Study the effect of physical exercises to decrease the dangerous of smoking By estimated some serological biomarkers

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Abstract

the smoke of cigarette consists of many harmful chemicals materials , such as , carcinogenic and free radicals, thus it cause damage for many organs of the body, also it affect on the lipid concentration of plasma. In this study we estimated the effect of exercises to decrease the dangerous of smoking by determination the level of lipid profile, TAC , Hs-CRP, MDA, AST and ALTtotal protein, in two groups of smokers one with exercise and the other is not, this study estimated the effect of sport in decrease the dangerous of smoking. Results showed that the level of lipid profile (total cholesterol, TG, HDL-C, LDL-C and VLDL-C) have significant differences in all groups of smokers as comparison with control. There is a parallel increased in the level of plasma lipid as increase in number of cigarette per day. except leveles of HDL-C which increased with decrease of duration and intensity of smoking. The liver function test ALT, AST ALP, STB were significant deference at (P value≤0.05) when compare both groups A1 and A2 with control. Total protein, albumin, globulin, have significant deference when compare both groups A1, B1 and A2, B2 with control. TAC, Hs-CRP, MDA have significant deference when compare both groups A1, B1 and A2 with control.

دراسة تأثير ممارسة التمارين الرياضية على تقليل مخاطر التدخين من خلال تقدير بعض الفحوصات المصلية

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الكلمات المفتاحية: التدخين, دهون الدم,مضادات الاكسدة,التمارين الرياضية

الخلاصة:

التدخين هو عامل خطير يسبب تصلب الشرايين، واضطرابات الأوعية الدموية المحيطية, و يؤثر سلبيا على تركيز الدهون في البلازما ومستويات البروتينات. في هذه الدراسة تم تحديد بعض مخاطر التدخين ومعرفة تأثير الرياضة على تقليل مخاطر التدخين من خلال قياس اختبارات وظائف الكبد ومستوى الدهون في الدم ونسبة البروتينات وبعض الفحوصات المناعية في 40 عينة, 20 منهم يمارسون الرياضة (طلاب كلية التربية الرياضية) و20 عينة لا يمارسون الرياضة (طلاب كلية الصيدلة) تراوحت اعمارهم 19-24 سنة. كلا المجموعتين مقسمتين إلى ثلاث مجموعات حسب مدة وكثافة التدخين، كل هذه المجاميع تقارن مع 10 افراد اصحاء غير مدخنين . وتبين الدراسة أن حدة ومدة التدخين تزيد في مستويات الكوليسترول الدهني ذو الكثافة المنخفضة جدا (VLDL) ، والكوليسترول منخفض الكثافة (LDL) , والكوليسترول الكلي في جميع مجاميع المدخنين تقريبا بالمقارنة مع غير

المدخنين. وفي الوقت نفسه لوحظ انخفاض كبير في مستوى كولسترول البروتين الدهني عالي الكثافة (HDL) عند مدخني السجائر مع زيادة كثافة ومدة التدخين. وأظهرت النتائج أيضا أن مستويات ALT و AST في مصل الدم كانت مرتفعة بشكل معنوي في مجموعة المدخنين عند مقارنتها مع مجموعة السيطرة ,اضهر فحص C-reactive protein عالي الحساسية ارتفاع في المدخنين مقارنة مع عينة السيطرة, بينما اضهر فحص بروتين الكلوبيولين والالبومين انخفاض معنوي. بينما مؤشر كتلة الجسم (BMI) والعمر ليس لديهم اختلافات كبيرة عند المقارنة بين المجموعات.

The introduction

smoking of cigarette is the first cause of morbidity and mortality for human, people who die every year because smoking are estimated over two million, the cause of dangerous the smoking can associated with many thing like number of cigarette which smoking per day, the time need for smoking and initiation age [1]. Cigarette smoke have many oxidant like: oxygen free radical which cause the damage for biomolecules, contains various oxidants such as oxygen free radicals and volatile aldehydes which are probably the major causes of damage to biomolecules[2]. There is a relationship between the increase in concentration of plasma lipid increase in risk of die [3]. Cigarette smoke contain many chemicals such as nicotine, and gaseous compounds including carbon monoxide,[4,3]. Smoke of Cigarette cause elevated in the concentration of serum lipid profile, lipid alteration mechanism by smoking occur by nicotine which cause stimulation for sympathetic adrenal system [5]. This will cause secretion of catecholamines and the result will be increasing in lipolysis and increasing in concentration of free fatty acids in serum or plasma, all this actions leading to increase of hepatic FFAs secretion and hepatic triglycerides along with VLDL in the stream of blood [6]. Also smoking cause Fall in oestrogen levels which causing decrease in the level of HDL [7]. The smokers with hyperinsulinaemia, have increase in cholesterol, low density lipid, very low density lipid, and triglyceride due to decreased the activity of lipoprotein lipase [8]. Doing exercise help in prevention of heart-related diseases and many other disease like: hypertension, osteoporosis, diabetes, back pain, respiratory and musculoskeletal, and neurological disorders [9].

Material and Methods

The present study was carried out on a tow groups of young men smokers apparently healthy, first group consist of 20 students from pharmacy college, the second group consist of 20 students from college of physical education. Both groups are compare with control group with consists of 10 adult of mean. Both smokers groups are sub-divided into three groups according to duration and intensity of smoking. All samples of study were age ranges of 19-24 years. The height in cm, weight in kg, for BMI calculated according to the method use by Martin and Crook,

and other information was taken. Samples of blood from individual were taken then waiting for 30 min until clotting, then separated the serum by using centrifugation at 3600 rpm for seven minutes. Then transfer for Alhussainy Hospital to determine: serum ALP, ALT and AST, and (STB), these testes were estimated by using the method of Kind and King [13], then use the method of Reitman and Frankel [14], and method use by Walters and Gerarde [15], respectively. The Serum Protein level were determined by method of Gornall [16], albumin was determined by using method of Doumas method [17], and globulin concentration was determined from the equation of Clarke and Dufour[18]. Also we determined the TAC(mmol/L), Hs-CRP(ng/mL), MDA(mmole/L), lipid profile parameters (total cholesterol, Triglycerides and HDL-Cholesterol). (LDL cholesterol and VLDL cholesterol were estimated mathematically by using Friedewalds formula.

The Statistical analysis: statistical analysis was doing by using SPSS version 14. data were showed as mean±SE using compare mean and one- way ANOVA. ($P \leq 0.05$) were considered significant.

Classification of Subjects

1-Non-Smokers: 10 adult men (age range 19-25 years) apparently healthy, having no history of cigarette were included in this group.

2- Cigarette smokers: were divided into 2 groups according to doing exercises. Then the tow groups were sub-divided to 3 sub groups according to duration and intensity of smoking.

2-1- Smokers of pharmacy college (without exercise) include: Group A1: smokers in heavy (num=7) (over 6 years; smoking 15-20 cigarettes in all day), Group B1: Moderate smokers (num=7) (from 4 to 6 year; smoking 10-15 cigarettes in all day), Group C1: Mild smokers (num=6) (from 2 to 4 years, smoking 5-10 cigarettesin all day).

2--2 Smokers of physical education college (with exercise) include: Group A2: smokers in heavy (num=8) (over 6 years, smoking 15-20 cigarettes in all day), Group B2: Moderate smokers (num=6) (from 4 to 6 years, smoking 10-15 cigarettes in all day), Group C2: Mild smokers (num=6) (from 2 to 4 years, smoking 5-10 cigarettesin all day).

Results and discussion

1-Age and BMI:

All cases of study were in age range 19-25 years, BMI were Convergent, there is no significant differences when compares between groups.

Table (1): Age and BMI in smokers without exercises as compare with control

Parameters	Non-smokers (n=10)	Cigarette smokers (without exercises)			P value
		GroupA1(n=7)	GroupB1(n=7)	GroupC1(n=6)	
Age(years)	24.44±0.53	23.83±0.79	24.0±0.2	25.5±1.14	NS
BMI(kg/m)	24.27±0.37	23.35±0.7	24.05±0.9	23.2±0.63	NS

Values as mean ±S.E

Table (2): Age and BMI in smokers with exercises as compare with control

Parameters	Non-smokers (n=10)	Cigarette smokers (with exercises)			P value
		GroupA2(n=8)	GroupB2(n=6)	GroupC2(n=6)	
Age(years)	24.44±0.53	24.33±0.95	25.11±0.85	24.33±0.6	NS
BMI(kg/m)	24.27±0.37	23.83±0.32	23.93±0.51	23.22±0.63	NS

Values as mean ±S.E

2- Lipid profile:

Table (3) shows the lipid profile level in the three groups of smokers without exercises, (Group-A1), (GroupB1) and (GroupC1). Comparison of these parameters in different groups with control shows that all three groups of smokers have significant differences in levels of T-C, LDL-C, VLDL-C, HDL-C. There is increase as parallel form in the level of lipid profil parameters with the increase in smoking status of intensity and duration, except level of HDL-C which increase with decrease of duration and intensity of smoking. While the level of triglycerides shows significant differences in group A1 as compare with control (nonsmokers), the mean value of lipid profile in smokers was significantly higher at ($P \leq 0.005$) as compared with control.

Table (3): serum lipid profile in smokers without exercises as compare with control

Parameters	Non smokers (n=10)	Cigarette smokers (non exercises)		
		GroupA1(n=7)	Group B1(n=8)	Group C1(n=5)

Total-C(mg/dl)	**145.03±15.28**	**239.51±12.77 [S]**	**229.88±8.92[S]**	**201.66±11.64[S]**
Triglycerides(mg/dl)	**136.21±17.53**	**198.46±5.89[S]**	**190.03±14.29[NS]**	**189.66±36.33[NS]**
HDL-C (mg/dl)	**49.93±3.83**	**34.54 [S]**	**36.02±1.77[S]**	**38.21±2.31[S]**
LDL-C (mg/dl)	**70.41±11.19**	**156.88 [S]**	**148.19±3.75[S]**	**141.67±7.70[S]**
VLDL-C(mg/dl)	**25.44±3.0**	**51.16 [S]**	**43.75±3.8[S]**	**40.33±5.04[S]**

Values as mean ±S.E

Table (4) shows lipid profile levels in three groups of smokers with exercises, (Group-A2), (GroupB2) and (Group C2). comparison of these parameters in different groups with control shows that the level of Triglyceride were significant in the Group A2 only, while T-C, HDL-C, LDL-C, VLDL-C level were significant in both Groups A2 and B2, whereas group C2 have not significant differences for all parameters. There was increase as parallel form in level of lipid profile parameters with the increase in smoking status of intensity and duration, except level of HDL-C which increase with decrease of duration and intensity of smoking.This information confirms the importance of doing physical exercise as an approach to improve the lipid profile. Weight loss by diet or exercise has shown a reduction of triglycerides (TG) levels and elevation of high density lipoprotein-cholesterol (HDL) levels.

Table (4): serum lipid profile in smokers with exercise as compare with control

Parameters	**Non smokers (n=10)**	**Cigarette smokers (exercises)**		
		GroupA2(n=8)	**Group B2(n= 6)**	**GroupC2(n=6)**
Total-C (mg/dL)	**145.03±15.28**	**212.27±43.11 [S]**	**200.95±17.63[S]**	**145.7±19.11[NS]**
Triglycerides(mg/dL)	**136.21±17.53**	**196.5±30.84[S]**	**193.15±19.59[NS]**	**170.76±22.7[NS]**
HDL-C(mg/dL)	**49.93±3.83**	**37.29±1.38 [S]**	**39.15±4.52 [S]**	**46.97±5.17[NS]**
LDL-C(mg/dL)	**70.41±11.19**	**133.33±21.08 [S]**	**111.45±7.62 [S]**	**93.31±9.18[NS]**
VLDL-C(mg/dL)	**25.44±3.0**	**39.0±3.29 [S]**	**40.55±3.52 [S]**	**25.33±2.21[NS]**

Values as mean± SE

Results shows in table (5) that the levels of HDL-C, LDL-C, VLDL-V have significant differences when compares between groups A1 and C2. HDL-C, LDL-C, VLDL-V, T-C levels shows significant differences when compares between groups B1 and C2. Triglyceride show no significant differences when compares between all smokers groups. T-C level have significant differences only when compare between B1 and C2, also A2 and C2 groups. HDL-C have significant differences only when compares between A1 and C2, also B1 and C2. LDL-C level have significant differences only when compares between A1 and C2, also B1 and C2, A2 and C2, A1 and B2, C1 and B2. VLDL-C level have significant differences only when compares between A1 and C2, also B1 and C2, A2 and C2, B2 and C2. Lipid alteration in cigarette smokers perhaps because the stimulation for secretion of catecholamines by nicotine, the secretion of catecholamines causing increasing in the rate of lipolysis then increase in

free fatty acids concentration, this mechanism result increasing the hepatic FFAs and hepatic triglycerides releasing to the blood stream along with VLDL [29].

Table (5): serum lipid profile as comparison between all groups of smokers with exercises and smokers without exercises

Groups	cholesterol	Triglyceride	HDL-C	LDL-C	VLDL-C
Group A1(n=7) GroupC2(n=6)	**[NS]**	**[NS]**	**[S]**	**[S]**	**[S]**
Group B1(n= 7) Group C2(n=6)	**[S]**	**[NS]**	**[S]**	**[S]**	**[S]**
GroupA2(n=8) GroupC2(n=6)	**[S]**	**[NS]**	**[NS]**	**[S]**	**[S]**
GroupB2(n=6) GroupC2(n=6)	**[NS]**	**[NS]**	**[NS]**	**[NS]**	**[S]**
GroupA1(n=7) GroupB2(n=6)	**[NS]**	**[NS]**	**[NS]**	**[S]**	**[NS]**
GroupC1(n=6) GroupB2(n=6)	**[NS]**	**[NS]**	**[NS]**	**[S]**	**[NS]**

3- Liver function parameters ALT, AST, ALP and STB

Result in table (6) shows that the level of ALT have a significant differences when compare groups A1and B1with control, whereas there is no significant differences when compare group C1 with control. Also groupA1 show significant differences for the level of AST when compare with control, while the both groups B1and C1 have no significant differences for AST level when compares with control.

Table (6): serum ALT, AST, ALP and STB levels for smokers without exercises as compare with control

parameters	Control	GroupA1(n=7)	GroupB1(n=7)	GroupC1(n=6)
ALT (IU/L)	**18.66±2.48**	**28.35±6.91[S]**	**28.62±2.71 [S]**	**24.33±2.15[NS]**
AST (IU/L)	**22.11±3.20**	**29.11±4.55 [S]**	**27.37±2.48[NS]**	**25.50±4.06[NS]**
ALP (IU/L)	**70.28±1.3**	**78.5±0.86 [S]**	**73.5±0.78 [S]**	**71.5±0.02 [S]**
STB(mg/dl)	**0.89±0.04**	**0.70±0.13 [S]**	**0.95±0.03 [S]**	**0.80± 0.02 [NS]**

Value as Mean ± SE, P≤0.005

Result in table (7) shows that the level of ALT and AST have no significant differences when compare all groups of smokers (with exercises) with control. Also there is no significant differences for both AST and ALT levels when compared between all groups which have the same duration and intensity of smoking. This will happen because of the action of nitrosative stress (the status that occurs when highly reactive nitrogen-containing chemicals production, such as nitrous oxide), this condition exceed the ability of the human body to neutralize and eliminate them [30]. protein structure will alter because of the reactions occurs by Nitrosative stress thus

interfering with normal body functions [21, 22]. present study shows there is a relationship between the whole number of cigarettes in all day and level of ALT and AST [29].

elevations significant was shown in serum alkaline phosphatase (ALP) when compare groups A1 and B1, with control, there is no significant differences when compare group C1 with control.

Table (7): serum ALT, AST, ALP and STB levels for smokers with exercises as compare with control

parameters	Control	GroupA2(n=8)	GroupB2(n=6)	GroupC2(n=6)
ALT (IU/L)	18.66±2.48	25.0±3.64 [NS]	20.09±3.05 [NS]	20.5±2.98 [NS]
AST (IU/L)	22.11±3.20	23.44±6.64 [NS]	25.64±3.22 [NS]	18.5±2.37 [NS]
ALP (IU/L)	70.28±1.3	77.5±0.56	72.5±0.77	71.5±0.022
STB(mg/dl)	0.89±0.04	0.73±0.03	0.95±0.03	1.03± 0.03

Value as Mean ± SE, P≤0.005

Table (8): serum ALT and AST levels, as compare between each groups of smokers (have same duration and intensity).

Groups	ALT (IU/L)	AST (IU/L)
GroupA1(n=7) and GroupA2(n=5)	NS	NS
Group B1(n= 8) and Group B2(n=9)	NS	NS
GroupC1(n=5) and GroupC2(n=6)	NS	NS

Result in table (9) show that the level of total protein, albumin and globulin have lower significant at (P value ≤ 0.005) in group A2 and B2 as compared with control, serum albumin-globulin ratio (A/G) have no differences when compare smoker groups with control.

Tabl (9): smokers (with exercises) total protein, albumin, globulin and (A/G) ratio as compare with control.

parameters	control	Group A2(n=8)	Group B2(n=6)	Group C2(n=6)
Total protein (g/dl)	8.55±0.4	7.93±0.05 [S]	7.98±0.33 [S]	8.21±0.02 [NS]
Albumin (g/dl)	5.41±0.06	4.91±0.55 [S]	4.22±0.3 [S]	5.20±0.33 [NS]
Globulin (g/dl)	3.14±0.3	3.02±0.04 [S]	3.76±0.05 [S]	3.01±0.34[NS]
A/G (g/dl)	2.43±0.05	2.67±0.06 [NS]	2.41±0.04 [NS]	2.39±0.44 [NS]

Value as Mean ± SE, P≤0.005

Result in table (10) show that there was lower significantly in total protein, albumin and globulin in group A2, B2 and C2 as compared with control, but no differences in albumin-globulin ratio (A/G) when compare smoker groups with control. smoking

causing increase in oxidative stress, Albumin binding to copper ions and scavenging HOCl there for Albumin has antioxidant properties, so the oxidized albumin may be degraded and cleared from the circulation.

Table (10): smokers without exercises total protein, albumin, globulin and (A/G) ratio as compare with control

parameters	control	GroupA1(n=7)	GroupB1(n=7)	GroupC1(n=6)
Total protein(g/dl)	8.55±0.4	6.94±0.05 [S]	7.67±0.33 [S]	7.79±0.02 [S]
Albumin (g/dl)	4.41±0.06	3.40±0.45 [S]	4.32±0.3 [S]	4.23±0.33 [S]
Globulin (g/dl)	4.14±0.3	3.54±0.04 [S]	3.35±0.05 [S]	3.56±0.34 [S]
A/G (g/dl)	2.66±0.05	2.87±0.06 [NS]	2.51±0.04 [NS]	2.49±0.44 [NS]

Value as Mean ± SE, P≤0.005

Result in table (11) show that TAC have higher significant difference when compare group A2 with control, while there are no significant differences for group B2 and C2 when compare with control. This study shows, higher significantly for hs-CRP level at ($p \leq 0.001$) in group A2 when compare with control, also there are significant differences when compare group B2 and C2 with control. hs-CRP level was increased as gradually form with years of smoking. There was higher significantly in Serum malondialdehyde level in group A2 as compared with control, but no significant differences when compare group B2 and C2 with control

Table (11): Serum malondialdehyde (MDA), TAC and Hs-CRP level for smokers with exercises as compare with control

parameters	control	GroupA2(n=8)	GroupB2(n=6)	GroupC2(n=6)
TAC(mmol/L)	1.20±0.33	0.94±0.12 [S]	1.03±0.35 [NS]	1.15±0.46 [NS]
Hs-CRP(ng/mL)	2045±876	2021±354 [S]	2032±203 [S]	2030±823 [S]
MDA(mmole/L)	4.87±0.22	6.98±0.32[S]	4.89±0.44 [NS]	4.99±0.65 [NS]

Value as Mean ± SE, P≤0.005

Result in table (12) show that TAC have higher significant difference when compare group A1 and B1 with control, while there are no significant differences for group C2 when compare with control, hs-CRP level was higher significantly at ($p \leq 0.001$) in group A1 and B1 when compare with control, there are no significant differences when compare C1 with control. Higher significantly was shown in Serum malondialdehyde (MDA) level in group A2 as compare with control, also significant differences was shown when compare group B2 and C2 with control.

Table (12): Serum malondialdehyde (MDA), TAC and Hs-CRP level for smokers without exercises

parameters	control	GroupA1(n=7)	GroupB1(n=7)	GroupC1(n=6)
TAC(mmol/L)	1.20±0.33	0.79±1.12 [S]	0.84±0.65 [S]	1.11±0.76[NS]

Hs-CRP(ng/mL)	**2045±876**	**2020±384 [S]**	**2039±205 [S]**	**2040±813 [NS]**
MDA(mmole/L)	**4.87±0.22**	**7.02±0.11[S]**	**5.89±0.44 [S]**	**5.02±0.05 [S]**

Value as Mean ± SE, P≤0.005

The reasons for why smokers have lipid peroxidation at higher level as compared with control are smokers are person who inhaled smoke of cigarette will be in oxidation in his body because of the radicals in smoke which increase oxidative damage [15], increase the antioxidants which protect the body from the damage of oxidative such as lipid peroxidation has been observed[16]. Oxidative stress will indused by smoke of cigarette, because of the smoke will induce NADPH oxidase then the smoke will be able to decreasing antioxidant defenses, this mechanism actions causing lipid peroxidation, then hepatocellular damage will occur [17]. In this our result, lower significantly shown in level of total antioxidant capacity in smokers as compare with control. total antioxidant capacity reduced, that is mean that the smokers had an increase in free radicals production [9], which corresponds with the results of our study. Rouzbahani *et al* [11], found that Serum malondialdehyde level was elevated in smokers more than control, this agree with our results. the end product of lipid peroxidation processes is Malondialdehyde, comes from many pathway, including peroxidation of endogenous lipid, during prostaglandin H2 and thromboxane (TXA2) synthesis, malondialdehyde will production in platelets and other sources [19, 22]. the marker for oxidative stress, is lipid peroxidation, which cause the damaging for living cells and tissues then have a role in cancer and inflammatory diseases [12,13]. free radicals will result from lipid peroxidation, this one will causing peroxidation starting over. multi double bond unsaturated fatty acid peroxidation will produce Malondialdehyde and is used as a measure for determine lipid peroxidation [28]. unstable between the production of free radicals and reactive oxygen species with antioxidant agents in the body can be resulted defect in the metabolism of fats, proteins and carbohydrates[24]. the smoking of cigarette cause inflammation development which can be estimated by hs-CRP level. The initiation of Inflammatory response will increase the polymorphonuclear neutrophil number from the bone marrow, then these cells will cause secretion of proinflammatory cytokine IL-6 and (TNF) [24]. These cytokines will attach to receptor of hepatocyte surface and increase the concentration of hs-CRP in serum [92],[23]. Nicotine which reduces the appetite and alters patterns of feeding, causing reduced in weight of body and decrease body mass index in persons who smoking [26]. Nicotine also cause constricting blood vessels then raise in blood pressure [30],[27].

Conclusion

Result of our study shows there is a relationship between higher serum lipids and smoking of cigarette. It proved that the changes in the lipid profile was associated with the changes in years and status of smoking. smoking can affect the liver functions, through its effect on ALT and AST. Also smoking effect in development the inflammation causing elevated in hs-CRP level. Doing exercise have a good effect to decrease the dangerous of smoking through decrease the lipid profile and increase in antioxidant mechanism, then decrease the free radicals and other harmful chemicals compound coming from smoke.

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