Effect of Lead Exposure on Some Biochemical Parameter of Battery Factory and Benzene Fuel Stations Workers


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Abstract:
Lead pollution is an important environmental problem, despite consciousness of its harmful health effects in the world. This study was an attempt to find the effects on some possible biochemical parameters. The study has been conducted in Baghdad City from March 2017 to April 2018 divided in to two groups exposure 75 workers and non-exposure 10 control. Biochemical tests of liver and kidney Functions showed clear lead effect on them. The average [ALK, GPT, and GOT] in battery factories workers were 75.52 ± 5.47, IU/L 33.54 ± 4.58, 42.64 ± 6.59 IU/L and fuel station workers 62.41 ± 3.70, 27.15 ± 2.16, 29.42 ± 1.48 IU/L compared with the control group respectively 45.10 ± 6.39, 17.95 ± 2.11 and 28.71 ± 2.65 IU/L the study showed significant increase in the mean liver enzyme of exposed workers, compare with control. The mean of serum urea and creatine level in battery factory workers, fuel station and control. Were 41.16 ± 2.21 mg/dl, 38.04 ± 1, 69 mg/dl and 20.40 ± 0.54 mg/dl and 0.986 ± 0.06 mg/d, 0.941 ± 0.05 mg/dl, and 0.710 ± 0.06 mg/dl respectively there was significant increase in serum urea and creatinin of the workers compared with control.

Key words: lead, urea, creatine, alkaline phosphates [ALK], Glutamic pyruvic Transaminase [GPT], and Glutamic Oxaloacetic Transaminase [GOT]

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Tأثير التعرض للرصاص على بعض المعايير البيوكيميائية من العاملين في مصنع البطاريات ومحطات الوقود البينزين

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الخلاصة:

The absorbed Pb is associated with liver, it has been considered the largest main store Pb repository and the target organ for its poisoning effects [6-7]. Experimental studies have shown that chronic Pb exposed can cause an arising in (AST), (GPT), and (ALK) [8]. In addition, Pb stimulates intercellular signaling between hepatocytes and Kupffer cells, which contributes to Pb-induced hepatotoxicity [9].

Liver Enzymes:
Enzymes were measured by using kits provided by Biomerieux - France for the colorimetric determination of Glutamic
Pyruvic Transaminase (GPT) and Glutamic Oxalat Transaminase (GOT) in serum according to the following reaction: NaOH its concentration 0.4 N. For GOT determination, reagents 3 and 4and sodium hydroxide were used, while reagent 2 was replaced with reagent 1 which consists of phosphate buffer pH 7.5, aspartate and α-ketoglutarate. Samples absorbance (A) was determined by using spectrophotometer on 505 nm. The number of GPT and GOT were measured as units/ml which calculated from applying the absorbance (A) of samples on the standard curves [11].

Enzymes were measured by using kits provided by Jourilabs -Switzerland for the colorimetric determination of alkaline phosphates readable at a 510 nm absorbance in the spectrophotometer.

Result and Discussion:

Table1. Shows the effect of lead on urea and creatinine parameters, regarding urea, high significant difference **(P<0.01) was found between fuel stations workers 38.04 ± 1.69 mg/dl, batteries factory workers 41.16 ± 2.21 mg/dl compare to control 20.40 ± 0.54 mg/dl, concerning Creatinine, significant difference *(P<0.01) was found between Fuel stations workers 0.941 ± 0.05 mg/dl, batteries factory workers 0.986 ± 0.06 mg/dl compare control 710 ± 0.06 mg/dl. Normal value of urea 18-45mg/dl, normal value of creatinine 0.5-1.1mg/dl. Blood urea and serum creatinine are parameters that can be used to detect the renal effects caused by occupational exposure to Pb. But, when these tests are found abnormal, the nephropathy has already reached the irreversible phase that may lead to renal insufficiency [12]. The results showed high significant increase in serum urea in workers compare to the control (p> 0.01) Through these results the probability of injury of workers to renal failure at the long term is greater than the control group, exposure to high level of lead >60 µg/dL may cause renal dysfunction, even a low level of lead ~10 µg/dL may also provide the same problem [13]. The pathological influence of the exposure of lead on the renal systems of workers seems to result to the renal toxicity development under the influence of the oxidative stress that it causes. That such effect only damages kidney in chronic exposure that becomes clinically significant, and that kidney damage does not usually occur in asymptomatic acute cases[14]. While in chronic accumulation of lead in the body eventually leads to impairment in renal function, urea and creatinine are a waste product of amino acid metabolism they removed by kidney, oxidative stress appears to be involved in the development of renal toxicity induced by the environmental lead exposure that causes significant pathological lesions on the renal systems of men and animals[15]. People with occupational lead exposure are at risk of developing hyperuricemia and renal impairment [16].
Table (1) Show the effect of lead on Urea and Creatinine test in Batteries factory workers Fuel stations workers and control.

<table>
<thead>
<tr>
<th>Groups</th>
<th>No.</th>
<th>Mean ± SE</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Urea (mg/dl)</td>
<td>Creatinine (mg/dl)</td>
</tr>
<tr>
<td>Fuel stations</td>
<td>25</td>
<td>38.04 ± 1.69 a</td>
<td>0.941 ± 0.05 a</td>
</tr>
<tr>
<td>Battery Factories.</td>
<td>50</td>
<td>41.16 ± 2.21 a</td>
<td>0.986 ± 0.06 a</td>
</tr>
<tr>
<td>Control</td>
<td>10</td>
<td>20.40 ± 0.54 b</td>
<td>0.710 ± 0.06 b</td>
</tr>
<tr>
<td>LSD value</td>
<td>---</td>
<td>7.401 **</td>
<td>0.220 *</td>
</tr>
</tbody>
</table>

* (P<0.05), ** (P<0.01).

Means having with the different letters in same column differed significantly.

As shown in the table 2, ALK-P, GOT and GPT, were highly significant increase fuel station and battery factory in workers compare with control p <0.01 was found between fuel stations workers regarding ALK phosphate (62.41 ± 3.70 U/L) , battery factories workers (75.52 ± 5.47 U/L) and control 45.10 ± 6.39 U/L , high significant difference P<0.01 was found between battery factories regarding GOT( 42.44 ± 6.59 U/L) and control (28.71 ± 2.65 U/L) and regarding GPT high significant difference **(P<0.01) was found between battery factories workers (33.54 ± 4.58 U/L) and control (17.95 ± 2.11 U/L). Lead can stimulate intercellular signaling between Kupffer cells and hepatocytes, which are enhanced synergistically in the presence of low lipopolysaccharide levels [17]. Higher concentration of lead cause severe periportal inflammation in chicken liver, therefore, it may be assumed that long term lead exposure cause liver damage in human, but low concentration of lead disturbs the normal biochemical process in the hepatobiliary system [18].

Table (2) Show the effect of lead on Liver functions (ALK, GOT, GPT) in Batteries factory workers, Fuel stations workers and control.

<table>
<thead>
<tr>
<th>The Groups</th>
<th>No.</th>
<th>Mean ± SE</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>[ALK [U/L]</td>
<td>[GOT [U/L]</td>
</tr>
<tr>
<td>Fuel stations</td>
<td>25</td>
<td>62.41 ± 3.70 a</td>
<td>29.42 ± 1.48 b</td>
</tr>
<tr>
<td>Battery Factory.</td>
<td>50</td>
<td>75.52 ± 5.47 a</td>
<td>42.44 ± 6.59 a</td>
</tr>
<tr>
<td>Control</td>
<td>10</td>
<td>45.10 ± 6.39 b</td>
<td>28.71 ± 2.65 b</td>
</tr>
<tr>
<td>LSD value</td>
<td>---</td>
<td>18.569 **</td>
<td>10.187 **</td>
</tr>
</tbody>
</table>

** (P<0.01).
References:


