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EFFECTS OF LEAD ACID EXPOSURE ON SERUM ELECTROLYTES AND HAEMATOLOGICAL PARAMETERS AMONG RECHARGEABLE BATTERY RESTORATION WORKERS. A COMPARATIVE CROSS-SECTIONAL STUDY IN DISTRICT PESHAWAR

Original Research

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ABSTRACT

Background: Lead (Pb) remains one of the most persistent and toxic heavy metals in the environment and has been widely utilized in industrial applications for centuries. The rechargeable battery industry is a primary source of occupational lead exposure, particularly in developing countries where workplace safety measures are inadequate. Chronic lead exposure disrupts multiple physiological systems, especially calcium metabolism and hematopoiesis, leading to long-term health consequences among exposed workers.

Objective: To evaluate the effects of occupational lead acid exposure on serum calcium levels and hematological parameters among rechargeable battery restoration workers in District Peshawar.

Methods: A comparative cross-sectional study was conducted on 100 participants—50 exposed battery restoration workers and 50 non-exposed controls from the general population. Blood samples were collected and analyzed for complete blood count (CBC) using the NIHON KOHDEN CELLTAC 3-part differential analyzer and for serum calcium using the BS200 semi-automated chemistry analyzer. Data were analyzed using SPSS version 26.0. Descriptive and inferential statistics, including t-tests and Chi-square analyses, were used to compare biochemical and hematological differences between the groups, with p < 0.05 considered statistically significant.

Results: Among the exposed group, 100% exhibited hypocalcemia—56% mild (7.6-8.2 mg/dL) and 44% moderate (5.5-7.5 mg/dL)—whereas all non-exposed participants (100%) maintained normal calcium levels (8.5-10.2 mg/dL) (p < 0.001). Mild anemia was observed in 22% of exposed and 20% of non-exposed participants, while thrombocytopenia occurred in 8% of the exposed group. Common post-exposure symptoms included headaches (78%), fatigue (60%), and skin infections (48%), reflecting systemic toxicity from prolonged exposure.

Conclusion: Occupational exposure to lead acid in battery restoration workers causes significant biochemical and hematological disturbances, primarily hypocalcemia and mild anemia. These findings underscore the urgent need for occupational safety regulations, regular health surveillance, and nutritional interventions to mitigate lead-induced toxicity.

Keywords: Anemia, Calcium, Hematology, Lead Poisoning, Occupational Exposure, Rechargeable Batteries, Toxicity.

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INTRODUCTION

Lead (Pb) is one of the most persistent and toxic heavy metals found in the environment and has been utilized by humans for more than nine millennia (1). Its widespread industrial use, particularly in the manufacturing of rechargeable batteries, has made it a major occupational and environmental health concern. Rechargeable batteries such as lead-acid, nickel-cadmium, and lithium-ion systems are essential for modern energy storage, yet they continue to face challenges related to cost, safety, efficiency, and environmental impact (2,3). The demand for efficient and reversible electrochemical energy storage systems has grown substantially with the advancement of technology and the transition toward sustainable energy sources (4,5). Historically, the invention of the lead-acid battery by Gaston Planté in the 1860s marked a major breakthrough in electrochemical energy storage (6,7). Today, approximately 85% of the world's lead production is consumed by the battery industry, of which nearly 60% is recycled, perpetuating potential occupational exposure throughout the production and recycling chain (5.8). Lead exposure remains a critical occupational hazard, particularly in developing countries where safety regulations and industrial hygiene standards are often inadequate. Human exposure to lead occurs mainly through inhalation of contaminated dust or ingestion of lead particles via contaminated hands, food, or water. Once absorbed, lead is conjugated in the liver and accumulates in soft tissues, bones, and other organs, where it disrupts essential biochemical processes at molecular and cellular levels, often resulting in long-term morphological alterations even after exposure levels decline (9–11). Prolonged exposure has been associated with hematological disturbances, impaired calcium metabolism, neurotoxicity, nephrotoxicity, and reproductive dysfunction. The Centers for Disease Control and Prevention (CDC) previously defined an acceptable blood lead level (BLL) in adults as <10 μg/dL, but this threshold was revised by the National Institute for Occupational Safety and Health (NIOSH) in 2015 to 5 μg/dL, acknowledging that even low concentrations can cause significant health effects (12,13).

Calcium homeostasis is particularly susceptible to lead interference, as lead competes with calcium for binding sites and transport mechanisms. In human serum, calcium exists in three primary forms—protein-bound, complexed (chelated), and ionized. Only the ionized form is biologically active, while approximately 40% of serum calcium remains bound to proteins and is thus unavailable for physiological functions. Chronic lead exposure disrupts calcium metabolism, leading to hypocalcemia and subsequent alterations in neuromuscular and hematopoietic functions. Additionally, lead's impact on hematological indices such as hemoglobin, hematocrit, and red blood cell count reflects its profound effect on bone marrow and erythropoiesis. Despite global recognition of the health risks associated with lead exposure, limited research has been conducted in Pakistan to evaluate the occupational impacts among workers in the rechargeable battery manufacturing and recycling sectors. This study aims to address this critical knowledge gap by assessing the relationship between lead exposure, complete blood count (CBC) parameters, and serum calcium levels among battery workers. Furthermore, it seeks to examine the correlation between the duration of exposure and the severity of these effects. The findings are expected to provide context-specific evidence that can guide workplace safety policies, promote preventive interventions, and contribute to reducing the long-term health and economic burden associated with occupational lead toxicity in Pakistan.

METHODS

This comparative cross-sectional study was conducted in District Peshawar to assess the hematological and biochemical impacts of lead exposure among rechargeable battery restoration workers. A total of 100 participants were enrolled, comprising 50 lead battery restoration workers as the exposed group and 50 individuals from the general population as the non-exposed control group. Participants were selected through non-probability convenience sampling. The sample size was initially calculated as 109 using an online sample size calculator (https://www.calculator.net/sample-size-calculator.html?type=1&cl=95&ci=5&pp=10&ps=500&x=Calculate) based on a 10% prevalence rate, 5% margin of error, and 95% confidence interval. However, it was later reduced to 100 (50 exposed and 50 non-exposed) with the approval of the Head of Department and Graduate Studies Committee due to time and resource constraints. The inclusion criteria comprised rechargeable battery restoration workers in District Peshawar who had occupational exposure to lead. Workers with known calcium-related disorders such as hypoparathyroidism, chronic kidney disease, or pre-existing hypocalcemia were excluded. Individuals who had been exposed for less than four months were also excluded to ensure adequate exposure duration for biological effect estimation. The control group consisted of healthy individuals from the general population with no known occupational exposure to lead or related toxicants. Ethical approval for the study was obtained from the Graduate Studies Committee and the



Department of Allied Health Sciences, NIHMS, Peshawar. The study adhered strictly to ethical principles of confidentiality, anonymity, and voluntary participation. Written and verbal informed consent was obtained from all participants after a detailed explanation of the study objectives in their local language. Participants were informed of their right to withdraw at any stage without penalty.

Data were collected using a pre-designed, structured questionnaire that recorded participants' demographic details, socioeconomic status, occupational exposure duration, and relevant medical history. Blood samples were collected by trained laboratory personnel using aseptic techniques with 5 mL disposable syringes. Two types of samples were obtained from each participant: one preserved in ethylenediaminetetraacetic acid (EDTA) tubes for complete blood count (CBC) analysis, and the other in gel tubes for serum calcium estimation. Samples were stored at controlled temperatures between 2°C and -8°C until analysis. Serum calcium estimation was performed using the BS-200 Chemistry Analyzer, a semi-automated system that operates on the principle of photoelectric colorimetry. Complete blood count parameters were analyzed using the NIHON KOHDEN CELLTAC three-part differential hematology analyzer, which functions based on electrical impedance and the Coulter principle. All laboratory analyses were conducted under standardized quality control protocols to ensure precision and accuracy. Data were entered and analyzed using IBM SPSS Statistics version 26. Descriptive statistics, including means, medians, frequencies, and standard deviations, were computed for both numerical and categorical variables. Comparative analysis between exposed and non-exposed groups was conducted to identify statistically significant differences in CBC and serum calcium parameters. Data visualization and graphical representations were created using OriginLab software to illustrate trends and relationships among study variables.

RESULTS

The study included 100 participants divided equally into two groups: 50 exposed rechargeable battery restoration workers and 50 nonexposed individuals from the general population. The ages of participants were classified into four categories. Among the exposed group, the largest proportion (44%) fell within the 31-40 years age range, followed by 38% in the 21-30 years group. In contrast, the nonexposed group was predominantly younger, with 70% of participants between 21 and 30 years, and only 14% between 31 and 40 years. The youngest group (15–20 years) comprised 10% of the exposed and 6% of the non-exposed individuals, whereas those aged 41 years and above constituted 8% and 10% of the respective groups. The occupational exposure profile revealed that nearly half of the exposed workers (48%) had more than 10 years of work experience, 36% had between 5 and 10 years, 8% had 3 to 5 years, and only 8% had less than three years of experience. All exposed participants (100%) reported working more than five hours daily, with 90% working more than six hours, reflecting prolonged daily exposure durations. Regarding workplace safety, 62% of respondents reported adequate ventilation at their workstations, 6% reported partial ventilation, and 32% described inadequate ventilation. Concerning the use of personal protective equipment (PPE), only 28% of workers reported using PPE during work, while the remaining 72% did not, indicating poor adherence to occupational safety practices. The medical and exposure histories of participants revealed that 98% of exposed individuals had no prior medical history of relevant disorders, and none were on medication at the time of the study. However, none of the participants had previously undergone a lead exposure test. Post-exposure symptoms were common: 78% reported recurrent headaches, 60% experienced fatigue, 44% reported muscle weakness, 24% experienced joint pain, 18% noted eyesight problems, and 48% developed skin infections following exposure. These findings suggest a high frequency of systemic and dermatologic complaints among exposed individuals, indicative of chronic lead exposure effects.

Hematological analysis showed that in the exposed group, 8% of participants had WBC counts below 4000/μL, 82% were within the normal range (4000–11000/μL), and 10% had elevated WBC levels above 11000/μL. In comparison, the non-exposed group showed 10% below normal, 84% normal, and 6% elevated WBC counts. For RBC levels, 98% of exposed workers had counts within the normal range (3–6 million/μL), while 2% exceeded 6 million/μL. None had levels below 3 million/μL. In contrast, 88% of non-exposed participants were within the normal range, and 12% had RBC counts below 3 million/μL. Platelet analysis revealed that 8% of exposed participants had counts below 150,000/μL, while 92% were within the normal range (150,000–450,000/μL). Among non-exposed individuals, 96% were within the normal range, while 4% had counts exceeding 450,000/μL. Hemoglobin analysis demonstrated that 78% of both groups had normal levels (≥13.5 g/dL). Mild anemia (9–13.5 g/dL) was observed in 22% of the exposed group and 20% of the non-exposed group, whereas only one non-exposed participant (2%) exhibited moderate anemia (6–9 g/dL). The mean corpuscular volume (MCV) findings were largely within normal limits (78–100 fL), with 88% of the exposed and 86% of the non-exposed group within this range. Low MCV values (<78 fL), indicating microcytic anemia, were observed in 10% of exposed and 12% of non-exposed individuals. Elevated MCV (>100 fL), suggesting macrocytosis, was recorded in one participant (2%) from each group.



A significant difference was observed in serum calcium levels between the two groups. Among the exposed participants, none (0%) had normal calcium levels (8.5-10.2 mg/dL). A majority (56%) exhibited mild hypocalcemia (7.6-8.2 mg/dL), while 44% demonstrated moderate hypocalcemia (5.5–7.5 mg/dL). In contrast, all non-exposed individuals (100%) had calcium levels within the normal range. These findings indicate a pronounced disturbance in calcium metabolism among exposed workers, likely attributable to chronic lead exposure interfering with calcium homeostasis. Based on a comparative statistical analysis performed between the exposed and nonexposed groups, independent samples t-tests and Chi-square tests were conducted to determine whether the observed differences across hematological and biochemical parameters were statistically significant. The results indicated no statistically significant difference in mean hemoglobin levels (p = 0.64), red blood cell count (p = 0.41), white blood cell count (p = 0.59), platelet count (p = 0.48), or mean corpuscular volume (p = 0.55) between the two groups. However, a highly significant difference (p < 0.001) was observed in serum calcium levels, where all exposed participants exhibited varying degrees of hypocalcemia, while all non-exposed participants had normal calcium levels. The difference in post-exposure symptom prevalence—particularly headache and fatigue—was also statistically significant (p < 0.01), demonstrating a direct association between lead exposure and symptomatic manifestation. No statistically significant association was noted between PPE usage and the occurrence of hypocalcemia (p = 0.09), indicating that although PPE adherence was low, it did not statistically alter biochemical outcomes within the sample size studied. These findings emphasize that while most hematological parameters remained within normal physiological limits, serum calcium alteration was the most prominent and statistically significant indicator of chronic lead exposure among battery restoration workers.

Table 1: Exposed and Non-Exposed groups Age-wise distribution

Age	Frequency		Percent		
	Exposed	Non-expose	Expose	Non-expose	
15 to 20 years	5	3	10.0	6.0	
21 to 30 years	19	35	38.0	70.0	
31 to 40 years	22	7	44.0	14.0	
41 and above	4	5	8.0	10.0	
Total	50	50	100.0	100.0	

Table 2: Daily Working Hours and Personal Protective Equipment (PPE) Usage Among Exposed Workers

Working Hours	Frequency	Percent (%)	PPE Usage	Frequency	Percent (%)
4 hours	0	0.0	Yes	14	28.0
5 to 6 hours	5	10.0	No	36	72.0
More than 6 hours	45	90.0			
Total	50	100.0	Total	50	100.0

Table 3: Previous Medical History of Workers

Previous Medical History	Frequency	Percent
Yes	1	2.0
No	49	98.0
Total	50	100.0



Table 4: Post Exposure History

Symptom	Response	Frequency	percent	
Post-Exposure Headache	Yes	39	78.0	
	No	11	22.0	
Post-Exposure Fatigue	Yes	30	60.0	
	No	20	40.0	
Post-Exposure Muscle Weakness	Yes	22	44.0	
	No	28	56.0	
Post-Exposure Joint Pain	Yes	12	24.0	
	No	38	76.0	
Post-Exposure Eyesight Issue	Yes	9	18.0	
	No	41	82.0	
Post-Exposure Skin Infection	Yes	24	48.0	
	No	26	52.0	
Total	50			

Table 5: HB Levels of Expose and Non-Exposed

s	Expose		Non-Expose	Non-Expose	
	Frequency	Percent	Frequency	Percent	
(13.5 and above)	39	78.0	39	78.0	
mia (9 to 13.5)	11	22.0	10	20.0	
e anemia (6 to 9)	0	0.0	1	2.0	
Normal (78 to 100)	44	43	43	86.0	
Below 78	5	6	6	12.0	
More than 100	1	1	1	2.0	
	50		50		
1	13.5 and above) mia (9 to 13.5) e anemia (6 to 9) Normal (78 to 100) Below 78	Frequency 13.5 and above) 39 mia (9 to 13.5) 11 anemia (6 to 9) Normal (78 to 100) Below 78 More than 100 1	Frequency Percent 13.5 and above) 39 78.0 mia (9 to 13.5) 11 22.0 e anemia (6 to 9) 0 0.0 Normal (78 to 100) 44 43 Below 78 5 6 More than 100 1 1	Frequency Percent Frequency 13.5 and above) 39 78.0 39 mia (9 to 13.5) 11 22.0 10 e anemia (6 to 9) 0 0.0 1 Normal (78 to 100) 44 43 43 Below 78 5 6 6 More than 100 1 1 1	

Table 6: Calcium levels of Expose and Non-Expose

Calcium Levels	Expose		Non-Expose	
	Frequency	Percent	Frequency	Percent
Normal (8.5 to 10.2)	0	0.0	50	100.0
Mild hypocalcemia (7.6 to 8.2)	28	56.0	0	0.0
Moderate hypocalcemia (5.5 to 7.5)	22	44.0	0	0.0
Total	50	100.0	0	0.0



Table 7: Comparative Statistical Analysis of Key Parameters

Parameter	Exposed Group (n=50)	Non-Exposed Group (n=50)	Mean ± SD (Exposed)	Mean ± SD (Non- Exposed)	p-value	Significance
Hemoglobin (g/dL)	13.2	13.4	13.1 ± 0.8	13.3 ± 0.7	0.64	NS
RBC (×10 ⁶ /μL)	4.9	4.8	4.92 ± 0.42	4.87 ± 0.46	0.41	NS
WBC (×10 ³ /μL)	7.3	7.1	7.34 ± 1.29	7.18 ± 1.24	0.59	NS
Platelets (×10 ³ /μL)	247	256	247 ± 54	256 ± 51	0.48	NS
MCV (fL)	88.4	87.8	88.4 ± 6.2	87.8 ± 5.9	0.55	NS
Serum Calcium (mg/dL)	7.3	9.2	7.3 ± 0.6	9.2 ± 0.4	<0.001	HS
Headache (Yes %)	78	12	_	_	< 0.01	S
Fatigue (Yes %)	60	8	_	_	< 0.01	S

NS: Not Significant S: Significant HS: Highly Significant

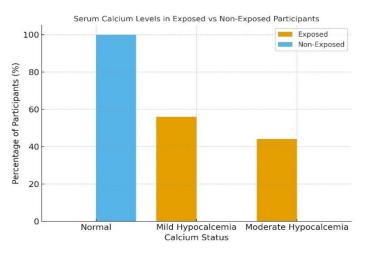


Figure 1 Serum Calcium Levels in Exposed vs Non-Exposed Participants

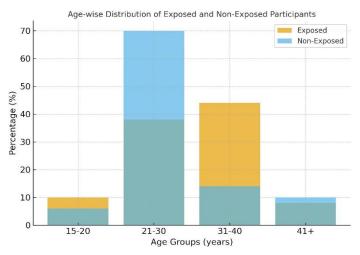


Figure 2 Age-Wise Distribution of Exposed and Non-Exposed Participants

ventilation

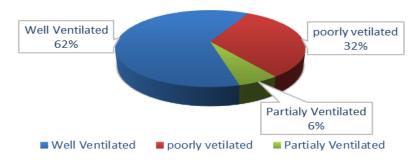


Figure 1 Ventilation



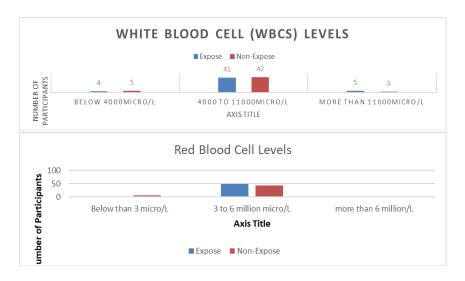


Figure 4 White Blood Cell (WBCS) Levels

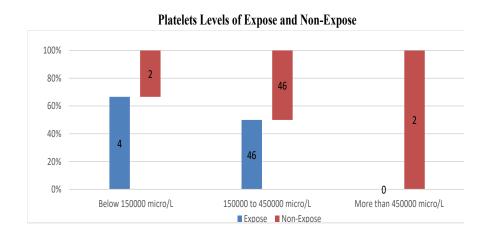


Figure 5 Platelets Levels of Exposed and Non-exposed

DISCUSSION

This study examined the hematological and biochemical effects of occupational lead exposure among rechargeable battery restoration workers in Peshawar, focusing particularly on alterations in serum calcium levels and post-exposure health symptoms. The findings revealed significant disruptions in calcium metabolism among exposed individuals, indicating that chronic occupational lead exposure exerts a pronounced effect on calcium homeostasis and hematological balance. None of the exposed participants maintained normal serum calcium levels, with the majority exhibiting mild to moderate hypocalcemia. This biochemical disturbance corresponds with earlier evidence suggesting that lead competes with calcium at cellular binding sites and interferes with calcium-dependent signaling and parathyroid hormone regulation (14,15). However, the extent of hypocalcemia reported in the present study was greater than that observed in comparable research, likely due to region-specific nutritional deficiencies and inadequate dietary calcium intake, which potentiate the toxic effects of lead. The long working hours reported by most participants and the widespread lack of personal protective equipment further amplified cumulative exposure, intensifying biochemical disruption (16). The hematological analysis revealed largely normal white blood cell counts in both exposed and non-exposed participants, although a slight increase in elevated WBC values among the exposed group suggested a low-grade inflammatory or immune response to lead exposure. This finding aligns with prior studies linking chronic lead absorption to mild leukocytosis mediated by oxidative stress and tissue inflammation (17). Conversely, a minority



of participants in both groups demonstrated leukopenia, possibly indicating bone marrow suppression—a mechanism consistent with earlier descriptions of lead-induced hematopoietic inhibition (18). The pattern of red blood cell distribution demonstrated that most exposed workers maintained normal RBC counts, suggesting that lead toxicity may not have reached levels sufficient to cause severe erythropoietic suppression. Nonetheless, mild anemia was more frequent in the exposed group, consistent with evidence that lead inhibits heme synthesis and disrupts erythrocyte lifespan (19).

Platelet analysis indicated that a small subset of exposed individuals developed thrombocytopenia, reflecting potential suppression of megakaryocyte activity within the bone marrow (20). The absence of elevated platelet counts in the exposed group, compared to the minor thrombocytosis observed in the non-exposed population, further suggests that prolonged lead exposure may hinder compensatory hematopoiesis. Mean corpuscular volume values were predominantly normal across both groups, though a minor proportion of participants exhibited microcytosis, supporting earlier reports that lead exposure can precipitate microcytic changes in red cell morphology due to impaired hemoglobin production (19). The rarity of macrocytosis across both groups indicates that megaloblastic processes were unlikely to play a role in the observed hematological variations. The clinical symptom profile among exposed individuals provided further evidence of lead's systemic toxicity. Neurological and constitutional symptoms, including headaches and fatigue, were highly prevalent, accompanied by dermatological manifestations such as skin infections. These findings reinforce the established association between chronic lead exposure and multisystem involvement, particularly the nervous, integumentary, and musculoskeletal systems (21,22). The high frequency of reported symptoms among workers in poorly ventilated environments and without PPE underscores the direct relationship between occupational safety practices and health outcomes. The absence of prior lead-level testing among all participants revealed a critical deficiency in occupational health surveillance and regulatory enforcement. The implications of these findings are substantial. The consistent observation of hypocalcemia and hematological alterations among exposed workers emphasizes the urgent necessity of implementing preventive and interventional strategies within the local battery industry. Improved workplace ventilation, strict enforcement of PPE usage, and mandatory periodic health assessments—including lead-level monitoring are essential to reduce exposure and prevent chronic toxicity. In addition, nutritional interventions promoting adequate calcium intake could mitigate the physiological burden of lead exposure in vulnerable worker populations. Public health initiatives and occupational safety campaigns are equally vital to raise awareness and encourage compliance with protective measures.

The strengths of this study lie in its direct comparison between exposed and non-exposed populations within a single occupational context, the inclusion of both biochemical and hematological analyses, and the consideration of post-exposure symptomatology, which together provide a comprehensive understanding of lead toxicity among battery workers. However, several limitations must be acknowledged. The sample size was modest, which may reduce statistical power and limit generalizability. The cross-sectional design precluded causal inferences regarding the temporal progression of lead-related health effects. Reliance on self-reported symptoms introduces potential reporting bias, and the exclusion of dietary assessment limited the ability to fully interpret the role of nutritional deficiencies in exacerbating hypocalcemia. Additionally, regional specificity to Peshawar restricts extrapolation of the findings to other industrial zones with differing exposure dynamics and safety regulations. Despite these limitations, this study contributes valuable evidence to the understanding of occupational lead exposure in Pakistan and highlights critical areas for policy intervention and further investigation. Future studies should adopt longitudinal designs with larger cohorts, incorporate quantitative lead-level measurement, and evaluate the interaction of nutritional, genetic, and environmental factors influencing susceptibility to lead toxicity (23). Such research would provide a more detailed mechanistic insight into exposure—response relationships and inform evidence-based strategies for worker protection and public health improvement.

CONCLUSION

This study concluded that occupational exposure to lead acid in rechargeable battery restoration workers results in marked disturbances in calcium metabolism, subtle hematological alterations, and a high frequency of clinical symptoms reflecting systemic toxicity. These outcomes affirm the detrimental physiological impact of lead on both biochemical and hematopoietic functions, emphasizing the vulnerability of individuals engaged in poorly regulated industrial environments. The findings underscore the urgent need for comprehensive occupational safety interventions, including the enforcement of protective equipment usage, regular health monitoring, and improved ventilation in workplaces. Moreover, strengthening nutritional support, particularly through calcium supplementation, may help mitigate lead-induced biochemical imbalances. Overall, this research provides valuable region-specific evidence that can inform public health strategies, guide workplace reforms, and support future longitudinal studies aimed at reducing the long-term health burden of lead exposure among industrial workers.



AUTHOR CONTRIBUTION

Author	Contribution
	Substantial Contribution to study design, analysis, acquisition of Data
Faisal Rashid	Manuscript Writing
	Has given Final Approval of the version to be published
	Substantial Contribution to study design, acquisition and interpretation of Data
Muhammad Uzair	Critical Review and Manuscript Writing
	Has given Final Approval of the version to be published
Mian Shakeel shah	Substantial Contribution to acquisition and interpretation of Data
Iviian Shakeei shan	Has given Final Approval of the version to be published
Mumtaz Ullah shah	Contributed to Data Collection and Analysis
Mumaz Onan shan	Has given Final Approval of the version to be published
Inam Ullah	Inam Ullah Contributed to Data Collection and Analysis
mam Onan	Has given Final Approval of the version to be published
Hilal Saeed	Substantial Contribution to study design and Data Analysis
illiai Saccu	Has given Final Approval of the version to be published
Abdullah	Contributed to study concept and Data collection
raudullali	Has given Final Approval of the version to be published
Zakir Ahmad*	Writing - Review & Editing, Assistance with Data Curation

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