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Evaluation of The Effect of Smoking and Chronic obstructive pulmonary disease(COPD) on Lipid profile

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Abstract

Background: (COPD) disease is consider an important disease that threat our life in recent years and its result mostly from smoking .the patients who affected by this disease will be at higher risk of death caused by frequent exacerbation ,also chronic obstructive pulmonary disease may influence the lipid profile regardless of smoking status.

Objective: To assessment the effect of COPD and smoking on lipid profile.

Patients and Methods: This case control study included 50 patients suffer from (COPD) and other age- and sex-matched 50 smokers (≥ 20 pack/yeas) healthy people. Demographic and clinical data were collected through a direct interview with each participant. Fasting lipid profile was measured according to the standard protocols.

Results: Mean serum level of TG and vLDL in smokers with COPD was 192.4±46.91 mg/dl and 61.8±12.23 mg/dl, respectively compared with 135.5±23.8 mg/dl and 32.68±14.8 mg/dl, respectively in smokers without COPD, with significant differences. Furthermore, the frequency of abnormal triglyceride value among smokers with COPD was 36% compared to 12% among smokers who don't have this disease . (OR= 4.12. 95%CI= 1.47-11.55, p= 0.007). Similarly, the frequency of abnormal vLDL among smokers with COPD was 18% compared to 4.12% among smokers without COPD (OR= 5.27. 95%CI= 1.08-25.8, p= 0.040). In smokers with COPD, smoking displayed a positive significant correlation with TG (r= 0.325, p= 0.021) and vLDL (r= 0.333, p= 0.018). In smokers without COPD, smoking had a positive and marked association with TC (r= 0.377, p= 0.007)

Conclusion: COPD is associated with negative effects on the lipid profile. In particular, triglycerides and very low-density lipoprotein cholesterol are mostly affected. Aside from COPD, smoking can also be associated with elevated lipids/lipoproteins that exceed normal limits at times.

Keywords: COPD , smoking

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Introduction

Cigarette inhalation is classified one of the causes of early mortality in about 6 million human over the world [1,2] . Most of these mortality happen in people who addicted

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smoking and those who affected by smoking in indirect way such as living in smoking environments. Also expected that smokers who continue for this bad habit lose approximately ten years of their life expectancy and begin to have many co-morbidities in compared with those who totally nonsmoker [3,4].

Major reasons for mortality in smokers is cancers [6-9] (mainly lung cancer) and respiratory diseases (mainly chronic obstructive pulmonary disease – COPD)[6,7] and cardiac diseases (mainly coronary heart disease) [7,8]. Furthermore, smoking is an important risk factor for stroke, visual and hearing loss, back pain and bone mass loss [5,6].

Tobacco smoke contains toxic gas which is carbon monoxide (CO). This gas compete with the oxygen on hemoglobin (Hb) molecules and lead to desaturation and as a compensatory mechanism the body produce high number of red blood cells to accommodate its need of oxygen and this increase the risk of thrombosis [9,10].

Patients and Methods

Design and Settings

This research included group of patients who have COPD who were attending the outpatient clinic at Baghdad Teaching Hospital. These patients diagnosed to have COPD according to GOLD criteria and on regular treatments. The study was approved by Iraqi Council for medical specializations.

Inclusion criteria

Adult aged 40 years or over at time of diagnosis.Patients who are don't have decompensating for more than three months prior to study and free of clinical signs of exacerbation and take their medication in regular pattern.

Exclusion criteria :Patients with

-History of chronic lung diseases such as asthma ,interstitial lung diseases , bronchiectasis, tuberculosis.

-History of inflammatory diseases such as rheumatoid arthritis, inflammatory bowel diseases.

-History of chronic medical debilitating illness such as liver cirrhosis, end stage renal failure and malignancy.

-History of frequent use of drugs that affect the lipid profile such as statins ,diuretics ,anticonvulsants.

Data Collection

Demographic data consist of age, sex, body mass index (BMI) smoking (pack/year) and residence; and clinical characteristics including comorbidities, and the frequency of exacerbation were collected through a direct interview with each participant.

Lipid Profile Assay

(5ml) of the venous blood was taken as a sample from all the patients in the fasting state of more than 8 hrs. The blood sample was centrifuged at 3000 rpm for 15 min; sera separated and stored at -80 °C until assayed and studied. Total cholesterol, High density lipoprotein (HDL) and triglyceride (TG),low density lipoprotein(LDL) ,very low density lipoprotein(VLDL) and total cholesterol levels were determined with Konelab auto analyzer (S.N: D13082) using Randox test reagents.

Statistical Analysis

Characteristics features of the patients with and without COPD according to their gender, age, and associated comorbidities, occurrence of pneumonia, CVD, malignancies, and

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mortality was examined using chi-squared tests for categorical variable and analysis of variance (ANOVA) test for continuous variables.

Results

Demographic Characteristics of the Study Population: The average age of smokers with COPD was 54.41±12.7 years (range 41-59 years) that not largely differ from that of smokers without COPD (50.48±11.61 years, range 37-68 years). Likewise, the two groups were comparable regarding BMI, marital status, educational levels and residence with no significant differences. However, smokers with COPD were much higher tobacco consumption (44.4±6.87 packs/years) than smokers without COPD (22.8±2.89 packs/years) with highly significant difference Table (1).

Table (1): Association of demographic factors

Variables	Smokers with COPD (n=50)	Smokers without COPD (n=50)	p-value
Age, years			
Mean±SD	54.41±12.7	50.48±11.61	0.413
Range	41-59	37-68	
BMI, kg/m^2			
Mean±SD	25.42±3.52	26.72±3.56	0.812
Range	20.75-37.43	21.8-38.62	
Marital status			
Single/divorced	9(18%)	8(16%)	0.790
Married	41(82%)	42(84%)	
Educational level			
Primary	14(32%)	11(26%)	0.621
Secondary	24(46%)	23(50%)	
Higher	12(22%)	16(24%)	
Smoking, pack/year			
Mean±SD	44.4±6.87	22.8±2.89	**<0.001**
Residence			
Urban	28(56%)	31(62%)	0.542
Rural	22(44%)	19(38%)	

Lipid Profile

Generally, all included parameters of lipid profile displayed higher levels in smokers who affected by COPD than smokers without COPD. However, only two parameters showed significant differences. Mean serum level of TG and vLDL in s

mokers with COPD was 192.4±46.91 mg/dl and 61.8±12.23 mg/dl, respectively compared with 135.5±23.8 mg/dl and 32.68±14.8 mg/dl, respectively in smokers without COPD, with significant differences Table (2).

Diyala Journal of Medicine

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Table (2): Lipid profile in smokers with and without COPD

Variables	Smokers with COPD (n=50)	Smokers without COPD (n=50)	p-value
Total Cholesterol (mg/dl)			
Mean±SD	218.7±38.2	184.78±41.84	0.118
Range	118-312	87-302	
Triglycerides (mg/dl)			
Mean±SD	192.4±46.91	135.5±23.8	**0.021**
Range	127-362	98-286	
LDL (mg/dl)			
Mean±SD	134.2±32.7	117.12±61.43	0.142
Range	88- 189	86- 177	
vLDL (mg/dl)			
Mean±SD	61.8±12.23	32.68±14.8	**0.011**
Range	18- 86	13-78	
HDL (mg/dl)			
Mean±SD	46.53±11.6	39.91±9.18	0.605
Range	36- 61	32- 67	

* LDL: low density lipoprotein-cholesterol, vLDL: very low density lipoprotein-cholesterol, HDL: high density lipoprotein-cholesterol. COPD: chronic obstructive pulmonary disease

Comparison Based on Normal Range

Almost similar results were obtained when the two groups were compared based on the normal range of lipid profile parameters. In univariate analysis, the frequency of smokers with COPD with abnormal values was more than smokers without COPD. However, a significant difference was reported only for TG and vLDL. The frequency of abnormal triglyceride among smokers with COPD was 36% compared to 12% among smokers without COPD (OR= 4.12. 95%CI= 1.47-11.55, p= 0.007). Similarly, the frequency of abnormal vLDL among smokers with COPD was 18% compared to 4.12% among smokers who don't have COPD (OR= 5.27. 95%CI= 1.08-25.8, p= 0.040) as shown in Table (3).

Table (3): Comparison based on normal range (univariante analysis

Variables	Smokers with COPD (n=50)	Smokers without COPD (n=50)	p-value	OR(95%CI)
TC (mg/dl)				
≤ 200	39(78%)	46(92%)	0.059	1.0
>200	11(22%)	4(8%)		3.24(0.95-11.0)
TG (mg/dl)				
≤ 165	32(64%)	44(88%)	**0.007**	1.0
>165	18(36%)	6(12%)		4.12(1.47-11.55)
LDL (mg/dl)				
≤ 130	43(46%)	48(96%)	0.10	1.0
>130	7(14%)	2(4%)		3.9(0.77-19.83)
vLDL (mg/dl)				
≤55	41(82%)	48(96%)	**0.040**	1.0
>55	9(18%)	2(4.12%)		5.27(1.08-25.8)
HDL (mg/dl)				
≥40	45(90%)	44(88%)	0.750	1.0
< 40	5(10%)	6(12%)		1.22(0.35-4.32)
Smoking (pack/year)				
≤ 25	4(8%)	45(90%)	**<0.001**	1.0
>25	46(92%)	5(10%)		103(26.1-410.3)

* TC: total cholesterol, TG: triglycerides, LDL: low density lipoprotein-cholesterol, vLDL: very low density lipoprotein-cholesterol, HDL: high density lipoprotein- cholesterol, OR: odds ratio, CI: confidence interval. COPD: chronic obstructive pulmonary disease

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As the smoking level (packs/year) was significantly higher among smokers with COPD than smokers without COPD, lipid profile parameters between the two groups were adjusted for smoking level in a multivariate analysis. Both TG and vLDL remains significant in this analysis indicating that they are independent factors associated with COPD Table (4).

Table (4): Comparison based on normal range (multivariate analysis)

Variables	p-value	OR(95%CI)
TC (mg/dl)		
≤ 200	0.078	1.0
>200		2.15(0.88-9.4)
TG (mg/dl)		
≤ 165	**0.014**	1.0
>165		3.48(1.36-14.22)
LDL (mg/dl)		
≤ 130	0.122	1.0
>130		2.9(0.74-16.43)
vLDL (mg/dl)		
≤55	**0.034**	1.0
>55		5.8(1.13-2356)
HDL (mg/dl)		
≥40	0.618	1.0
< 40		1.44(0.47-6.51)
Smoking (pack/year)		
≤ 25	**<0.001**	1.0
>25		89.4(18.3-312.6)

*TC: total cholesterol, TG: triglycerides, LDL: low density lipoprotein-cholesterol, vLDL: very low density lipoprotein-cholesterol, HDL: high density lipoprotein-cholesterol, OR: odds ratio, CI: confidence interval

Discussion

Compared with other international studies, Mitra, *et al*.[11-13] we found apparently increase in serum levels of all lipoproteins TC, TG, LDL in COPD cases in comparable with controls. HDL levels apparently low in cases comparable with controls. Attaran D, *et al*.[14-16] assess the serum lipid profile in patients affected by COPD and the result was statistical change in TC and TG levels between patients and controls but no any of the lipid parameter express any relation with severity of airflow limitation assessed by FEV1. Same findings was reported in another studies [17-19] .Zafirova-Ivanovska *et al*.[20,-22] reported that hypercholesterolemia in COPD group clearly elevated with disease severity. In contrast, Basili *et al*.[23-25] measured serum levels of TC, HDL-C, LDL-C and TG in ninety patients with COPD and in ninety normal subjects matched for age, sex and smoking habit. The study revealed almost similar values of these compounds between patients and controls. Furthermore, Fekete *et al*. [26-28] observed almost identical values of TC, LDL-C, and HDL-C between COPD patients and controls. This discrepancy between different studies may explained, at least in part, by the variation in study design, BMI, sample size and disease severity and duration.

There are some hypotheses by which the COPD can influence the lipid profile. The most reasonable hypothesis assumes that some medications used for treatment of COPD can influence the lipid profile. For example β-2 agonists, might be responsible

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for an increased level of HDL[29-31]. The other interesting result in the present study was that smoking itself associated with increased the most components of lipid profile because most of these components in smokers without COPD were within normal limits. Furthermore, some individuals in this groups showed abnormal values for almost all lipid profile components.

Many previous studies have reported the similar findings that smoking was associated with a higher lipid profile than non-smokers. In an Indian study 25 in 9 compared serum lipid profile in chronic smokers with healthy non-smokers. Mean serum levels of TC, TG, LDL-C vLDL-C were significantly higher in smokers than non-smokers. In another study, 'a total of 200 age and sex matched subjects comprising of 100 healthy non-smokers as controls and 100 healthy smokers as cases were investigated for lipid profile. Serum levels total TC, TG, VLDL and LDL cholesterol very significantly increased and decrease in HDL cholesterol in smokers when compared to non-smokers[34].In another study, the lipid profile was measured from 100 selected smokers and nonsmokers and the study showed that as the intensity and duration of smoking increases a significant increase in the levels of vLDL-C, LDL-C, TG and TC were noted in almost all groups of cigarette smokers as compared to nonsmokers. Simultaneously a significant reduction in the level of HDL-C was observed [33]. National Health and Nutrition Examination Survey (NHANES) data reveal that the incidence of high values of TG was 3.7% more in smokers than nonsmokers. The chance of having abnormal values for smokers was a 60% higher for HDL and 31% higher for TG than nonsmokers. In contrast, some other study did not find any significant difference [13].

Although the way by which the smoking change the serum lipid values is not completely known till now , many hypothesis can be suggested. For example , increase the level of catecholamine can led to a surge in circulating free fatty acids that give stimulation for hepatic TG production and cause changes in lipid metabolism [25].other example is the smoking led to reduce the action of lecithin-cholesterol acyltransferase, the enzyme in charge of esterifying free cholesterol, that led to change in lipid metabolism[14] other suggestion that the higher TG level in smokers is due to the TG metabolism occur at slow rate in smokers, and the activity of lipoprotein lipase is decreased in smokers[16].

Conclusions

Chronic obstructive pulmonary disease is associated with effect on lipid profiles. In particular, triglycerides and very low density lipoprotein cholesterol are mostly affected. Smoking also associate with elevated lipid/lipoproteins which sometime skip the normal limits. Triglyceride and vLDL significantly associated the intensity of smoking.

Recommendations

Patients with COPD should have a regular checking for their lipid profile in order to reduce the risk of cardiovascular diseases. Cessation of smoking is important to reduce the adverse effect on lipid profile.

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ORIGINAL RESEARCH
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Ethical clearance: A written consent from each participant was obtained prior to data collection after explaining the aim of study. Each patient was given the complete unconditioned choice to withdraw anytime.

Conflict of interest: Nill

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تقييم تأثير التدخين ومرض الانسداد الرئوي المزمن على بروفايل الدهون

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الملخص

خلفية الدراسة: مرض الانسداد الرئوي المزمن هو أحد مشاكل الصحة العامة الرئيسية في المجتمعات الحديثة ويحصل اساسا بسبب التدخين. المرضى الذين يعانون من مرض الانسداد الرئوي المزمن معرضون لخطر متزايد من دخول المستشفى والوفاة بسبب أمراض القلب والأوعية الدموية. لذلك ، قد يؤثر مرض الانسداد الرئوي المزمن على بروفايل الدهون بغض النظر عن حالة التدخين.

اهداف الدراسة: : لاستقصاء العلاقة بين مرض الانسداد الرئوي المزمن ببروفايل الدهون في المرضى المدخنين الذين يعانون من مرض الانسداد الرئوي المزمن وبدونه.

المرضى والطرائق: تضمنت هذه الدراسة المقطعية ٥٠ مريضًا تم تشخيصهم بمرض الانسداد الرئوي المزمن بالاضافة الى ٥٠ مدخنا (≤ ٢٠ عبوة / سنة) مطابقا بالعمر والجنس الذين ليس لديهم أي دليل على مرض الانسداد الرئوي المزمن. تم جمع البيانات الديموغرافية والسريرية من خلال المقابلة المباشرة مع كل مشارك. تم قياس بروفايل الدهون الصائم وفقًا للبروتوكولات القياسية.

النتائج: بلغ متوسط مستوى المصل من الدهون الثلاثية وكوليسترول البروتين الدهني منخفض الكثافة للغاية لدى المدخنين المصابين بمرض الانسداد الرئوي المزمن ١٩٢,٤ ± ٤٦,٩١ مجم / ديسيلتر و ٦١,٨ ± ١٢,٢٣ مجم / ديسيلتر ، على التوالي مقارنة بـ ١٣٥,٥ ± ٢٣,٨ مجم / ديسيلتر و ٣٢,٦٨ ± ١٤,٨ مجم / ديسيلتر ، على التوالي لدى المدخنين غير المصابين بمرض الانسداد الرئوي المزمن ، وبفوارق معنوية. علاوة على ذلك، كان معدل تكرار الدهون الثلاثية غير الطبيعية بين المدخنين المصابين بمرض الانسداد الرئوي المزمن ٣٦٪ مقارنة بـ ١٢٪ بين المدخنين غير المصابين بمرض الانسداد الرئوي المزمن (OR = 4.12. 95٪ CI = 1.47-11.55، p = 0.007) وبالمثل ، كان معدل تكرار كوليسترول البروتين الدهني منخفض الكثافة للغاية غير الطبيعي بين المدخنين المصابين بمرض الانسداد الرئوي المزمن ١٨٪ مقارنة بـ ٤٪ بين المدخنين غير المصابين بمرض الانسداد الرئوي المزمن ((OR = 5.27. 95٪ CI = 1.08-25.8, p=0.04) . في المدخنين المصابين بمرض الانسداد الرئوي المزمن ، أظهر التدخين ارتباطًا إيجابيًا معنويًا مع الدهون الثلاثية (r= 0.325, p= 0.021) و كوليسترول البروتين الدهني منخفض الكثافة للغاية (r= 0.333, p= 0.018) في حين كان للتدخين علاقة ارتباط موجب (r= 0.377, p= 0.007) في المدخنين الذين لايعاونون من مرض الانسداد الرئوي المزمن.

الاستنتاجات: يقترن مرض الانسداد الرئوي المزمن بتأثيرات سلبية على بروفايل الدهون. على وجه الخصوص ، تتأثر في الغالب الدهون الثلاثية وكوليسترول البروتين الدهني منخفض الكثافة للغاية. بصرف النظر عن مرض الانسداد الرئوي المزمن ، يمكن أن يرتبط التدخين أيضًا بارتفاع نسبة الدهون / البروتينات الدهنية التي تتجاوز الحدود الطبيعية في بعض الاحيان.

الكلمات المفتاحية: مرض الانسداد الرئوي المزمن،التدخين

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