author upon reasonable request.

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Ethical considerations

Ethical approval for this investigation was achieved from the College of Education for Pure Sciences, University of Mosul (Iraq), under reference number 6590, dated 11 November 2024.

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Conflict of interest

The authors declare no conflict of interest.

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تأثیرات آسیب‌شناسی بافتی و ایمنوهیستوشیمیایی کلرید سرب بر مخ بلدرچین ژاپنی (Coturnix coturnix japonica)

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چکیده

هدف: مسمومیت با سرب یکی از نگرانی‌های مهم بهداشت محیطی است که اثرات نوروتوکسیک (سمیت عصبی) آن در گونه‌های مهره‌دار به خوبی مستند شده است. بلدرچین ژاپنی (Coturnix coturnix japonica) به‌دلیل حساسیت بالا به آلاینده‌های محیطی و اهمیت آن در سلامت حیات‌وحش و طیور، یک مدل پرنده‌ای ارزشمند برای مطالعه سمیت فلزات سنگین محسوب می‌شود. این مطالعه به بررسی اثرات آسیب‌شناسی بافتی کلرید سرب ($PbCl_2$) بر مخ بلدرچین و ارزیابی تغییرات ایمنوهیستوشیمیایی با اندازه‌گیری میزان بیان پروتئین اسیدی فیبریلی گلیال (GFAP) می‌پردازد.

مواد و روش‌ها: سی بلدرچین از هر دو جنس به‌طور تصادفی به سه گروه ده‌تایی تقسیم شدند. گروه کنترل به مدت ۶۰ روز آب مقطر دریافت کرد. گروه اول آزمایشی روزانه به مدت ۳۰ روز $PbCl_2$ با دوز ۲۵ میلی‌گرم به‌ازای هر کیلوگرم وزن بدن دریافت کرد، و گروه دوم آزمایشی روزانه به مدت ۳۰ روز $PbCl_2$ با دوز ۵۰ میلی‌گرم به‌ازای هر کیلوگرم وزن بدن دریافت کرد. نمونه‌برداری برای بررسی‌های آسیب‌شناسی بافتی و ایمنوهیستوشیمیایی در روزهای ۱۵، ۳۰ و ۶۰ از آغاز آزمایش انجام شد.

نتایج: بررسی‌های بافت‌شناسی نشان‌دهنده بروز ضایعات متعدد در تمامی زمان‌های نمونه‌برداری بود. احتقان گسترده در بیشتر لایه‌های قشر مخ، به‌ویژه لایه پیرامیدال داخلی مشاهده شد. تخریب سلول‌های گلیال و نورونی در لایه گرانولی خارجی به‌همراه خوشه‌بندی سلول‌های گلیال به ثبت رسید. هیپرتروفی سلول‌های پیرامیدال در لایه پیرامیدال خارجی دیده شد. بررسی ایمنوهیستوشیمیایی بیان GFAP در گروه اول آزمایشی واکنش مثبت قوی (۱۱ تا ۲۵ سلول رنگ‌آمیزی‌شده) را در روز ۱۵، واکنش

مثبت ضعیف (۱ تا ۳ سلول رنگ‌شده) در روزهای ۳۰ و ۶۰ نشان داد. در گروه دوم، بیان GFAP در روز ۱۵ بسیار قوی (۲۵ سلول <)، در روز ۳۰ قوی (۱۱ تا ۲۵ سلول)، و در روز ۶۰ نیز قوی بود.

نتیجه‌گیری: قرار گرفتن در معرض کلرید سرب باعث آسیب‌های شدید بافتی در مخ بلدرچین می‌شود که با تخریب نورونی، فعال‌سازی سلول‌های گلیال، و پاسخ‌های التهابی همراه است. یافته‌های ایمنوهیستوشیمیایی و آسیب‌شناسی بافتی، سازوکارهای سلولی نوروتوکسیسیته ناشی از سرب را روشن می‌سازند. این نتایج درک بهتری از سمیت فلزات سنگین در گونه‌های پرنده فراهم می‌کنند و از بلدرچین ژاپنی به‌عنوان گونه‌ای شاخص برای مطالعات آلودگی محیطی با سرب حمایت می‌کنند.

واژگان کلیدی: ایمنوهیستوشیمی، بلدرچین ژاپنی، کلرید سرب، مخ، GFAP

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