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Research Article

Evaluation of Blood Pressure, Liver Function, and Hemoglobin Concentration Alterations in Cigarette Smokers on the West Coast of Libya

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Abstract: Background: Cigarette smoking is one of the 10 greatest contributors to global death and disease. Cigarette smoking is the most common type of tobacco use. In average, to date 47.5% of men and 10.3% of women are current smokers. Tobacco continues to be the second major cause of death in the world. It consists of many chemicals, including cytotoxic, carcinogenic and free radicals, therefore it affects many organs if not all. **Objective:** The aim of this study is to assess the extent of adverse effect of cigarette smoking on blood pressure, liver function, and hemoglobin concentration in male population on the West Coast of Libya. **Methods:** This study was conducted on (200) healthy male subjects, their ages ranged from 15 to 85 years. They were divided into four groups; 50 non-smokers as control group (age between 15-35 years), 50 smokers (age between 15-35 years), 50 non-smokers as control group (age between 36-84 years) and 50 smokers (age between 36-84 years). The blood was collected in EDTA tubes to test the Complete blood count (CBC) and in plain tubes for biochemical assay (activities of liver enzymes). The Blood pressure of subjects was measured directly after sample collection. **Result**: The blood pressure among the group of age 36-84y was significantly higher ($P < 0.05$) when compared between the smokers and the nonsmokers group, while among the group of age 15-35 was not significant. The results of liver enzymes activities among the group of age 36-84y showed that ALT and ALP were significantly high ($P <0.05$) in the smoker group when compared with the nonsmoker group, while serum AST was not significant ($P>0.05$). However, among the group of age between 15-35 years, the results showed that statistically significant differences ($P<0.05$) were observed in ALT and AST activities for the smoker group when compared with the nonsmoker group. Regarding RBCs count and hemoglobin concentration among both of groups (age between 15-35 & 36-84 years), the results showed that a significant difference in mean RBCs count and hemoglobin concentration were noted between the smokers and the nonsmokers. **Conclusion**: It can be concluded that exposure to cigarette smoking leads to an increase the blood pressure. Cigarette smoking can lead to an increase in liver enzymes activities, RBCs count and Hb concentration.

Keywords: Cigarette smoking, Blood pressure, Liver enzymes, Hemoglobin concentration, West Coast of Libya

1. Introduction

Cigarette smoke contains over 4000 different chemicals, 400 of which are proven to be carcinogenic; it also contains various oxidants such as oxygen free radicals and volatile aldehydes which are probably the major causes of damage to biomolecules. Cigarette

smoking yields chemical substances with high cytotoxic potentials. It consists of many chemicals, including nicotine, tar with its many carcinogens, and gaseous compounds including carbon monoxide. However, shortly the constituents of smoke are contained in either the particulate phase or the gas phase. The particulate phase components include tar, polynuclear hydrocarbons, phenols, cresol, catechol, and trace elements (carcinogens), nicotine (ganglion stimulator and depressor), indole, carbazole (tumor accelerators) [1] and 4-aminobiphenyl [2]. While the gas phase contains carbon monoxide (impairs oxygen transport and utilization), hydrocyanic acid, acetaldehyde, acrolein, ammonia, formaldehyde, and oxides of nitrogen (irritant) nitrosamines, hydrazine and vinyl chloride (carcinogens) [1].

Tobacco smoking is one of the most potent and prevalent addictive habits, influencing the behavior of humans. Smoking is now increasing rapidly throughout the developing world and is one of the biggest threats to current and future world health [3]. Furthermore, while the prevalence of tobacco use has declined among men in some high-income countries, it is still increasing among young people and women [3]. Cigarette smoking is the most common type of tobacco use on average, to date, 47.5% of men and 10.3% of women are current smokers. Tobacco continues to be the second major cause of death in the world.

Some smoke components, such as carbon monoxide (CO), and hydrogen cyanide (HCN) may affect the human respiratory system through their toxic effects on the cilia that line the respiratory tract. At the same time, HCN may cross the placenta and have toxic effects on the growing fetus. In addition, HCN also may cause nerve damage in cigarette smokers with optic neuropathy [4].

Benzene and formaldehyde in the liquid-vapor portion of the smoke also may be carcinogenic. Aside from specific chemical constituents, certain physical-chemical properties of smoke may participate in disease processes. Thus, the pH of the smoke may affect the site and degree of nicotine absorption as well as the smoker's depth of inhalation. The oxidation-reduction state of the smoke can be important because oxidants influence the maturing of cholesterol-laden plaques in the coronary arteries and other blood vessels. Tobacco smoking affects multiple organ systems resulting in numerous so-called tobacco-related diseases. The well-known health risks in tobacco smoking pertain to diseases of the respiratory tract such as COPD and cancer, particularly lung cancer and cancers of the larynx and tongue [5, 6].

The local and systemic effects of acute smoke exposure on oxidative stress and inflammatory mediators were reviewed by van der Vaart *et al*., [7]. Cigarette smoke contains approximately 1017 oxidant molecules per puff [8]. Numerous markers for oxidative damage in smoking have been proposed, including oxidation and nitration of proteins. For example, nitration of tyrosine residues of proteins leads to the production of 3-nitrotyrosine, which may be considered as a marker of nitric oxide (NO)-dependent oxidative damage. Indeed, NO mediated and peroxynitrite-mediated formation of 3-nitrotyrosine is elevated in plasma and platelets of chronic smokers [9, 10].

Smoking causes a variety of adverse effects on organs that have no direct contact with the smoke itself such as liver. The liver is responsible for processing drugs, alcohol and other toxins to remove them from the body. Heavy smoking yields toxins which induce necro-inflammation and increase the severity of hepatic lesions (fibrosis and activity scores) when associated with hepatitis C virus (HCV) [11] or hepatitis B virus (HBV) infection [12]. Cigarette smoking increases the risk of developing hepatocellular carcinoma (HCC) among chronic liver disease (CLD) patients [13] independently of liver status. Association of smoking with HCC irrespective of HBV status has been reported [14].

The association between smoking and liver function in the general population is less clear. A few population studies have examined the relationship between smoking and enzymes measuring liver function such as alanine aminotransferase (ALT), aspartate aminotransferase (AST), and alkaline phosphatase (ALP). Most of these studies have shown

no positive association between smoking and ALT or AST, enzymes more specific to the liver and indicators of liver damage [15].

Smoking is strongly associated with inducing inflammation [16]. Cigarette smoking causes various adverse cardiovascular events [17, 18] and acts synergistically with hypertension and dyslipidemia to increase the risk of coronary heart disease [19]. Smoking causes an acute increase in blood pressure and heart rate and has been found to be associated with malignant hypertension [20]. Nicotine acts as an adrenergic agonist, mediating local and systemic catecholamine release and possibly the release of vasopressin [21].. Long-term passive smoking has been shown to impair endothelium-dependent vasodilatation in healthy adults [22], which is potentially reversible [23].

Cigarette smoking is known to cause an increase in hemoglobin (Hb) concentration that is believed to be mediated by exposure to carbon monoxide [24]. Carbon monoxide bonds to Hb to form carboxyhemoglobin (HbCO), an inactive form of Hb that has no oxygen carrying capacity [25]. To compensate for the decreased oxygen delivery capacity, smokers maintain a higher Hb level than nonsmokers [26].

2. Objectives

The aim of this study is to assess the extent of the adverse effect of cigarette smoking on blood pressure, liver function, and hemoglobin concentration in the male population on the West Coast of Libya.

3. Materials and methods

3.1. Subjects

This study was performed in a medical research center in Zawia city. A total of (200) healthy males were enrolled in the present study. They were divided into four groups according to if they smoking or not and their ages as follows; 50 non-smokers as the control group (age between 15-35 years), 50 smokers (age between 15-35 years), 50 non-smokers as the control group (age between 36-84 years) and 50 smokers (age between 36-84 years) (Figure 1).

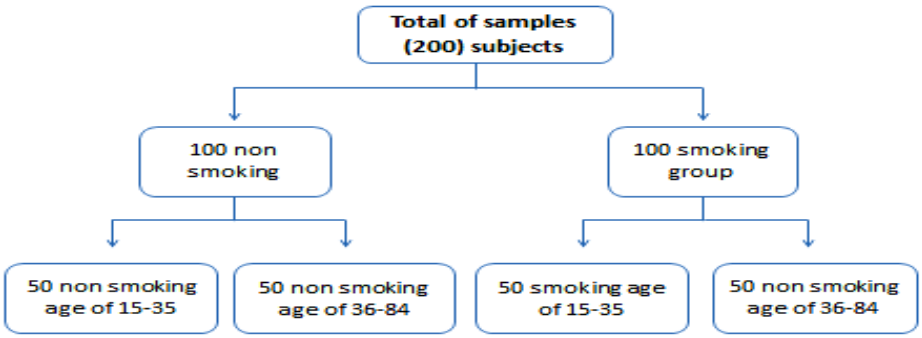

Figure 1. Flow diagram showing the experimental design

3.2. Sample collection

Three milliliter of blood was withdrawn from venous blood by sterile syringes, there capacity 3 ml for each subject. The blood was collected in EDTA tubes to test the Complete

blood count (CBC) and in plain tubes for biochemical assay (concentration of liver enzymes). The blood was allowed to clot in a plain tubes for 20 minute at room temperature. The serum was separated by centrifugation at 3000 rpm for 5 minutes, then each subject's serum was stored in five plain tubes (5 aliquots), frozen at -20 C until the day of the biochemical assay (except for the enzyme studies which were done directly).

3.3. Methods

CBC was analyzed using Mindray Hematology Analyzer (BC 2800), while serum liver enzymes activities were determined by using Mindray Chemistry Analyzer (BS-200).

The blood pressure of subjects was measured directly after sample collection. Systolic blood pressure (SBP), and diastolic blood pressure (DBP) were measured by the arm-cuff method with an automatic upper arm blood pressure monitor (OMRON M2 machine-Japan). The occlusion cuff was placed proximally 22-32cm upper the elbow of the person. After that, about three stable measurements of blood pressure were taken, and the averages of the readings were calculated.

3.4. Statistical Analysis

Results are shown as the mean ± SEM. The data were analyzed using GraphPad Prism 9. The Kolmogorov-Smirnov test was used to assess the normality of the distribution of continuous variables. The statistical significance of differences between groups was evaluated with the independent t-test. A P-value of <0.05 was considered significant for all statistical test.

4. Result

4.1. Blood pressure

The blood pressure among the group of age between 15-35 years was determined, the result indicates that exposure to cigarette smoking in age between 15-35 years does not effect blood pressure, it showed that the blood pressure of the smoker group was not significant ($P > 0.05$) (Table 1) when compared with nonsmokers group as shown in Figure 1.

Table 1. The level of systole and diastole of smokers group compared with nonsmokers group among the age between 15-35y.

Parameters	Nonsmokers	Smokers	P-Value
Systole (mm/Hg)	118.2	118.8	0.8775
Diastole (mm/Hg)	71.90	69.89	0.3493

Whereas, the results show that the cigarette smoking among the group of age 36-84 leads to increase in the blood pressure. The blood pressure among the group of age 36-84y was significant higher ($P < 0.0001$) (Table 2) when compared between smokers and nonsmokers group (Figure 2 and Figure 3).

Table 2. The level of systole and diastole of smokers group compared with nonsmokers group among the age between 36-84y.

Parameters	Non-smokers	Smokers	P-Value
Systole (mmHg)	119.2	137.7	**<**0.0001
Diastole (mmHg)**P-**	71.96	82.82	**<**0.0001

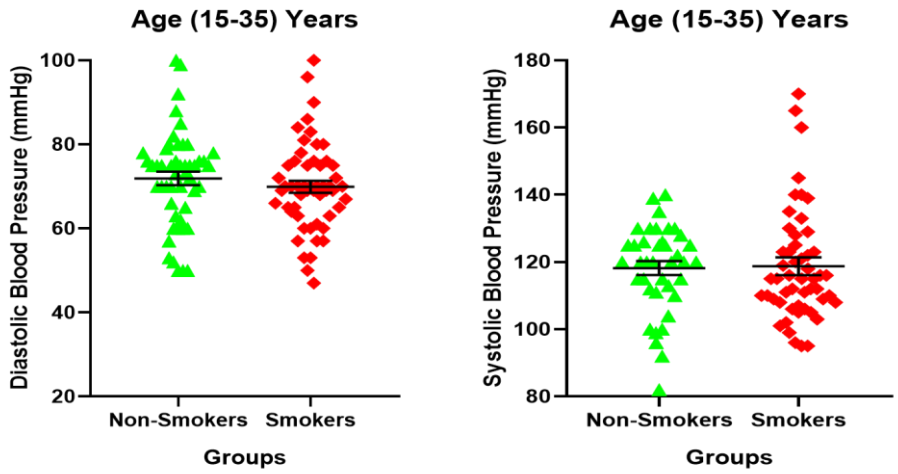

Figure 2. The effect of cigarette smoking on blood pressure among the group of age between (15-35) y.

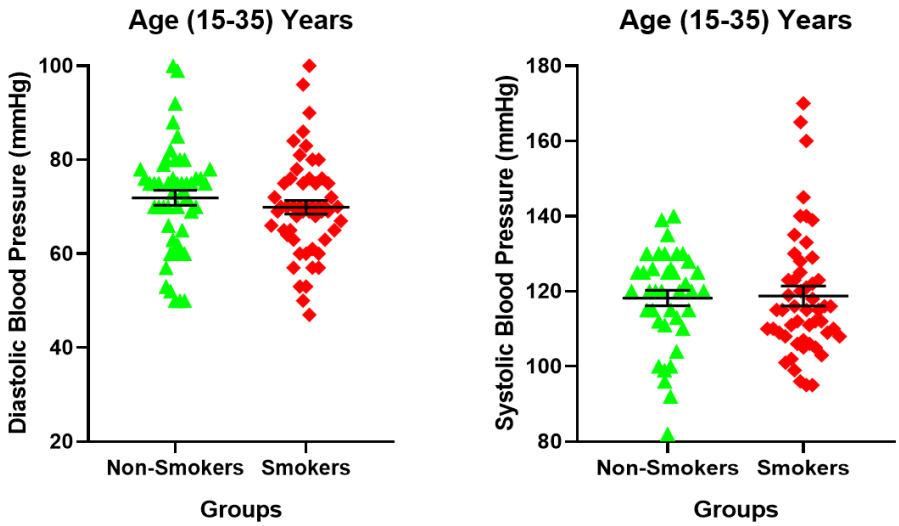

Figure 3. The effect of cigarette smoking on blood pressure among the group of age between (36-84) y.

4.2. Liver enzymes activities:

Regarding liver enzyme activities among the group of age between 15-35 years, the results showed that statistically significant differences ($P<0.05$) (Table 3) were observed in ALT and AST activities and no statistically significant differences ($P>0.05$) was observed in ALP activities for the smoker group when compared with the non-smoker group (Figures 4-6).

Table 3. The serum concentration of ALT, AST & ALK of smokers group compared with non-smokers group among the age between 15-35y.

Parameters	Non-smokers	Smokers	P Value
ALT (U/L)	18.98	23.35	0.0441
AST (U/L)	17.88	21.80	0.0346
ALK (U/L)	85.68	92.85	0.2793

While, among the group of age between 36-84 years, the results showed that ALT and ALK were significant high (*P<0.05*) in smoker group when compared with nonsmoker group, however serum AST was no significant (P value >0.05) in smoker group when compared with nonsmoker group (Table 4 & Figures 4-6).

Table 4. The serum concentration of ALT, AST & ALK of smokers group compared with non-smokers group among the age between 36-84y.

parameters	Non-smokers	smokers	P Value
ALT (U/L)	17.86	27.51	0.0177
AST (U/L)	18.22	31.13	0.0928
ALK (U/L)	77.88	101.7	0.0006

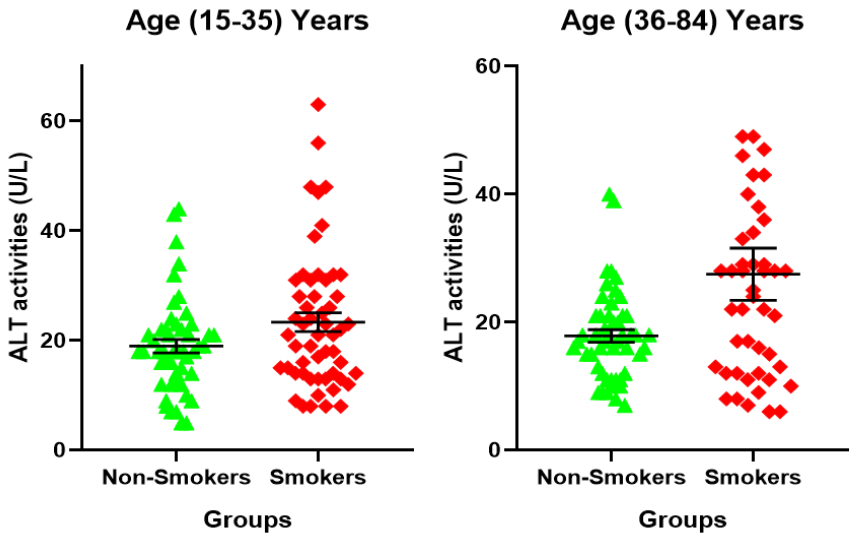

Figure 4. The effect of cigarette smoking on ALT activities among the group of age between 15-35 & 36-84y.

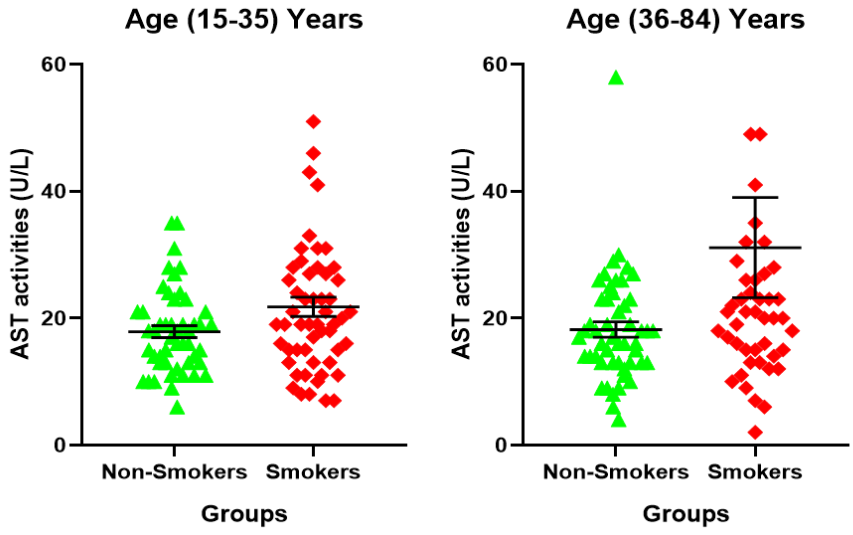

Figure 5. The effect of cigarette smoking on AST activities among the group of age between 15-35 & 36-84y.

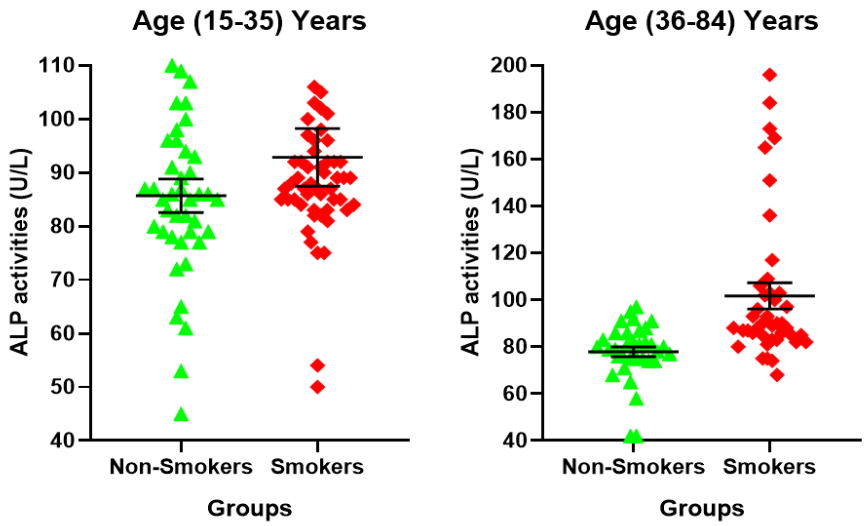

Figure 6. The effect of cigarette smoking on ALP activities among the group of age between 15-35 & 36-84y.

4.3. RBCs count and hemoglobin concentration

Among the group of age between 15-35 years, the results showed that the significant difference in mean RBCs count and hemoglobin was noted between smokers and non-smokers. Figure 6 & 7 illustrates the mean RBCs count and Hb level of smokers and non-smokers of age between 15 – 35 years.

As well as, among the group of age between 15-35 years, the mean RBCs count and Hb level of smokers group was significant higher when compared with nonsmokers group. As shown in Figure 7 and Figure 8.

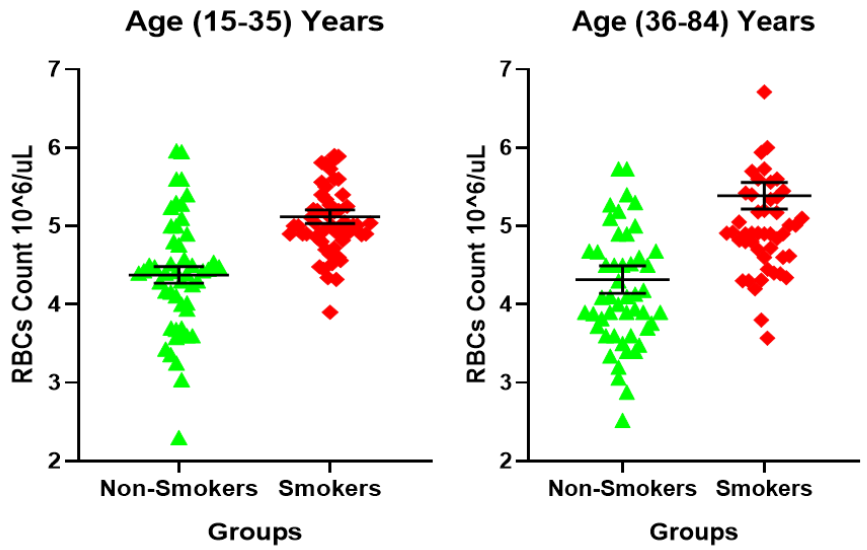

Figure 7. The effect of cigarette smoking on RBCs count among the group of age between 15-35 & 36-84y.

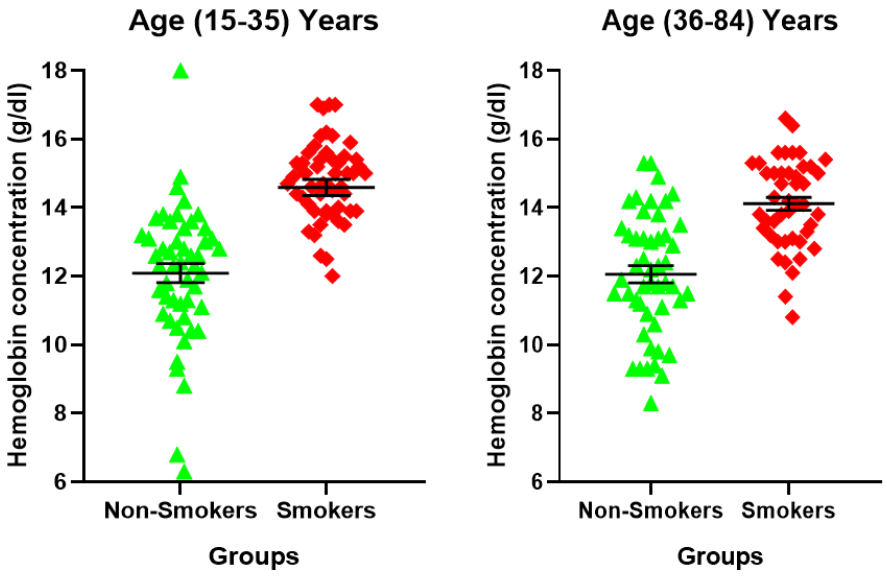

Figure 8. The effect of cigarette smoking on hemoglobin (Hb) among the group of age between 15-35 & 36-84 y.

5. Discussion

Previous studies have indicated that the cigarette smoke cause various adverse cardiovascular events [17, 18], such as hypertension. It has established that the chemical complex mixture of cigarette such as nicotine and tobacco can be causes an acute increase in blood pressure and heart rate [20].

The results from our study show that cigarette smoking among the group of age 36-84 leads to an increase in blood pressure. The current study was in agreement with the previous study which has been reported that blood pressure can be increased during smoking due to the presence of nicotine in tobacco [27]. We think that the nicotine in tobacco smoke increased blood pressure in two ways: 1) it increases heart rate and 2) it causes the narrowing of arteries and blood capillaries. On the other hand, our study indicates that exposure to cigarette smoking in age between 15-35 years does effect on blood pressure. We think this because the period of exposure to nicotine in tobacco smoke was not long among the group of age 36-84 as the group of age 15-35 years.

Cigarette smoke contains a large number of chemical substances with hepatotoxic potential include nicotine and tobacco, which induce inflammation that leads to increased liver enzymes concentration [16].

From the results showed that statistically significant differences ($P<0.05$) were observed in ALT and AST activities and no statistically significant differences ($P>0.05$) was observed in ALP activities for the smoker group when compared with the non-smoker group.

While, among the group of age between 36-84 years, the results showed that ALT and ALK were significant high ($P<0.05$) in smoker group when compared with nonsmoker group, however serum AST was no significant (P value >0.05) in smoker group when compared with nonsmoker group. From the results, it can be suggested that cigarette smoke contain many potential hepatotoxic substances which effect on liver function through its effect on metabolism in liver. The effect of potential hepatotoxic substances on liver metabolism can be leads to disorder on serum level of liver enzymes.

It was found in this study that RBCs count and hemoglobin concentration were increased significantly in cigarette smokers when compared with nonsmokers. The increase in RBCs count and hemoglobin concentration were in both groups (group of age 15-35y

and group of age 36-84y). These results correspond to studies conducted by Sagon and Balcerzak [24] which reported that cigarette smoking causes an increase in Hb concentration that is believed to be mediated by exposure to carbon monoxide bonds to Hb to form carboxyhemoglobin (HbCO). This suggests that HbCO an inactive form of Hb that has no oxygen carrying capacity [25], to compensate for the decrease oxygen delivery capacity, smokers maintain a higher Hb level than nonsmokers [26].

6. Conclusion

Cigarette smoking can cause various adverse cardiovascular events such as hypertension. Our study indicates that exposure to cigarette smoking leads to an increase in blood pressure. Cigarette smoking can affect liver efficiency and functions. Results from the present study showed that cigarette smoking can lead to an increase in liver enzymes activities. It was also noted that cigarette smoking causes an increase in RBCs count and Hb concentration, this study confirmed that RBCs count and Hb levels were significantly higher for smokers than for nonsmokers.

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