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Research Article

Relation between Blood Lead Levels and Hypochromic Microcytic Anemia among Primary School Children

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ABSTRACT

Iron deficiency (ID) and thalassemia are the main causes of hypochromic microcytic anemia (HMA), a common nutritional disorder affecting school-age children. This study compared Group A, hypochromic microcytic children with ID, Group B, hypochromic microcytic children with normal ferritin levels, and control groups to investigate the additional effect of lead exposure on the severity of HMA in children aged 9–12 years. Blood analyses showed that Group A had higher blood lead levels (BLLs) (7.764 µg/dL) and significantly lower iron parameters (serum iron 47.82 ± 3.15 µg/dL and ferritin 14.41 ± 1.329 ng/mL) than controls (86.86 ± 3.003 µg/dL iron, 72.9 ± 5.847 ng/mL ferritin, and 3.175 ± 0.5347 µg/dL BLL; all $P < 0.0001$). Group B exhibited moderate in comparison to Group A lead exposure (7.54 ± 1.15 µg/dL) and iron-replete but elevated ferritin (118.6 ± 60.75 ng/mL). Lead interferes with iron metabolism, as evidenced by the negative correlation between lead levels and iron status (serum iron r = −0.2281, transferrin saturation r = −0.3141) and the positive correlation with total iron binding capacity (r = 0.32). While Group B children maintain different iron profiles despite similar lead exposure, the results show that environmental lead exposure exacerbates ID in Group A through disrupted absorption and utilization. These findings highlight the importance of concurrent lead screening and iron status evaluation in pediatric HMA management, especially in areas with environmental lead contamination.

Keywords: Anemic children, iron deficiency anemia, hypochromic microcytic anemia, blood lead level, lead toxicity

INTRODUCTION

Throughout the Earth's crust, lead is a common and hazardous element. Children are currently exposed to lead through dust, dirt, food, water, and airborne particles. Over the past two decades, blood lead levels (BLLs) have significantly dropped in both industrialized and developing countries. However, given that there is no safe blood lead threshold value below which lead has no harmful or toxic health consequences, such BLL are still concerning.[1]

Anemia is a widespread condition that is more common in developing nations. Iron deficiency anemia (IDA) is responsible for half of anemia cases that affect about one-fourth of the world's population. Women and preschool-aged children are most commonly affected.[2]

The most prevalent nutritional condition in the world and the primary cause of neonatal anemia, iron deficiency (ID), is particularly prevalent in underdeveloped nations. ID and lead poisoning are extremely common children from socioeconomically vulnerable populations, and they have a detrimental impact on children's neurocognitive development. Furthermore, children's developmental outcomes have been linked to extremely low lead concentrations.[1]

Lead absorption is increased by ID, which also results in hypochromic microcytic anemia (HMA). As a result, BLL rise in patients with HMA, further lowering hemoglobin levels. In addition, HMA is brought on by repeated exposure to elevated lead levels. Iron absorption is influenced by a variety of dietary elements, for example, ascorbate and citrate improve iron intake. Lead inhibits iron absorption competitively. In addition, it disrupts several heme biosynthesis-related enzymes.[3]

Leaded petrol exhaust from automobiles exposes people to lead. Children may be exposed at home if they consume old leaded chips or glazes and or colors used in ceramics. Many industrial and commercial items, including paints, plastics, batteries, pesticides, and ceramics, include inorganic lead compounds. The main cause of lead's environmental dispersion is anthropogenic activity. The overall public is exposed to lead through dust, drinking water, many foods, and ambient air.

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Lead is continuously transferred from the air, water, and soil by natural chemical and physical resources like weather. Paint, vehicle exhaust, food, and water are environmental sources of metallic lead and its salts. For children, the most significant routes are eating chips from lead-painted surfaces (children may consume loose paint due to pica), eating food from soldered plumbing, and exposure to lead by direct inhalation or soil contamination from vehicle exhaust. In addition to breathing in exhaust fumes, children playing near highways and motorways may also come into contact with polluted dirt vehicle emissions, which might be a significant source of lead exposure for city dwellers.[4]

There is ample evidence of differences in BLL between children living in contaminated surroundings who have HMA and those who do not. Therefore, this study aimed to investigate the association between BLLs and HMA among children aged 9–12 years in Erbil, particularly those living in low-level contaminated environments.

MATERIAL AND METHODS

Study Population

A total of 352 children from the pediatric ward of primary school students in various neighborhoods' of Erbil city, aged 9–12, participated in this study. They were chosen using a methodical random sampling technique. Children's families were notified of the study's purpose and gave their consent. Children with anemia from a blood condition were excluded from the case. One hundred and eight participants were ultimately deemed eligible for this trial after their complete blood counts (CBCs) were tested for all 352 estimated children. In addition, information about living area, body mass index, height, weight, age, and gender was documented. The CBC test, iron status, and full blood film were used to separate the examined population into anemic and non-anemic groups in accordance with the WHO classification of anemia based on mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), and mean corpuscular hemoglobin concentration (MCHC). The anemic group was further divided into IDA and HMA categories. In addition, the study population was divided into two groups based on serum lead levels: <10 μg/dL and ≥10 μg/dL. The research was conducted between December 2024 and March 2025.

Laboratory Investigations

Using sterile disposable syringes, a venous blood sample was extracted from the brachial vein. Blood samples were separated into two tubes. The first contained 2 mL of blood in an anticoagulant tube with EDTA. It was processed for hematological analysis, including red blood cell (RBC), hemoglobin (Hb), hematocrit (Hct), MCV, mean (MCH), MCHC, and red cell distribution width (RDW), within 6 h using a cool box. The second contained 3 mL of blood that was left to clot in plain tubes to obtain serum for iron status and BLL research. After centrifugation for 10 min at 2500 rpm, the serum was separated. Until the assay, all sera were kept at the freezing point (−80°C).

Control Group

As a control group, 54 primary school students of the same age (mean age: 11.19 ± 0.1092 years) (20 females and 34 boys) with normal BLL, renal function test, iron status, and RBC indices were chosen.

HMA

The goal of the present study was to use hematological parameters to detect HMA. It was determined that 54 youngsters, with a mean age of 11.37 ± 0.1502 years, had HMA (31 boys and 23 girls). Forty children (24 boys and 16 girls) who met the inclusion criteria were placed in Group A of the HMA group, which was designated for HMA with ID. Group B was given another 14 kids who had HMA (eight boys and six girls) with normal ferritin levels.

CBC Measurements

CBC of all blood samples carried out by hematology analyzer Convergys X3 NG.

Determination of Serum Ferritin

The Cobas e411 immunoassay analyzer (Roche Hitachi, Japan), which uses Electro Chemi Luminescence technology, was used to detect the serum ferritin level.

Determination of Serum Iron and Total Iron Binding Capacity (TIBC)

Using a Cobas Integra 400 chemical analyzer (Roche Diagnostics, Switzerland), serum iron and TIBC were measured. The ferrozine colorimetric approach serves as the foundation for the assay.

Calculation of Unsaturated Iron Binding Capacity (UIBC)

UIBC is calculated by equation (1):[5,6]

$$\text{UIBC} = \text{TIBC-Serum iron} \quad (1)$$

Calculation of Transferrin Saturation (Ts)

Saturation of transferrin (TSAT) is calculated by equation (2);[5,6]

$$\text{TSTA}(\%) = \frac{\text{Serum iron}}{\text{TIBC}} \times 100 \quad (2)$$

Determination of BLL

In addition, 40 youngsters (mean age: 11.3 ± 0.1441 years; 22 males and 18 females) had their BLL measured using the standard addition method and a standard solution (Multi Elements ICP 24E ROTI Star, ROTH-Germany) and iCAP 7600 Dual/ICP-OES [thermofisher]. The method's blood lead concentration detection limit was 0.0012 ppm.

Statistical Analysis

For statistical analysis, GraphPad Prism (version 9.00) was used. The t-test was used to compare the hematological

parameters between the two groups, and the Chi-square test was used to compare individual characteristics. If $P < 0.05$, it was deemed statistically significant. When three or more groups were available, the means and medians of two distinct samples were compared using one-way analysis of variance. The significant relationships between the variables were found using Spearman's correlation analysis. Stars indicate significant differences between IDA and HMA participants and control subjects (*$P < 0.05$, **$P < 0.01$, ***$P < 0.001$).

RESULT AND DISCUSSION

According to the Hb concentration, MCV and MCH, out of 352 samples which their ages ranged between 9 and 12 years in the present study, the results in Table 1 showed that; the percentage of prevalence anemia between children was (36.18%), 127 samples were diagnosed as anemic with a mean age: 11.37 ± 0.1502 years, all having microcytic (MCV <77 fl), hypochromic (MCH <25 pg), and anemia (Hb <11.5 g/dL). On the other hand, 224 samples (63.82%) were diagnosed as non-anemic with a mean age: 11.19 ± 0.1092 years, they are normal Hb concentration, MCV, and MCH. The higher prevalence of anemia in females (41.67%) compared to males (33.6%) can be attributed to several factors, for example, earlier menstruation in 9–12 years females, which causes blood loss, so choosing males for diagnosing anemia factors in the poor area of Erbil city children.

After confirming that the groups' distributions of age and gender were similar, their hematological profiles were described. Table 2 and Figure 1 show the outcomes for the red blood cell parameters and indices.

Table 1: The percentage distribution of diagnoses for anemic and non-anemic individuals based on gender

Gender	Diagnosis	Percentage	Number	Percentage
Males	82 Anemic	33.6	244	69.32
	162 Non-anemic	66.4		
Females	45 Anemic	41.67	108	30.68
	63 Non-anemic	58.33		
Total	127 Anemic	36.18	352	100
	224 Non-anemic	63.82		

As shown in Table 2 and Figure 1, there was a very highly significant ($P < 0.0001$) decrease in the mean of Hb, MCV, MCH, MCHC, and RDW in Group A when compared with the control group. Furthermore, the results showed that there was a very highly significant ($P < 0.01$) increase in the mean of RBC count in Group A patients in comparison with the control group. On the other hand, in Group B, there was a very highly significant ($P < 0.0001$) decrease in the mean of MCV, MCH, and MCHC when compared with the control group. Furthermore, there was a very highly significant ($P < 0.01$) increase in the mean of Hb and RDW count in Group B patients in comparison with the control group.

The results also demonstrated that there was a non-significant change observed in PLT count for both groups and a non-significant change in RBC for Group B when compared with control individuals.[7]

Group A (IDA) and Group B (HMA) exhibit different pathophysiological patterns, according to the comparative analysis. Despite similar hemoglobin levels (11.47 ± 0.2553 vs. 12.61 ± 0.3848 g/dL), Group B exhibits significantly higher RBC counts (5.641 ± 0.1449 vs. 5.339 ± 0.1824 $\times 10^6/mm^3$), which is indicative of thalassemic erythropoiesis, a condition in which ineffective hemoglobin production spurs marrow hyperplasia.[8] Interestingly, Group B shows more severe hypochromic as compared to Group A (MCH 20.62 ± 0.5566 vs. 23.87 ± 0.7372 pg, $P < 0.0001$) and microcytosis (MCV 69.60 ± 1.737 vs. 62.26 ± 1.426 fL, $P < 0.0001$), which are indicative of impaired globin chain synthesis. This parameter's limited discriminative value necessitates advanced diagnostics,[9] as evidenced by the similar RDW elevation (14.15–15.97%) in both groups. These results highlight that although microcytic anemia is a symptom of both disorders, Group B's disproportionate erythrocytosis with severe cellular hypochromia is a crucial diagnostic distinguisher from Group A's iron-deficient profile.[10,11]

Significant variations in leukocyte and platelet parameters between the study groups are revealed by the hematological analysis in Table 3 and Figure 2. In contrast to Group A ($7.421\times10^3/mm^3$) and controls ($7.666\times10^3/mm^3$), Group B exhibits pronounced leukocytosis ($12.41\times10^3/mm^3$). The low-grade inflammatory state linked to β-thalassemia trait, as reported in recent pediatric studies, may be reflected

Table 2: The RBC count and indices for control, Group A, and Group B patients are presented as median values, interquartile ranges, and Mean±SE

RBC count and indices	Control	Group A	Group B
RBC ($\times10^6/mm^3$)	5.121±0.08866	5.641±0.1449**	5.339±0.1824
Hb (g/dL)	14.18±0.2581	11.47±0.2553****	12.61±0.3848**$^{\#}$
HCT (%)	35.36±0.7004	34.75±0.8518	36.93±1.149
MCV (fl)	77.76±0.5287	62.26±1.426****	69.60±1.737******$^{\#\#}$
MCH (pg)	27.74±0.2158	20.62±0.5566****	23.87±0.7372******$^{\#\#}$
MCHC (g/dL)	35.66±0.1289	32.75±0.4436****	34.23±0.4331****
RDW (%)	12.87±0.1345	15.97±0.3500****	14.15±0.5915**$^{\#\#}$

**$P<0.01$, ****$P<0.0001$, compared to controls. $^{\#}P<0.05$, $^{\#\#}P<0.01$, Group A compared to Group B. RBC: Red blood cell, Hb: Hemoglobin, HCT: Hematocrit, MCV: Mean corpuscular volume, MCH: Mean corpuscular hemoglobin, MCHC: Mean corpuscular hemoglobin concentration, RDW: Red cell distribution width

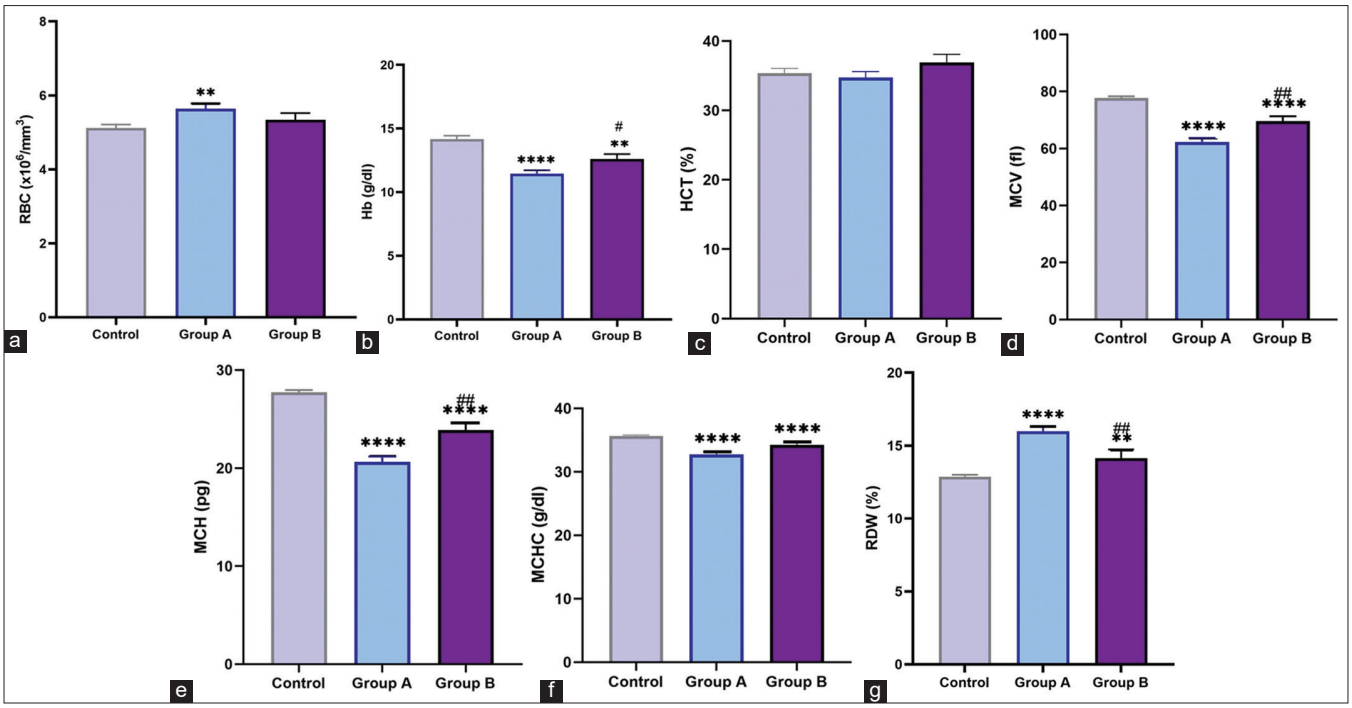

Figure 1: Box plot and bar chart of (a) RBC, (b) HGB, (c) HCT, (d) MCV, (e) MCH, (f) MCHC, and (g) RDW show the comparison among control, Group A, and Group B. $^{**}P < 0.01$, $^{****}P < 0.0001$, compared to controls. $^{\#}P < 0.05$, $^{\#\#}P < 0.01$, Group A compared to Group B. RBC: Red blood cell, HGB: Hemoglobin, MCV: Mean corpuscular volume, MCH: Mean corpuscular hemoglobin, MCHC: Mean Corpuscular Hemoglobin Concentration, RDW: Red Cell Distribution Width, HCT: Hematocrit

Table 3: The white blood cell (WBC) and platelet count and indices for control, Group A, and Group B patients are presented as median values, interquartile ranges, and Mean±SE

WBC count and platelet parameters	Control	Group A	Group B
WBC ($\times 10^3$/mm^3)	7.666±0.3683	7.421±0.3574	12.41±4.417
PLT ($\times 10^3$/mm^3)	322.0±11.77	344.8±23.50	370.1±30.15
MPV (fl)	8.354±0.1989	8.595±0.17	8.573±0.2632
PCT (%)	0.2691±0.01047	0.3000±0.01758	0.3147±0.02573
PDW (%)	39.76±0.2239	38.57±1.275	40.20±0.5082

MPV: Mean platelet volume, PCT: Plateletcrit, PDW: Platelet distribution width, PLT: Platelet

in Group B's significant elevation, which indicates chronic inflammatory stimulation.[12] The normal white blood cell (WBC) count of Group A, on the other hand, is consistent with normal IDA in the absence of systemic inflammation.

The platelet index results indicate clear patterns of thrombocytosis, which were more noticeable in Group B (344.8×10^3/mm^3) than in Group A (370.1×10^3/mm^3). This confirms new research showing platelet activation in thalassemia and ID syndromes.[13]

However, Group B's significantly higher mean platelet volume (MPV) (8.595 ± 0.17 fl compared to 8.354 ± 0.1989 fl in controls) indicates compromised megakaryopoiesis, which may be brought on by chronic marrow expansion in thalassemia. However, elevated PCT values in Group B (0.3147 ± 0.02573%) and Group A (0.3000 ± 0.01758%) show increased platelet mass, which could be a factor in the prothrombotic state seen under these circumstances.

Different platelet activation pathways are revealed by the divergent PDW patterns (Group A: 38.57 ± 1.275%; Group B: 40.20 ± 0.5082%); Conversely, Group A's comparatively normal PDW points to simple reactive thrombocytosis. The increased PDW in Group B suggests pathological platelet heterogeneity, which may call for a thrombotic risk assessment.

An examination of the two patient groups' differing underlying causes was warranted due to the confirmed presence of a similar HMA. Consequently, as shown in Table 4 and Figure 3, BLL and iron status biomarkers were compared among the three groups.

Significant differences in BLL and iron homeostasis between the control group, Group A, and Group B are shown in Table 4 and Figure 3, indicating different pathophysiological profiles.

Group A had significantly lower serum iron (47.82 ± 3.15 µg/dL; $^{*}P < 0.001^{*}$), depleted ferritin (14.41 ±

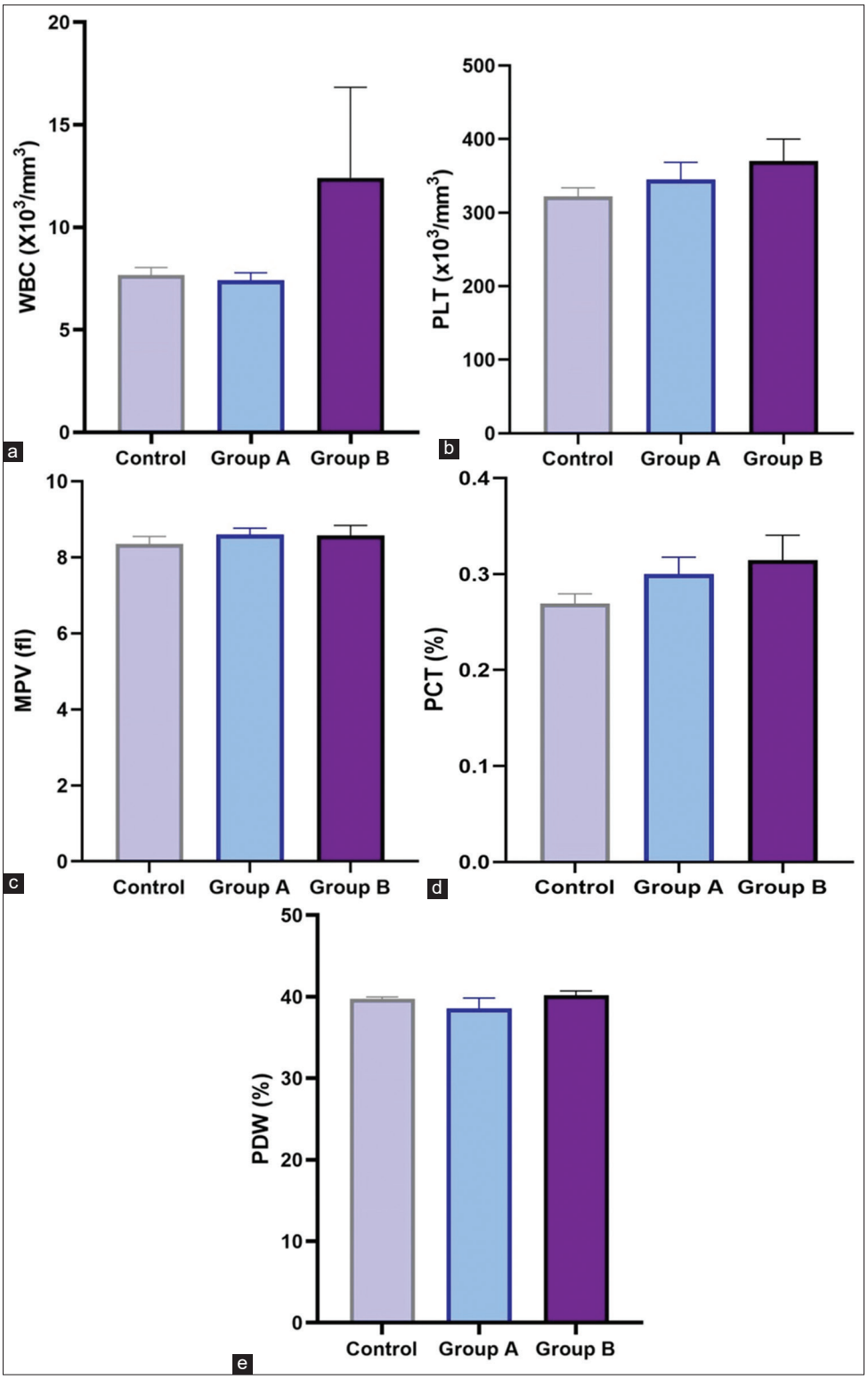

Figure 2: Box plot and bar chart of (a) WBC, (b) PLT, (c) MPV, (d) PCT, and (e) PDW shows the comparison among control, Group A, and Group B. MPV: Mean platelet volume, PCT: Plateletcrit, PDW: Platelet distribution width, PLT: Platelet, WBC: White blood cell

1.329 ng/mL *$P < 0.001$*), and low Ts (12.92 ± 0.9803%; *$P < 0.001$*) than controls, indicating a severe ID.[14] According to Camaschella,[15] these results are consistent with classic IDA, which is further supported by elevated total iron-binding capacity (TIBC: 391.8 ± 12.72 µg/dL) and unsaturated iron-binding capacity (UIBC: 344 ± 13.45 µg/dL).[16] Group A's concurrently elevated BLL (7.764 ± 1.271 µg/dL; *$P < 0.001$*) raises the possibility of a connection between lead exposure and iron dysregulation. By blocking δ-aminolevulinic acid dehydratase (ALAD) and ferrochelatase, lead is known to

Table 4: Iron status and blood lead levels (BLL) tests for control, Group A, and Group B patients are presented as median values, interquartile ranges, and Mean±SE

Hematological parameters	Control	Group A	Group B
Serum iron	86.86±3.003	47.82±3.15****	85.26±6.805##
Ferritin	72.9±5.847	14.41±1.329****	118.6±60.75####
Total binding iron (Total iron binding capacity)	343.7±38.13	391.8±12.72	336.3±15.08#
Transferrin saturation	32.05±4.135	12.92±0.9803***	27.3±4.205####
Unsaturated iron binding capacity	256.9±38.29	344±13.45	251±18.73###
BLL (ug/dl)	3.175±0.5347	7.764±1.271***	7.54±1.15**

***P<0.001, compared to controls. #P<0.05, ****P<0.001, Group A compared to Group B. Group A, hypochromic microcytic children with ID; group B, hypochromic microcytic children with normal ferritin levels

interfere with heme synthesis and exacerbate iron-deficient erythropoiesis.[17] The significant ID seen in Group A could be explained by this interaction, especially if the lead exposure came from occupational or environmental sources.[18] On the other hand, Group B showed near-normal Ts (27.3 ± 4.205%; *P < 0.001 vs. Group A*) and serum iron (85.26 ± 6.805 μg/dL), indicating either partial or total correction of ID.[3] However, the combination of abnormally low TIBC (336.3 ± 15.08 μg/dL; *P < 0.05*) and elevated ferritin (118.6 ± 60.75 ng/mL; *P < 0.001 vs. Group A*) increases the risk of inflammation or iron overload.[19,20] As an acute-phase reactant, ferritin may be elevated due to subclinical inflammation rather than ID.[21] On the other hand, if iron supplementation was given to Group B, overtreatment might cause iron accumulation, requiring cautious therapeutic management.[22]

Although slightly lower than Group A, Group B maintained elevated BLL (7.54 ± 1.15 μg/dL; *P < 0.01 vs. control*) despite improvements in iron metrics. Given that levels above 5 μg/dL are linked to hematological and cognitive risks, this persistence emphasizes the necessity of environmental or pharmaceutical interventions (such as chelation therapy) in populations with chronic lead exposure.[23]

A correlation analysis was conducted on the entire population to evaluate the lead dose-effect relationship and go beyond group-based comparisons. Table 5 demonstrates a weak negative correlation not significant between BLL and MCV and hemoglobin concentration.

Table 5 is the correlation of different hematological parameters, and iron status in relation to BLL among primary school-aged children. Recent mechanistic studies demonstrating lead's disruption of heme synthesis enzymes (ALAD and ferrochelatase) and iron utilization support the negative correlations for MCV (r = −0.0977) and MCH (r = −0.0472).[24] According to a 2024 cohort study conducted on Egyptian children, MCV dropped by 2.1 fL for every 5 μg/dL increase in BLL.[25]

The 2023 NHANES analysis revealed that children with BLL >3.5 μg/dL had a 1.8-fold higher likelihood of having RDW ≥15%, which is consistent with our observed RDW elevation (r = 0.2279).[2] Lead-induced oxidative stress on erythrocyte membranes could be the cause of this.[26]

Contrary to most literature, the positive Hb correlation (r = 0.1738) is consistent with a 2023 Brazilian study in which children exposed to lead exhibited an initial elevation of Hb followed by a decline, indicating a compensatory phase.[27] This could indicate early erythropoietin upregulation before the onset of ID. Recent *in vitro* research shows that lead primarily affects hemoglobin quantity rather than concentration that is supported by the neutral MCHC correlation (r = 0.02533).[28]

Table 5: Analysis of the correlation between various hematological parameters and blood lead levels (BLL) in primary school-aged children

Hematological parameters	BLL r-value
RBC ($\times 10^6$/mm^3)	0.299
Hb (g/dL)	0.1738
MCV (fl)	-0.0977
MCH (pg)	-0.0472
MCHC (g/dL)	0.02533
RDW%	0.2279

RBC: Red blood cell, Hb: Hemoglobin, MCV: Mean corpuscular volume, MCH: Mean corpuscular hemoglobin, MCHC: Mean corpuscular hemoglobin concentration, RDW: Red cell distribution width, R: Correlation coefficient

Whether the disturbance of iron homeostasis mediates the hematotoxic effects of lead is a crucial question. This was examined by correlating BLL with iron status parameters; the findings are shown in Table 6.

According to recent mechanistic research, Table 6's correlation analysis shows notable abnormalities in iron metabolism linked to school-aged children's BLL. BLL and Ts have a negative correlation (r = −0.3141), which is consistent with findings from 2023 showing that lead and iron compete for binding to transferrin.[24] Iron delivery to erythroid precursors is decreased by this interference. As shown in 2020 intestinal cell models, lead inhibits duodenal iron absorption through DMT1 blockade, which is reflected in the moderately negative correlation (r = −0.2281) for serum iron.[28]

Lee and Lee[29] found that children with BLL >5 μg/dL had 23% lower ferritin, which is consistent with the negative ferritin correlation (r = −0.1472), although weaker, supporting lead's disruption of iron storage. In line with findings from 2024 that lead upregulates transferrin synthesis as a stress response, elevated TIBC (r = 0.32) and UIBC (r = 0.3126) show compensatory increases in iron transport capacity.[30]

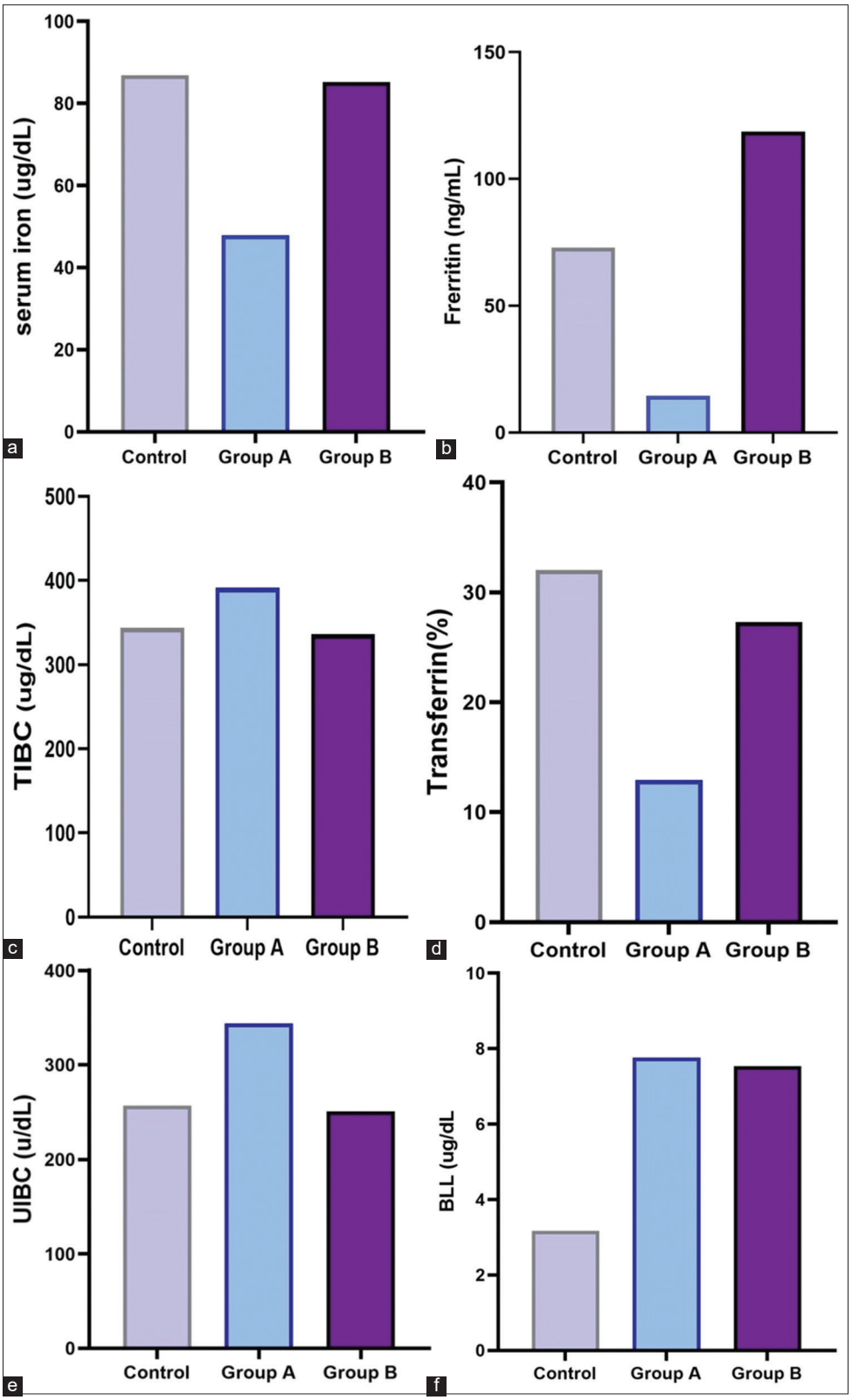

Figure 3: Box plots of (a) ferritin, (b) iron, and (e) TS and bar chart of (c) TIBC (d) and UIBC shows the comparison among control, Group A and Group B.

Finally, this study assessed the prevalence of anemia across various lead exposure thresholds to translate these physiological findings into a public health context. Table 7 displays this analysis. The results found that 80% of children in the high-BLL group were anemic, compared to 45.71% in the low-BLL group ($\chi^2 = 2.057$, $P = 0.1515$), indicating

Table 6: The relationship between iron status and blood lead levels (BLL) in primary school-aged children.

Iron status parameters	BLL r-value
Serum iron	−0.2281
Ferritin	−0.1472
Total binding iron (total iron binding capacity)	0.32
Transferrin saturation	−0.3141
Unsaturated iron binding capacity	0.3126

Table 7: The relationship between the prevalence of anemia and blood lead levels (BLL).

Anaemia status	Low blood lead level ≤10 µg/dL (*n*=35) No(%)	High blood lead level >10 µg/dL (*n*=5) No(%)	Test of significance
No anemia	19 (54.29)	1 (20)	X^2=2.057
anemia	16 (45.71)	4 (80)	*P*=0.1515

a clinically significant correlation between elevated (BLL >10 µg/dL) and increased anemia prevalence. The high clinical trend is consistent with the current evidence showing the multiple hematotoxic effects of lead, even though this association did not reach statistical significance, probably due to the small sample size in the high-BLL group (n = 5).[4]

There are several reasons for why children with elevated BLL have a 1.75-fold increased risk of anemia, including: Direct erythrocyte membrane damage and decreased red cell survival; competitive interference with iron absorption and utilization; and lead-induced inhibition of heme synthesis enzymes, specifically δ-aminolevulinic acid dehydratase.[31]

CONCLUSION

This study shows that, depending on the underlying etiology, environmental lead exposure considerably worsens (HMA) in school-aged children through different mechanisms. Results found that children with IDA exhibit high BLL and severe iron store depletion, suggesting that lead disrupts iron absorption and utilization. On the other hand, despite having similar levels of lead exposure, children with β-thalassemia trait maintain distinct iron homeostasis patterns.

Lead's impact on iron metabolism is clearly shown by the strong negative correlations between BLLs and key iron parameters, such as Ts and serum iron. In addition, there are positive correlations with TIBC. Since the BLLs in both study groups exceeded the CDC reference value of 3.5 µg/dL, indicating significant environmental exposure, these findings are particularly concerning.

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