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Serum Leads Level, Some Haematological Parameters and Cd4+ Cells Count among Fueling Station Attendants in Jigawa State, North Western Nigeria

Muhammad Alhassan[1], Bilkisu Magaji[1], Abdullahi Abubakar[1], Mustapha Muhammad[1], Dauad Rabiu[1], Shamsu Adamu[1], Bashir Abdulrazak[1], Garba, N.[2]

[1]*Department of medical laboratory science, college of health sciences and Technology Jahun P.M.B. 3011, Jigawa State, Nigeria.*
[2]*Department of medical laboratory Science, Bayero University PMB3011 Kano Nigeria*

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***Corresponding Author:** Muhammad Alhassan

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Abstract **Original Research**

Background: Occupational exposure to lead remains a significant public health concern, particularly among fueling station attendants who are routinely exposed to petroleum products. Chronic lead exposure has been associated with alterations in haematological indices and impairment of immune function.

Objective: This study evaluated the effect of blood lead levels (BLL) on selected haematological parameters and CD4+ T-cell counts among fueling station attendants in Jigawa State, north-western Nigeria.

Methods: A comparative cross-sectional study was conducted among 378 apparently healthy adults aged 18 years and above, recruited using purposive sampling. Venous blood samples were collected for determination of full blood count (FBC), blood lead levels, and CD4+ T-cell counts. Haematological parameters were analyzed using a Sysmex KN-21 automated haematology analyzer, CD4+ counts were determined using the Partec CyFlow system, and blood lead levels were measured using a Shimadzu 6800 Atomic Absorption Spectrophotometer. Data were expressed as mean ± standard deviation. Group comparisons and Spearman's correlation analysis were performed to evaluate associations between BLL and the measured variables, with statistical significance set at $p < 0.05$.

Results: Blood lead levels were significantly higher among fueling station attendants than in the control group ($p = 0.001$). Total white blood cell count, mixed cell percentage (MXD%), lymphocyte percentage (LYM%), and red cell distribution width (RDW) were significantly elevated among exposed participants ($p < 0.05$). In contrast, neutrophil percentage (NEUT%), red blood cell count (RBC), haemoglobin (Hb), haematocrit (HCT), mean corpuscular haemoglobin concentration (MCHC), mean corpuscular volume (MCV), and mean corpuscular haemoglobin (MCH) were significantly reduced ($p < 0.05$). Blood lead levels showed significant positive correlations with lymphocyte and neutrophil counts, while a significant negative correlation was observed with MXD%. Haemoglobin, haematocrit, mean corpuscular volume, and neutrophil counts declined significantly with increasing blood lead levels, whereas lymphocyte counts increased significantly. CD4+ T-cell counts were also significantly lower among fueling station attendants than among controls ($p < 0.05$).

Conclusion: Elevated blood lead levels among fueling station attendants are associated with significant alterations in haematological indices and reduced CD4+ T-cell counts, suggesting that chronic occupational lead exposure may adversely affect both haematopoietic and immune functions. Routine biological monitoring and implementation of effective occupational health and safety measures are recommended to minimize lead exposure in this population.

Keywords: Blood lead level, fueling station attendants, haematological parameters, CD4+ T cells, occupational exposure, Nigeria.

INTRODUCTION

Lead is a highly toxic heavy metal that exists naturally as four stable isotopes: ^{204}Pb, ^{206}Pb, ^{207}Pb, and ^{208}Pb (Kabata-Pendias & Mukherjee, 2007). As a major constituent of leaded gasoline, lead enters the human body primarily through inhalation of contaminated air or ingestion via the gastrointestinal tract. Once absorbed, it is transported in the bloodstream and distributed to soft tissues before being deposited in the bones (Patrick, 2009). Absorption occurs mainly through the mucosal surfaces of the respiratory and gastrointestinal tracts. Following entry into the circulation, lead is initially distributed to soft tissues and subsequently accumulates in the skeletal system. Approximately 10–20% of the body's lead burden is retained in soft tissues, whereas 70–85% is stored in bones, which serve as the principal reservoir for long-term lead accumulation (Ani *et al.,* 2007).

Lead exerts toxic effects on multiple organ systems, including the renal, cardiovascular, reproductive, nervous, haematopoietic, and immune systems. It is also recognized as a mutagen, and the severity of its biological effects depends largely on the concentration and duration of exposure (Nilima *et al.,* 2011). One of the major mechanisms of lead toxicity is the disruption of haem biosynthesis through dose-dependent inhibition of key enzymes involved in the pathway. These enzymes include δ-aminolevulinic acid synthase (ALAS), a mitochondrial enzyme responsible for the synthesis of δ-aminolevulinic acid (ALA); δ-aminolevulinic acid dehydratase (ALAD), a cytosolic enzyme that catalyses the conversion of ALA to porphobilinogen; and ferrochelatase, a mitochondrial enzyme that inserts ferrous iron into protoporphyrin IX to form haem (Piomelli, 2002).

In addition to impairing haem synthesis, lead shortens the lifespan of circulating erythrocytes by increasing the fragility of their cell membranes, thereby contributing to the development of anaemia (Guidotti *et al.,* 2008). Lead absorbed from the gastrointestinal tract binds to plasma proteins such as ceruloplasmin and transferrin, disrupting normal iron transport to erythroblasts and further impairing erythropoiesis (Keramati *et al.,* 2009). Chronic lead exposure may therefore result not only in anaemia but also in characteristic haematological abnormalities, including basophilic stippling of erythrocytes and mild-to-moderate haemolytic anaemia. Consequently, examination of red blood cell morphology remains an important component in the diagnosis of lead poisoning (Orisakwe, 2009). Furthermore, exposure to environmental lead has been associated with immune dysfunction, particularly among young children, highlighting its widespread impact on human health (Li *et al.,* 2005).

MATERIALS AND METHODS

Study Area

Jigawa State is situated in North-Western geo political zone of Nigeria, between latitudes11.00^{o} N to 13.00^{o}N and longitudes 8.00^{o}E to 10.15^{o}E. Kano and Katsina States border Jigawa to the west, Bauchi State to the east and Yobe State to the north. Jigawa shares an international border with the Republic of Niger, which is a unique opportunity for cross-border trading activities. Government readily took advantage of this by initiating and establishing a free trade zone at the border town of Maigatari in Jigawa State. The state has a total land area of approximately 22,410 square kilometers. Its topography is characterized by undulating land, with sand dunes of various sizes spanning several kilometers in parts of the state.

Study Population

Homogeneous purposive sampling was used to select 378 apparently healthy fueling station attendants, >18 years who offered written inform consent to participate in this research and met up with inclusion criteria of working in fueling station within Jigawa State North-Western Nigeria. These constituted the subjects for this study while 150 apparently healthy >18 years, non-exposed individuals were recruited as controls.

Study Design

This was an observational case control study designed to investigate the serum lead level, some haematological parameters and oxidative stress biomarkers of 378, >18 years, apparently healthy fueling station attendants in Jigawa, North-Western Nigeria, using 150, >18 years, apparently healthy non-fueling station attendants as controls.

Sample Size Estimation

The sample size was calculated using the following formular by Charan and Biswas (2013).

$$\text{Sample size} = \frac{r+1\ SD^2\ (Z_\beta + Z\alpha_{/2})}{1 \qquad d2}$$

r = ratio of controls to cases.

SD^2= standard deviation of previously published study.

Z_β = Standard normal variate for power set at 90% the value is 1.28

$Z_{1-\alpha/2}$ = Standard normal variate at 5% type 1 error (P< 0.05) it is (1.96)

d^2= expected mean difference between case and control from previously published study.

Attrition rate of 10% will be added.

Thus,

Sample size =?

r =1

SD^2= 15.51 (Ibeh *et al*., 2016).

Z_β =1.28

$Z_{1-\alpha/2}$ == (1.96)

d^2 =3.83 (Ibeh *et al*., 2016).

$$= \frac{1+1\ (15.15)^2\ (1.28+1.96)^2}{1 \qquad (3.83)^2} = \frac{240.56 \times 10.94}{14.67} = 344$$

Attrition rate of 10% = 344 x 10% = 34.4 = 34.4 +344 = 378.

Subjects Selection

Inclusion criteria for Subjects

Only adults (>18years) participated in this research and included subjects who work in fueling station and who gave written consent to participate in the study. They were free from any disease that could affect haematological variables or oxidative stress biomarkers. Subjects to be selected for this study, must be HIV sero-negative and should not have been transfused with blood or its products in the last two month.

Exclusion criteria for subjects

Children and individuals <18 years, chronic smokers (smoking >20 cigarettes per day), subjects that refused to give written consent, those who were not attendants in fueling station as well as those with history of diseases that affect oxidative stress biomarkers or haematological variables were excluded. HIV sero-positive subjects and those transfused with blood or its product in the last 2 month were also excluded.

STUDY INSTRUMENT

Questionnaire

Questionnaire was administered to each subject to collect bio-data such as age, gender, duration of exposure and history of other diseases.

Ethical consideration

This study was approved by the Ethical Committee of Ministry of Health, Jigawa State **(REFERENCE NUMBER: MOH/SEC/3/S/640/1/26)** and written informed consent was obtained from all participants in this study (copy of approval attached in the appendix section) under the following agreements:

1. I ensured that the information obtained from the subjects was kept confidential.

2. All the investigations were offered at no cost to the subjects.

3. Participation was voluntary and the

recommendation communicated to participants at the end of the research.

Sample Collection and Processing

Seven milliliters (7ml) of blood sample were aseptically collected from each subject via venipuncture. Exactly 4 ml of blood was transferred into sample bottle containing EDTA as an anticoagulant, for haematological analysis. Another 3 ml was transferred into plain sample container, allowed to clot. Serum was obtained after centrifuging the clotted blood samples at 4500 × *g* for 15 min and was stored at 4^{o}C (Cheesbrough, 2006).

METHODS

Determination of Serum Lead Level by Atomic Absorption Spectroscopy (Aas) Using Model: Shimadzu 6800 Machine **(**Subramanian, 1987).

Principle:

AAS is based on the fact that free atoms absorb light at wavelengths characteristic of the element of interest. The amount of light absorbed can be correlated in a linear fashion to the concentration of the analyte in the sample. To conduct an AAS measurement, the lead-containing sample must first be processed by the instrument so as to generate ground-state atoms as a vapor within the light path of the instrument (atomization). The electrons of the atoms in the atomizer can be promoted to higher orbitals (excited state) for a short period of time (nano seconds) by absorbing a defined quantity of energy (radiation energy) at a given wavelength, which is specific to a particular electron. The amount of light absorbed at this wavelength will increase as the number of atoms of the selected element in the light path increases, and is proportional to the concentration of absorbing atoms. It requires standards with known analyte content, to establish the relation between the measured absorbance and the analyte concentration and relies therefore on the Beer-Lambert law. Each wavelength corresponds to only one element.

Sample preparation

1. Clean beakers were labeled from one to the last number of the samples and samples were labeled from one to the last number.
2. Two milliliters (2 ml) of serum were dispensed in to each corresponding beaker.
3. Exactly 15 ml of concentrated nitric acid were added.
4. The beakers were placed on the hot plate at 100^{0}C for 45 minutes.
5. They were allowed to cool for 20 minutes.
6. The mixture from each beaker was filtered with Whatman filter paper no. 1.
7. Distilled water was added to each beaker to make it content 60 ml.
8. Atomic Absorption Spectrophotometer (AAS6800**)** was used to estimate the serum lead level from each beaker

FULL BLOOD COUNT

Method: automation (Using Auto Haematology Analyser Sysmex KS-21-N)

Principle:

The blood counting machine aspirates a small amount of the blood specimen through narrow tubing. The blood sample is diluted in saline, a good conductor of electrical current, and the cells are pulled through an aperture by creating a vacuum. Electrical current is applied to the external electrode and the internal electrode. Electrical resistance or impedance occurs as the cells pass through the aperture causing a change in voltage. This change in voltage generates a pulse. The number of pulses is proportional to the number of cells counted. The size of the voltage pulse is also directly proportional to the volume or size of the cell.

Procedure:

1. Blood sample was gently mixed using a rotary shaker for 5 minutes.

2. Analysis button on the monitor of the device was pressed and patient's ID and other data were entered.
3. Sample bottle was placed under the 'sample needle' and start key was pressed.
4. The instrument aspirated the sample and analysed it.

Result:

1. The samples were automatically analysed and the results displayed on the monitor
2. The results were then printed out with the in-built printer.

Blood Film Preparation and Examination (Cheesebrough, 2006).

Blood **Smear Preparation**

1. A cleaned, grease free slide was placed on a flat surface.
2. A drop of well mix EDTA blood specimen was placed on the slide, 1cm away from the edge with plain capillary tube.
3. A cleaned, smooth edge spreader was placed in front of the drop of blood.
4. It was drawn back to touch the drop of blood.
5. The blood was allowed to extend along the edge of the spreader at an angle of about 45^0.
6. The spreader was moved forward with constant speed. A thin blood film, about 40-50 mm in length was obtained.
7. It was labeled with a sharp pointed pencil.
8. It was allowed to air dry, protected from dust and flies.

Principle of Leishman's staining:

In this method, staining reaction take place at a neutral pH. Buffered water at pH 6.8 is used to bring the pH of blood (7.4) to neutral pH i.e., pH 7.0. At this pH, the basic part of the stain, methylene blue, stains the acidic part of the cell i.e., the nucleus while the acidic part of the stain, eosin, stains the basic part of the cell i.e., cytoplasm.

Staining procedure:

1. Air dried blood film was flooded with Leishman's stain.
2. It was allowed to stay for two minutes.
3. After 2 minutes, the stain was diluted with buffered distilled water, pH6.8.
4. It was allowed to stay for 7 min.
5. It was washed for two minutes in a stream of buffered water until it has acquired a pinkish tinge color.
6. The back of the slide was wiped clean.
7. It was placed upright on a draining rack to air dry.
8. Dried, stained blood films were examined microscopically using ×100 objective lens and morphological changes were recorded.

CD_4^+T-LYMPHOCYTE COUNTS

Method: FLOW CYTOMETRY (Greve *et al*., 2003)

Source of Kit: Sysmex Partec, Gorlitz, Germany.

Principles:

Individual cells stained with fluorescent labels dyes are suspended in physiological solution and introduced under a slight pressure through a flow chamber into the center of a stream of cell-free sheath fluid. The light scattered by the individual particles and the fluorescence emitted by the cells is used for analysis and sorting of the cells based on the fluorescent antibody directed against a specific surface marker. This combination of scattered and

fluorescence light is picked up by the detectors in the flow cytometer. These detectors then produce electronic signals that are proportional to the optical signals received.

The visible light undergoes deflection based on the size and internal structures of the cell. Forward Scatter (FSC) correlates with the cell volume. Side Scatter (SSC) depends on the inner complexity of the cells (i.e., shape of the nucleus, the amount and type of cytoplasmic granules or the membrane roughness). The fluorescence emitted by the cell depends upon the florescence tagged specific monoclonal antibodies against the cell surface markers. The data collected on each cell are stored in the computer. The data are then processed and analyzed to provide information about cell populations within the sample.

Procedure:

1. Twenty microliters (20µl) of monoclonal antibodies were dispensed in to cleaned test tube.
2. Twenty microliters (20µl) of EDTA-anticoagulated blood were added.
3. After 15 min of incubation, 800µl of no lyse dilution buffer was added.
4. It was mixed gently.
5. The sample tube was attached to the CyFlow counter for automated counting.
6. Same procedure was applied to all samples.
7. Results were printed with in-build printer.

RESULT

The results obtained from this study are presented in Tables 4.1 to 4.4.

Table 4.1 presents the serum lead levels of fuelling station attendants and the control group. The mean serum lead level of the fuelling station attendants was 11.4338 ± 3.3568 µg/dL, which was significantly higher ($p = 0.001$) than that of the control group (7.8380 ± 3.9838 µg/dL).

Table 4.2 presents the red blood cell-related haematological parameters of the study subjects and controls. The mean values of red blood cell count (4.25 ± 1.08 × 10^9/L), haemoglobin concentration (13.19 ± 8.59 g/dL), haematocrit (43.28 ± 12.45%), mean cell volume (79.01 ± 9.14 fL), mean cell haemoglobin (26.49 ± 10.09 pg), and mean cell haemoglobin concentration (34.54 ± 3.42 g/dL) were significantly lower ($p < 0.05$) among the fuelling station attendants than among the controls, whose corresponding values were 5.20 ± 1.36 × 10^9/L, 16.01 ± 1.01 g/dL, 45.21 ± 3.27%, 88.64 ± 11.48 fL, 32.17 ± 1.45 pg, and 35.21 ± 2.67 g/dL, respectively. However, red cell distribution width-standard deviation (RDW-SD) (46.99 ± 3.80 fL) and red cell distribution width-coefficient of variation (RDW-CV) (14.72 ± 1.23%) were significantly higher ($p < 0.05$) in the study subjects than in the controls (40.93 ± 11.80 fL and 14.05 ± 1.11%, respectively).

Table 4.3 presents the leukocyte-related parameters of the study subjects and controls. The mean total white blood cell count (5.24 ± 2.15 × 10^9/L), percentage lymphocytes (42.36 ± 14.39%), percentage mixed cells (18.69 ± 14.32%), absolute lymphocyte count (4.10 ± 2.23 × 10^9/L), and absolute mixed cell count (4.92 ± 2.61 × 10^9/L) were significantly higher ($p < 0.05$) among the study subjects than among the controls, whose corresponding values were 4.33 ± 2.11 × 10^9/L, 25.85 ± 10.02%, 14.38 ± 5.73%, 2.34 ± 1.26 × 10^9/L, and 3.71 ± 8.83 × 10^9/L, respectively. Conversely, the percentage neutrophils (33.70 ± 15.52%) and absolute neutrophil count (2.11 ± 1.99 × 10^9/L) were significantly lower ($p < 0.05$) in the study subjects than in the controls, whose corresponding values were 61.07 ± 11.71% and 4.02 ± 3.15 × 10^9/L, respectively.

Table 4.4 presents the platelet-related parameters, CD4+ T-cell count, and methaemoglobin levels of the study subjects and controls. The platelet count of the study subjects (224.67 ± 60.18 × 10^9/L) was slightly higher than that of the controls (221.28 ± 59.18 × 10^9/L); however, the difference was not statistically significant ($p = 0.558$). Similarly, platelet distribution width (14.86 ± 5.32 fL) was slightly higher than that of the controls (14.10 ± 3.41

fL), but the difference was not statistically significant (p = 0.393). In contrast, mean platelet volume (13.13 ± 7.20 fL) was significantly higher (p < 0.05) in the study subjects than in the controls (10.38 ± 1.73 fL). Furthermore, platelet large cell ratio (P-LCR) (38.31 ± 14.02%) and methaemoglobin level (4.80 ± 5.80%) were significantly higher (p = 0.001) among the study subjects than among the controls, whose corresponding values were 29.27 ± 7.29% and 2.13 ± 1.38%, respectively.

Table 4.5 shows the result of blood film examination. Out of the 378 recruited subjects, normocytic normochromic red cells were observed in 52 (13.8%), mild microcytic hypochromic seen in 114 (30.1%) and markedly microcytic hypochromic red cells with acanthocytosis were observed in 212 (56.2%)

Table 4.6 shows the effect of duration of exposure to lead on red blood cells related parameters within the study subjects. The mean standard deviation of serum Pb (11.74±4.20), in group II (5-10 years exposure) and that of group III (Above 10years exposure) (11.73±4.25 µg/dl) are statistically significantly higher (p-value=0.047) compared with that in group I (less than 5 years exposure) (10.66±3.94 µg/dl). RBC was decreased in group I ($5.08\pm0.65\times10^{12}$/l)and group III, ($5.01\pm0.77\times10^{12}$/l) and increased in group II, ($5.11\pm0.74\times10^{12}$/l)but statistically not significant (p= 0.563). Haemoglobin concentrations in group I (14.54±1.94 g/dl), group II (13.73±2.53g/dl), group III (13.69±2.01, g/dl)and haematocrit in group I (41.61±6.38%), group II(39.06±8.42%), group III(39.029±6.35%) as well as mean cell volume of group I (80.71±8.65fl), group II (78.54±11.10fl) and group III (78.17±8.40fl)significantly decreased (p= ≤0.05). Mean cell haemoglobin in group I (28.96±5.09pg), group II (30.21±10.65pg), group III (29.18±7.33pg) and mean cells haemoglobin concentration in group I (34.90±2.94g/dl), group II (34.68±3.06g/dl), group III (34.76±3.24 g/dl) were not statistically significant (p=≥0.05).Red cell distribution width standard deviation in group I (42.12±9.56fl) group II(41.70±11.37fl), group III (42.85±9.36fl) and red cell distribution width coefficient variation in group I (13.83±1.15%), group II (13.74±1.08%) and group III(13.81±1.00%), across the three groups were not statistically significant (p=>0.05)

Table 4.7 shows the white blood cells related parameters among study subjects, % Lymphocyte increased in group II (49.53±8.80%), and group III (46.70±9.82%) when compared with group I (45.41±9.84%) with list exposure period;(p=0.014). %neutrophils significantly decreased statistically (p=0.002) in group II(36.70±7.67%) and group III, (38.13±8.56%) when compared with group I (39.53±9.22%). WBC counts increased in group II ($4.26\pm2.06\times10^{9}$/l) and group III ($4.07\pm1.92\times10^{9}$/l) when compared with group I, ($3.91\pm1.99\times10^{9}$/l) though statistically insignificant (p= 0.342).For %mix cells in group I (14.98±10.72%), group II (14.67±8.54%), group III(16.07±12.7%) and absolute lymphocyte in group I ($2.32\pm1.12\times10^{9}$/l), group II($2.30\pm1.11\times10^{9}$/l), group III($2.45\pm1.25\times10^{9}$/l); the effects of lead exposure were not statistically significant (p= >0.05).

For absolute mix cells in group I ($1.10\pm1.41\times10^{9}$/l), group II($0.82\pm0.50\times10^{9}$/l), group III ($0.88\pm0.94\times10^{9}$/l) as well as absolute neutrophils in group I (2.18±1.15), group II (2.03±0.86) and group III ($2.01\pm1.00\times10^{9}$/l) no significant difference was observed(p= >0.05).

Table 4.8 shows platelet related parameters, CD4+ count and methaemoglobin of study subjects. For platelet in group I ($228.47\pm66.182\times10^{9}$/l), group II ($223.36\pm62.38\times10^{9}$/l), group III($228.45\pm64.64\times10^{9}$/l) and platelet distribution width in group I (13.26±3.41fl), group II (13.14±3.07fl), group III (13.50±4.02fl); as well as mean platelet volume in group I (12.16±6.39fl), group II (12.81±6.57fl), group III(12.24±6.94fl); and platelet large cells ratio in group I (27.79±7.23%), group II (29.13±7.86%) and group III (28.06±7.91%) were statistically in significant (p=> 0.05). Cluster of differentiation four positive cells in group I (1023.65±414cells/µl),group II (1071.07±430.77cells/µl), group III(987.95±326.11cells/µl) and methaemoglobin in group I (5.67±5.63%), group II(4.16±2.28%), group III (4.33±2.86%) across the groups were statistically in significant (p= > 0.05).

Figure 1 shows a positive and significant correlation between serum lead level and some haematological parameters including % lymphocyte (rho= 0.174), absolute lymphocyte(r=0.181), % mix cells (r=0.162), absolute mix cells (0.152) red cell distribution width standard deviation(r= 0.367) and red blood cell distribution width coefficient variation (r= 0.067) showed positive statistically significant correlations (p=≤0.05) while platelet (r= 0.253), platelet distribution width(rho= 0.08), white blood cells (r= 0.042), red blood cells (r= 0.090), mean cell volume (r= 0.066) cluster of differentiation four (CD4+) positive cells (r= 0.087) showed positive correlations but statistically not significant (p=> 0.05).

Figure 2 shows a negative correlation between serum lead and haematological parameters. Absolute neutrophils (r= -0.018), % neutrophils (r= -0.017), haemoglobin(r= - 0.070), haematocrit(r= -0.034), mean cell haemoglobin(r=-0.030), mean cell haemoglobin concentration(r=-0.002), mean platelet volume(r=-0.084), platelet large cell ratio(r= -0.049) and methaemoglobin(r=-0.056), showed negative but statistically insignificant correlations (p= > 0.05).

Table 4.1: Serum Lead Level of Fueling Station Attendants and Controls

Parameters	Test subjects (n=378)	Controls (n=150)	t-value	p-value
Lead (µg/dl)	11.334±3.357	7.838±3.984	70.894	0.001

This table shows the serum lead level of study subjects and controls. Values are presented as mean standard deviation.

Table 4.2: Red Cells Indices of Study Subjects and Controls

Parameters	Subjects	Controls	t-value	P-value
RBC ($\times 10^{12}$/l)	4.25±1.08	5.20±1.36	9.46	0.001
HGB (g/dl)	13.19±8.59	16.01±1.01	6.74	0.001
HCT (%)	43.28±12.45	45.21±3.27	2.41	0.016
MCV (fl)	79.01±9.14	88.64±11.48	168.81	0.001
MCH (pg)	26.49±10.09	32.17±1.45	7.86	0.001
MCHC (g/dl)	34.54±3.42	35.21±2.67	191.40	0.001
RDW-SD (fl)	46.99±3.80	40.93±11.80	5.45	0.001
RDW-CV (%)	14.72±1.23	14.05±1.11	3.27	0.002

This table shows the values for red blood cells parameters of study subjects and controls as mean standard deviation as well as the p-values of the different variables. Key - HGB = haemoglobin, HCT

= haematocrit, MCV = mean cells volume, MCH = mean cells haemoglobin, MCHC = mean cells haemoglobin concentration, RBC = red blood cells, RDW-SD = red cells distribution width standard deviation, RDW-CV = red cells distribution width coefficient variation, fl = femtolitre, % = percent,g/dl = gram/deciliter,pg = picogram,

4.3: Total and Differential White Blood Cells Count of Study Subjects and Controls.

Parameters	subjects	Controls	t-value	p-value
WBC ($\times 10^9$/l)	5.24±2.15	4.33±2.11	67.27	0.001
LYM (%)	42.36±14.39	25.85±10.02	4.22	0.001
MXD (%)	18.69±14.32	14.38±5.73	3.40	0.003
NEUT (%)	33.70±15.52	61.07±11.71	21.10	0.001
LYM ($\times 10^9$/l)	4.10±2.23	2.34±1.26	12.67	0.001
MXD ($\times 10^9$/l)	4.92±2.6	3.71±8.83	1.28	0.004
NEUT ($\times 10^9$/l)	2.11±1.99	4.02±3.15	11.41	0.001

This table shows the values for White cells parameters of study subjects and controls as mean standard deviation as well as the p-values of the different variables. WBC= white blood cells, LYM = lymphocyte, MXD = mix cells, NEUT = neutrophil, l% = percent, x = multiply.

4.4 Platelet Parameters, CD4+ Count, Hi of Study Subjects and Controls.

Parameters	Subjects	Controls	t-value	p-value
PLT ($\times 10^9$/l)	224.67±60.18	221.28±59.18	1.87	0.558
PDW (fl)	14.86±5.32	14.10±3.41	1.55	0.393
MPV (fl)	13.13±7.20	10.38±1.73	5.65	0.001
PL-CR (%)	38.31±14.02	29.27±7.29	3.39	0.001
CD4+ (cells/µl)	1133.95±583.35	1558.20±622.57	6.03	0.001
Hi (%)	4.80±5.80	2.13±1.38	6.55	0.001

This table shows the values for platelet parameters, CD4+, Hi, of study subjects and controls as mean standard deviation as well as the p-values of the different variables. PLT= platelet, PDW = platelets distribution width, MPV = mean platelets volume, PL-CR= platelet large cell ratio, CD4+ = cluster of differentiation four positive cells, Hi= methemoglobin, fl = femtolitre, % = percent

4.5 Shows Morphological Appearance of the Subjects' Red Blood Cells

Appearance of red cells	Number of subjects	Percentage (%)
Normocytic normochromic	52	13.8
Mild microcytic hypochromic	114	30.1
Markedly microcytic hypochromic/acanthocytosis	212	56.1
Total	378	100

4.10: Effect of Duration of Pb Exposure on Red blood cells indices in Fuel Station Attendants.

Parameters	Less than 5 years	5-10 years	Above 10 years	p-value
Lead (µg/dl)	10.66±3.94	11.74±4.20	11.73±4.25	0.047
RBC ($\times 10^{12}$)	5.08±0.65	5.11±0.74	5.01±0.77	0.563
HGB (g/dl)	14.54±1.94	13.73±2.53	13.69±2.01	0.002
HCT (%)	41.61±6.38	39.06±8.42	39.029±6.35	0.002
MCV(fl)	80.71±8.65	78.54±11.10	78.17±8.40	0.016
MCH(pg)	28.96±5.09	30.21±10.65	29.18±7.33	0.393
MCHC(g/dl)	34.90±2.94	34.68±3.06	34.76±3.24	0.836
RDW-SD(fl)	42.12±9.56	41.70±11.37	42.85±9.36	0.693
RDW-CV (%)	13.83±1.15	13.74±1.08	13.81±1.00	0.76

This table shows the effect of duration of lead exposure on the values for red blood cells parameters of study subjects and controls. Key = HGB = haemoglobin, HCT = haematocrit, MCV = mean cells volume, MCH = mean cells haemoglobin, MCHC = mean cells haemoglobin concentration, RBC = red blood cells, RDW-SD = red cells distribution width standard deviation, RDW-CV = red cells distribution width coefficient variation, fl = femtolitre, % = percent, g/dl = gram/deciliter, pg = pictogram

Post Hoc Test (Tukey)

Group(n=3)	HB		Hct		MCV	
	Mean difference	p-value	Mean difference	p-value	Mean difference	P-value
Less than 5yrs vs. 5-10yrs	0.812	0.007	2.651	0.003	3.882	0.001
Less than 5yrs vs. above 10yrs	0.381	0.371	1.244	0.326	0.791	0.778
5-10yrs vs. less than 5yrs	0.812	0.007	2.656	0.003	3.882	0.001
5-10yrs vs. above 10yrs	0.431	0.472	1.411	0.294	3.091	0.039
Above 10yrs vs. less than 1 yrs.	0.381	0.371	1.244	0.326	0.791	0.775
Above 10yrs vs. 5-10yrs	0.431	0.342	1.411	0.294	3.091	0.039

The mean difference is significant at the 0.05 level.*

4.11: Effect of Pb exposure on white blood cells in fuel station attendants

Parameters	Less than 5 years	5-10 years	Above 10 years	p-value
Lead (µg/dl)	10.66±3.94	11.74±4.20	11.73±4.25	0.047
WBC ($\times 10^9$/l)	3.91±1.99	4.26±2.06	4.07±1.92	0.342
LYM (%)	45.41±9.84	49.53±8.80	46.70±9.82	0.014
MXD (%)	14.98±10.72	14.67±8.54	16.07±12.7	0.596
NEUT (%)	39.53±9.22	36.70±7.67	38.13±8.56	0.002
LYM ($\times 10^9$/l)	2.32±1.12	2.30±1.11	2.45±1.25	0.55
MXD ($\times 10^9$/l)	1.10±1.41	0.82±0.50	0.88±0.94	0.070
NEUT ($\times 10^9$/l)	2.18±1.15	2.03±0.86	2.01±1.00	0.466

This table shows the effect of duration of lead exposure on values for White cells parameters of study subjects. Keys = WBC= white blood cells, LYM = lymphocyte, MXD = mix cells, NEUT = neutrophil, l% = percent, x = multiply.

Post Hoc Test (Tukey)

Group(n=3)	Pb		% LYMP		%NEUT	
	Mean difference	p-value	Mean difference	p-value	Mean difference	P-value
Less than 5yrs vs. 5-10yrs	1.343*	0.021	0.0809	0.997	0.427	0.911
Less than 5yrs vs. above 10yrs	1.600*	0.009	4.061	0.003	2.125	0.136
5-10yrs vs. less than 5yrs	1.343*	0.021	0.0809	0.997	0.427	0.911
5-10yrs vs. above 10yrs	0.257	0.900	3.979	0.008	1.698	0.337
Above 10yrs vs. less than 1 yrs.	1.600*	0.009	4.061	0.003	2.125	0.136
Above 10yrs vs. 5-10yrs	0.257	0.900	3.971	0.911	1.698	0.337

The mean difference is significant at the 0.05 level.

4.12: Effect of the Duration of Pb Exposure on Platelet, CD4+ count and Hi in Fuel Station Attendants.

parameters	Less than 5 years	5-10 years	Above 10 years	p-value
Lead (µg/dl)	10.66±3.94	11.74±4.20	11.73±4.25	0.047
PLT ($\times 10^9$/l)	228.47±66.18	223.36±62.38	228.45±64.64	0.766
PDW (fl)	13.26±3.41	13.14±3.07	13.50±4.02	0.732
MPV (fl)	12.16±6.39	12.81±6.57	12.24±6.94	0.691
PL-CR (%)	27.79±7.23	29.13±7.86	28.06±7.91	0.318
CD4+ (cells/µl)	1023.65±414.83	1071.07±430.77	987.95±326.11	0.861
Hi (%)	5.67±5.63	4.16±2.28	4.33±2.86	0.060

This table shows the effect of duration of lead exposure on the platelet parameters, CD4+, Hi, of study subjects Keys = PLT= platelet, PDW = platelets distribution width, MPV = mean platelets volume, p-LCR= platelet large cell ratio, CD4+ = cluster of differentiation four positive cells, Hi= methaemoglobin, fl = femtolitre, % = percent,

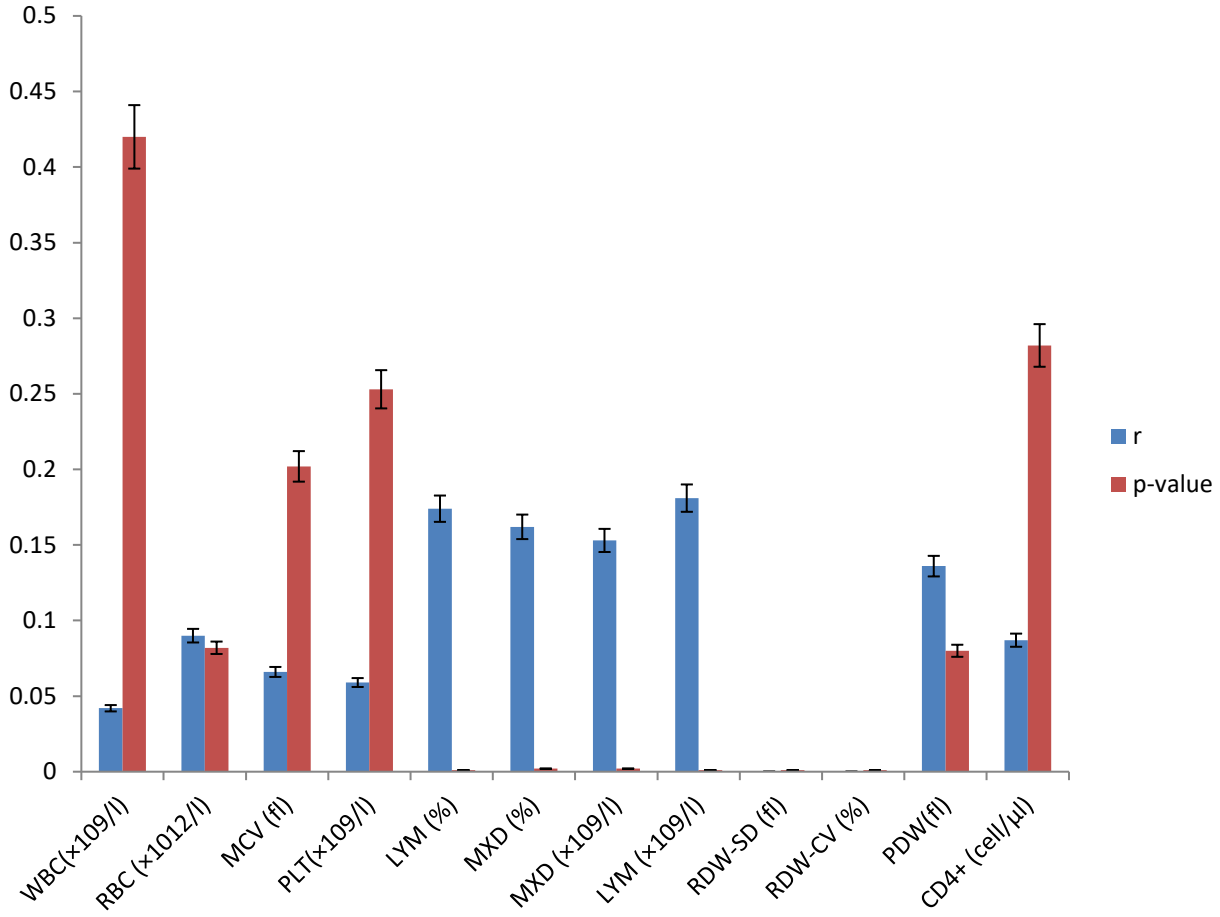

Figure 1: Shows Correlation between Serum Lead Level and Some Haematological Parameters among Fuel Station Attendants.

This Figure shows positive correlation between serum lead level and haematological parameters of study subjects, reported as Spears son correlation coefficient = r with the p-values. Keys = MCV = mean cells volume, RBC = red blood cells, WBC= white blood cells, RDW-SD = red cells distribution width standard deviation, PLT= platelet, LYM = lymphocyte, MXD = mix cells, PDW = platelets distribution width, CD4+ = cluster of differentiation four positive cells, fl = femtolitre, l = litre, % = percent, x = multiply.

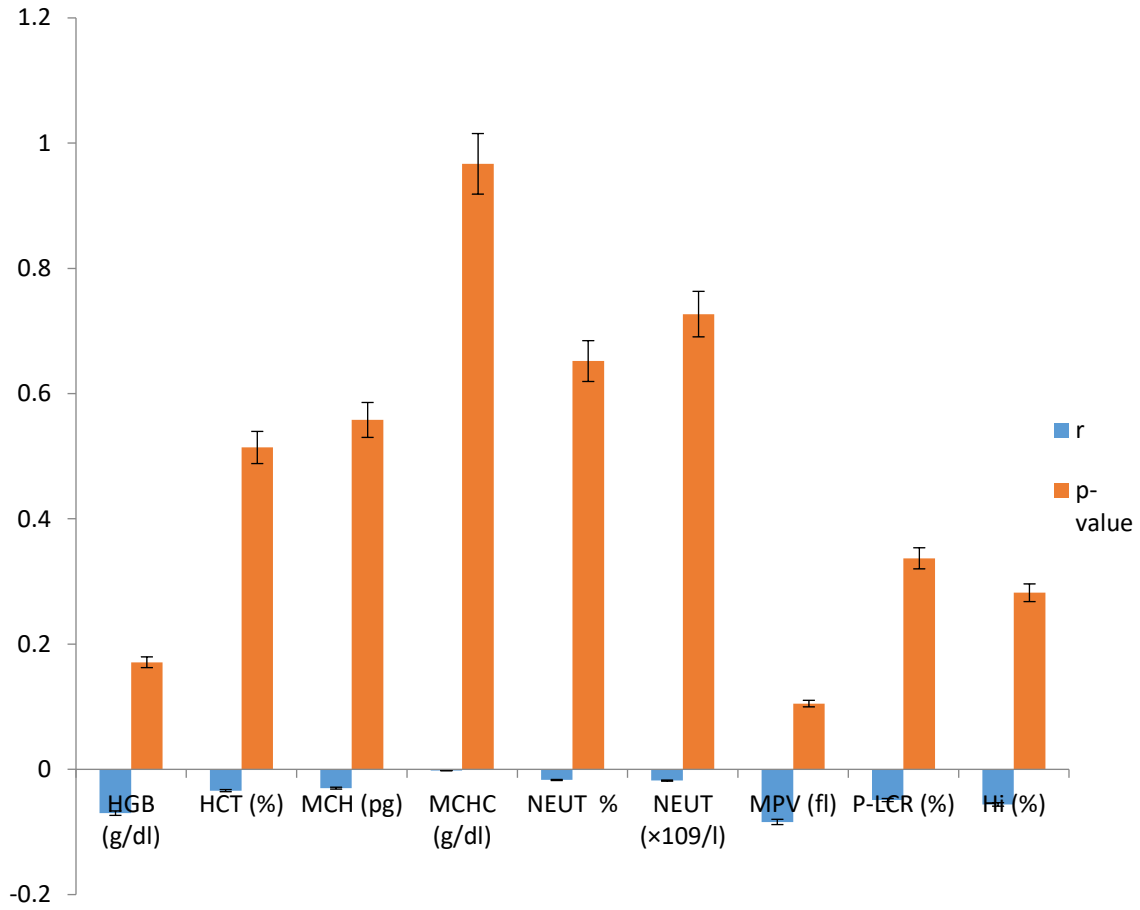

Figure 2 Shows Some Haematological Parameters That Correlate Negatively with Serum Lead

This Figure shows negative correlation between serum lead and some haematological parameters of study subjects reported as r= Spears son correlation and the p-value. Keys = HGB = haemoglobin, HCT = haematocrit, MCH = mean cells haemoglobin, MCHC = mean cells haemoglobin concentration, NEUT = neutrophil, MPV = mean platelets volume, p-LCR= platelet large cell ratio, Hi= methaemoglobin, g/dl = gram/deciliter, fl = femtolitre, pg = picogram, l = litre, % = percent, x = multiply. µg = microgram

DISSCUSSION

In this study, the serum lead level was significantly higher (p=0.001) in occupationally-exposed fuel station attendants when compared to that of controls. This apparently is an indication of Pb accumulation in the body of the subjects as a result of inhalation of petroleum fume. Our finding agrees with previous reports by Mandakini *et al.* (2015), Dongre *et al.* (2011) and Alasia *et al*. (2010), which indicated a significant elevation of blood lead level among workers occupationally exposed to lead. Significant increase in blood lead level was reported among policemen who were highly exposed to lead as recorded by Arshad and Khan (1997) in relation to controls. Also, Ojo *et al*., (2016) and Babalola and Babajide (2009) observed significant increase in blood lead level of the bricklayers who practiced their profession without protective coverings compared to the controls. However, the findings of this study disagree with that reported by Christian *et al.* (2016), Ahed *et al.*, (2013) and Al-Malki (2009)

who reported statistically insignificant difference in blood lead level among occupationally exposed prisoners, Saudi Arabian fire fighters and fuel station attendants compared to controls respectively.

The significant increase in serum lead level of fuel station attendants obtained may not just be due to intermittent exposure but a continuous inhalation of fumes from lead laden gasoline in the station. High levels of lead in the blood affect the activity of many enzymes needed for the proper functioning of red blood cells and can lead to anaemia (ATSDR, 2007).

Statistically significant increase in serum lead level($p= 0.047$) based on duration of exposure was also observed in this research. This finding agrees with the work of Cocco *et al*. (1997) who reported that lead toxicity was observed to increase proportionally to duration of exposure. This indicates non-complaint to the use of PPE among the study subjects.

The present study, observed statistically significant decreases in the RBC, HCT and Hb of the subjects ($p= 0.001$). This may be due to the effects of lead on erythropoiesis. This finding agrees with that of Uko *et al*. (2015) who reported significantly low haemoglobin, packed cell volume and red blood cell count among petrol station attendants occupationally exposed to premium motor spirit. However, Mandakini *et al.*, (2015) reported a decrease only in HGB and HCT in Pb handlers. Decreased haemoglobin concentration, packed cell volume was also observed by Dede and Kagbo (2002). Moreover, a significant decrease in Hb, PCV was reported by Ukaejiofo *et al*. (2009). Our findings on Hct disagree with work of Shah (2006) who reported that moderate lead exposure resulted in a significant increase in Hct. In this research, no statistically significant correlation was observed between Hb, Hct and serum lead, which disagreed with work of Amal *et al.* (2010) who reported statistically insignificant negative correlation between Hb, Hct and serum lead in children with anaemia. Also, a significant negative correlation between haemoglobin and serum lead level was also reported by Drita *et al.* (2014) in children exposed to lead. Haemoglobin and haematocrit study subjects were significantly decreased ($p= \leq 0.05$) in accordance with duration of Pb exposure. Our finding concord with work of Ukaejiofo *al.* (2009) who reported a decrease in Hb and PCV in accordance with duration of exposure to lead, ranging from 6 month to 5years, 6 to 10years and above 10 years respectively, in lead exposed worker.

Decreased MCV, MCH, MCHC were also observed in this study. Our finding may be due to the effect of lead on red cells membrane and enzymes. This agrees with a previous report by Mandakini *et al.* (2015) which indicated a decrease in the mean cell volume (MCV) and mean cell haemoglobin (MCH) as well as in mean cell haemoglobin concentration (MCHC) in Battery Manufacturing Workers of Western Maharashtra. Uko *et al*. (2015) reported a significantly lower mean cell haemoglobin concentration (MCHC) among gasoline station attendants in a duration dependent manner compared to controls. Our findings disagree with work of Sina *et al*. (2013) who reported normal haematological indices and Shah, (2006) who reported that severe and extreme lead exposure caused a significant increase in MCV and MCH, and significant decrease in MCHC. The decrease may be due to inhibition of heme-synthesizing enzyme, δ-aminolevulinic acid dehydratase (ALAD) resulting in reduced haemoglobin synthesizing power by the red and it may be due to the ability of lead to compete with iron on DMT-1. MCH and MCHC were negatively correlated with serum lead in this research which agrees with the work of Ibeh *et al.,* (2016) among petrol station attendants and automobile mechanics in Nnewi, South-East Nigeria. Sina *et al*. (2013) worked on lead exposed workers of a Car Battery Industry and made similar finding, while MCV correlated positively with serum lead, though statistically not significant ($p= > 0.05$). This insignificant correlation may be due to the deleterious effect of Pb on haem synthesis. Mean cells volume of study subjects were also significantly decreased ($p<0.05$) in accordance with duration Pb exposure. Our finding agrees with that of Ukaejiofo *et al.,* (2009), in lead exposed worker with respect to duration Pb exposure.

Increased of RDW-SD and RDW-CV was observed in this research. This may be due to the effect of lead

on transferrin and ceruplasmin. Our finding correlated with that of Abozer *et al.* (2015) and Gurer *et al.* (1998) who reported variation in the size of red blood cells in individuals exposed to lead but disagree with finding of Ukaejiofo and Thomas (2009) as well as Rashid *et al*. (1995), who reported normocytic normochromic appearance of blood cells in lead exposed subjects. However, significant positive correlation was observed between blood lead level, RDW-SD and RDW- CV ($p < 0.05$) in this study. This finding is in concord with work of Amal *et al.* (2010) who reported positive correlation ($r = 0.458$) between the RDW and serum lead level (p-value = 0.001) in children exposed to lead. The finding of this research indicates that as serum lead level increases variation in size of red blood cells also increase, probably due to the effect of Pb on iron absorption and transportation. No statistically significant difference in the level of RDW-SD and RDW- CV was observed in this research based on the duration of Pb exposure.

In this study, there was a significant increase ($p = 0.001$) in the total WBC counts ($5.24 \pm 2.15 \times 10^9$) when compared with the controls ($4.33 \pm 2.11 \times 10^9$). The increase in the number of TLC in fuel station attendants in comparison with controls in the present study indicates a positive immune response to petroleum fume toxicity. This finding agrees with those of Hatemabdel and Shaaban (2015) and Ismail *et al.* (2006), who reported a significant increase in white blood cells count in the entire subjects exposed to lead. Mandakini *et al.* (2015) and Patil *et al.* (2006) reported a statistically significant elevation of total WBC count ($p=0.001$) in exposed battery manufacturing workers as compared with controls. Elevated WBC in traffic policemen was also reported by Shafaat *et al.* (2016). Ahed *et al.* (2013) observed increased in WBC in lead exposed prisoners ($p=0.000$). Moreover, increase in total leucocytes count (TLC) was detected in Nigerian industry workers involved in lead handling (Ukaejiofo *et al*., 2009). The findings of this study disagree with that of Shaik and Jamil, (2008) on Pb handlers and Uko *et al*. (2015) among fuel station attendants in Sokoto State, Nigeria. However, Dede and Kagbo (2002) observed a decrease in WBC of rat exposed to gasoline. However, WBC count correlated positively with serum lead level but statistically not significant ($p= 0.420$). This suggests that serum lead level has no significant effect on WBC count based on duration of exposure ($p= 0.342$).

A statistically significant increase ($p=0.001$) in the number of lymphocytes was observed in this study compared to the controls. This means that petroleum workers had significantly higher number of lymphocytes than the controls. This finding suggests a possible involvement of cellular immune response against this particulate pollutant in highly exposed persons that may have resulted in the increased lymphocyte count.

This finding agrees with the work of Obidike *et al*. (2010) who reported significant increase in the number of lymphocytes in albino rats exposed to graded levels of lead acetate. However, our finding disagrees with work of Ukaejiofo *et al.* (2006) who reported decreased in lymphocytes number among workers exposed to some petroleum products in Enugu State, Nigeria.

Significantly positive correlation between both % lymphocytes and absolute lymphocytes count ($r= 0.174$, $r= 0.181$), and serum lead level were observed in this research ($p= 0.001$). Our result disagrees with that reported by Sina *et al*. (2013), who observed a negative correlation ($r= -0.087$) between Pb level and lymphocyte count ($p= 0.437$). However, his findings were not statistically significant. A significant increase in the number of lymphocytes based on duration of exposure was also observed in this study ($p= 0.014$). This agrees with the findings of Ekanem *et al*. (2015) in experimental animals after exposure to lead acetate at different concentration and duration of exposure, but disagrees with the finding of Ukaejiofo *et al.* (2009), who reported decreased of lymphocyte in accordance with duration of exposure.

Decrease in both absolute and percentage neutrophil was observed in this study compared to the controls. Our findings are in concord with the results reported by Ukaejiofo *et al.* (2006) among workers exposed to some petroleum products. McMurry *et al.* (1995) reported non-significant increase in neutrophils in cotton rats subjected to sub chronic lead poisoning. However, our finding is at variance with the work of

DiLorenzo *et al*. (2006), who reported a significant elevation of absolute neutrophils count in lead-exposed workers. The finding of reduced number of neutrophils in this present study may be due to cell-mediated immune response induced by the lead exposure. The neutrophils being the phagocytic cells act as first-line of defense in the immune response against the invading organism and many may have been lost in the process. This decrease in their number may also be due to an increase of phagocytosis without effective production and replacement of the white cells from the bone marrow. Decrease neutrophils production may as well be due to accumulation of Pb in bone marrow matrix or the magnitude of the neutrophil's reduction may not be sufficient enough to trigger a satisfactory response and production from bone marrow.

Negative correlation between serum lead level and % neutrophils as well as absolute neutrophil count (r= -0.071, r= -0.018) noticed in this research disagreed with finding of Sina *et al*. (2013) in Lead Exposed Workers, though his findings were not statistically significant. Statistically significant decreased of % neutrophils were also observed based on duration of Pb exposure (p= 0.002), Our result agrees with that reported by Ekanem *et al.* (2015) in experimental animals after exposure to lead acetate at different concentration and duration but disagrees with finding of Ukaejiofo *et al.* 2009, that reported normal neutrophil count in lead exposed worker with respect to duration of exposure. This may indicate that duration of exposure may play a vital role in lead toxicity.

Profound statistically significant increase of MXD cells was observed in this study. MXD cells consist of other white blood cells outside neutrophils and lymphocytes, including eosinophils, basophils and monocytes. Arshad *et al.* (2000) and McMurry *et al*. (1995) reported a significant increase in the number of eosinophils in cotton rats and in individuals having great chance of being exposed to lead. Therefore, a significant increase in MXD observed in this study may be due to increase in eosinophils as observed by the previous workers. However, the result from present study disagrees with that of Kuo *et al.* (2001), who reported significant decreased in monocytes and eosinophils in long-term lead exposure among Taiwanese workers. Similar result was observed by Ukaejiofo *et al*. (2009) among lead handlers in Enugu Urban, Enugu State, Nigeria. McMurry *et al.* (1995) found no significant effect on monocytes in cotton rats. Arshad *et al*.(2000) reported a significant increase in the number of eosinophils in persons with high chances of being exposed to lead, while the monocytes was observed as being less significant in number (Shafaat *et al*., 2016). In another study, monocytes count of highly exposed persons was noticed to be significantly lower than the controls (Ahed *et al*., 2013). Significantly positive correlation (r= 0.162) between the mix cells and serum lead level were observed in this research (p= 0.001). This finding may be due to an increase in the number of eosinophils as it was previously reported to be increased in lead exposed individuals. No statistically significant difference was observed on % and absolute mix cells count based on duration of exposure (p= 0.596, p= 0.070).

There was no statistically significant difference in platelets count of the study subjects (p= 0.609) compared with controls. This finding agrees with work of Christian *et al.* (2016) who reported normal platelets count in fueling station attendants compared with control subjects. It however, disagrees with the work of Ahed *et al*. (2013), which reported statistically significant decreased of platelet count in prisoners with higher blood lead levels. Ibeh *et al.* (2016) reported a decreased platelet count among petrol station attendants and automobile mechanics in Nnewi, South-East of Nigeria as well as Ukaejiofo *et al.* (2006) among workers exposed to some petroleum products in Enugu State, Nigeria. Nevertheless, Hala Samir *et al.* (2015) reported significant increase of platelets count among fueling station attendants, compared with the control group. The findings of the present research indicated that Pb toxicity has mild effect on platelet count, though as reported to be increased by other researchers, it can potentially cause thrombosis.

Statistically significant increase ($p = \leq 0.05$) of mean platelet volume (MPV) and platelet large cells ratio (PL-CR) were observed in this research. Our finding correlates with that of Ahed *et al*. (2013) who noticed

statistically significant (p= 0.001) increased in the number of platelets in prisoners exposed to lead. The findings of this research may indicate activation of platelet by Pb despite the fact that, their count is within the normal range. No significant correlation was observed between platelet count and serum lead level (p= 0.253). Our finding therefore, is in accordance with that of Sina *et al*. (2013). Moreover, duration of Pb exposure has no statistically significant effect on platelet and platelet related parameters (p= > 0.05).

The results of this study shows that the blood film of 30.1% of the subjects showed mild microcytic hypochromic red cells appearance while 56% showed markedly microcytic hypochromic appearance with acanthocytosis. This finding agrees with the work of Abozer *et al.* (2015) and Gurer *et al.* (1998) as well as Staudinger and Roth (1998) who reported microcytic hypochromic appearance of red blood cells but disagrees with previous observations by Ukaejiofo and Thomas (2009) and Rashid *et al*. (1995), who reported normocytic normochromic appearance of the red blood cell. This may be due to the effect of Pb on iron absorption and haemoglobin production.

Statistically significant decrease in the number of CD4+ cells count was noticed in this current study (p=0.001). This finding agrees with previous reports (Li *et al*., 2005; Basaran and Undeger, 2000; Undeger and Colleagues, 1996; Fischbein *et al*., 1993) that noted a reduction in the absolute number of CD4+ cells among lead exposed workers. Our finding however disagrees with work of Sata *et al*., (1997) who reported no difference in the absolute number of CD4+ cells between lead exposed subjects and controls. Reduction in the number CD4+ cells may be due to the effects of lead on the expression of the CD4+ surface antigen. The number of CD4+ cells correlate positively with serum lead level but was statistically not significant (p= 0.282). Moreover, duration of Pb exposure was observe to have no statistically significant effect on CD4+ cells count (p= 0.861). our finding on CD4+ cells may be associated with effect of lead on CD4+ surface protein.

Increase in the level of methaemoglobin was observed among the research subjects as compared with controls. This result agrees with that reported by Cristine *et al.* (1997), who noticed a significant increase in the level of methaemoglobin (p= 0.01) in lead exposed workers. The finding of this work may be due to increase in oxygen free radical's generation induced by Pb as it was reported that it can change the anti-oxidative activities of some enzymes by suppressing the sulfhydryl group. Active sulfhydryl groups are needed to prevent the oxidation of haemoglobin to methaemoglobin (Nehru and Kanwar, 2007; El-Sokkary *et al.,* 2005). Similar observation was reported in individuals with nitrite poisoning (Ohashi *et al.,* 1998). The finding of this research may be due to ability of lead to facilitate or hasten the conversion of Hb into met-Hb during Hb oxidation in the presence of lead. It may also probably be due to its ability to interfere with activity of NADH-dependent cytochrome b5-methaemoglobin reductase, the enzyme that naturally converts methaemoglobin to haemoglobin. Hi concentration correlated negatively with serum lead level (r= -0.056) but was statistically not significant (p= 0.282). It was also noted that the duration of Pb exposure had no statistically significant (p= 0.060) effect on methaemoglobin level.

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