Serum Lipid Profile in Tobacco Users

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In Bangladesh 43.3% of adults (41.3 million) use tobacco in smoking and or smokeless form. Immediate availability and the low price gives rise to high consumption of chewing tobacco. This may affect lipid profile. Consumption of tobacco products are the world's leading preventable cause of death. To observe lipid profile level in tobacco users. This cross-sectional study was conducted from January 2014 to January 2015 in the Department of Physiology, Rangpur Medical College, Rangpur. A total number of 150 subjects were selected, among them 50 were apparently healthy subjects of non-tobacco chewer non-smoker: group A (Control), 50 were apparently healthy tobacco chewer non-smoker subjects: group B and 50 were apparently healthy tobacco chewer smoker subjects: group C. The subjects were collected from different areas of Rangpur district. Lipid profile levels were determined and data were analyzed by one – way ANOVA (post Hoc test) and Pearson's Correlation Coefficient 'r' test. In this study mean lipid profile levels were significantly higher (P<0.001) in tobacco chewer non-smoker and tobacco chewer smoker subjects than those of control subjects. This study reveals that lipid profile levels tend to rise with tobacco use.

[Dinajpur Med Col J, 2017 Jul; 10 (2):314-328]

Key words: Tobacco, Tobacco chewer, Smoker, Lipid profile

Introduction

There are two kinds of commonly used tobacco products in Bangladesh i.e. smoking and smokeless tobacco products. Smoking tobacco products in Bangladesh include manufactured cigarettes, bidis, hand-rolled cigarettes, pipes, cigars, water-pipes or hukkah. Smokeless tobacco products include a wide range: betel quid with zarda, zarda only or betel quid with sadapata; pan masala with tobacco; sadapata chewing; gul, khoinee. However, women usually used smokeless tobacco but rarely smoke or both. In Bangladesh 43.3% of adults (41.3 million) use tobacco in smoking and / or smokeless form. Currently more than 5 million people die globally each year due to tobacco related illness, the figure expected to increase to 8.3 million by 2030. Tobacco-attributable deaths are projected to decline by 9% between 2002 and 2030 in high income but to double from 3.4 million to 6.8 million in low and middle income countries.[1] When a person smoke a cigarette or chew a quid, the person is exposing himself or herself to 4,000 and more hazardous and cancer causing chemicals.[2] Nicotine is one of the main constituent of tobacco. Nicotine is absorbed

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in substantial quantities from smokeless tobacco and could contribute to the adverse consequences of smokeless tobacco use.[3] Nicotine together with cholesterol and other fat deposits contribute to the hardening of the arteries, damaging linings of the blood vessels which develop through the years.[4] Nicotine is one of the main constituent of tobacco. Nicotine is absorbed in substantial quantities from smokeless tobacco and could contribute to the adverse consequences of smokeless tobacco use.[5] Chronic systemic exposure to nicotine could contribute to accelerated coronary artery disease, acute cardiac ischemic events and hypertension. Nicotine also stimulate the production of adrenaline which causes increase heart rate, constrict blood vessels, increase peripheral resistance, poor blood circulation and blood clots lead to high blood pressure, stroke and heart attacks.[6] Other effects of adrenaline which acts through the β_1 receptor leads to lipolysis in adipose tissues. This leads to increase in serum concentration of free fatty acids, total cholesterol, triglycerides and LDL-C.[7] Some researchers found highly significant decrease in HDL-C in tobacco users.[8] The result of this study will increase awareness about the changes in lipid profile & ill effects of tobacco use.

Methods

This cross-sectional study was conducted from January 2014 to January 2015 in the Department of Physiology, Rangpur Medical College, Rangpur. The study protocol was approved by the Ethical Committee. Study subjects were selected by the following purposive sampling procedure. The study was approved by the ethical review committee of Rangpur Medical College. A total number of 150 subjects were enrolled in the study of age group between 20 – 45 years. Among them 50 were apparently healthy subjects of non-tobacco chewer non-smoker (group-A), 50 were apparently healthy tobacco chewer non-smoker subjects (group–B) and 50 were apparently healthy tobacco chewer smoker subjects (group–C). Age and sex matched 50 apparently healthy subjects (group–A) was served as control group. The experimental subjects were recruited from Rangpur city and outskirts. Any subject suffering from diabetes mellitus and other chronic diseases (liver, kidney and heart) or having obvious congenital anomalies were excluded from the study. After selection of subjects, the objectives and the procedures of the study were explained in detail to the subjects. They were informed about the risk and benefit before enrollment of the study. Then the informed written consent of the subjects were obtained from the willing subjects. Tests for determination of serum total cholesterol, triglyceride, low density lipoprotein and high density lipoprotein cholesterol were carried out as early as possible. Serum total cholesterol was determined by enzymatic colorimetric (CHOD-PAP) method; serum triglyceride was determined by enzymatic colorimetric (GPO-PAP) method; serum low density lipoprotein cholesterol was determined by enzymatic colorimetric (Fried Wald) method & serum high density lipoprotein cholesterol was determined by enzymatic colorimetric (Precipitant) method.

All data were recorded systematically in a preformed history sheet and all statistical analysis was done by computer using the software SPSS 17.0 version for windows. Comparison of serum total cholesterol, serum triglyceride, serum low density lipoprotein cholesterol and serum high density lipoprotein cholesterol levels in tobacco chewer non-smoker and tobacco chewer smoker subjects with control group were done by one-way ANOVA (post–Hoc) 'F' test and Pearson's correlation coefficient 'r' test. In the interpretation of results < 0.05 level of probability (p) was accepted as significance.

Results

Table I: Shows mean weight, height and BMI of the study subjects of different groups

Group	Age Groups in Years	Weight Kg	Height m^2	BMI Kg/m^2
A(n=50)	20-45	56.5	2.36	23.9
B(n=50)	20-45	57.5	2.32	24.7
C(n=50)	20-45	58	2.44	23.7

A= Apparently healthy subjects of non-tobacco chewer non-smoker (Control).

B= Apparently healthy subjects of tobacco chewer non-smoker (Experimental).

C= Apparently healthy subjects of tobacco chewer smoker (Experimental).

n= Number of subjects.

L= Lowest value.

H= Highest value

#= Normal range of BMI 18.5 – 24.9.[20]

Serum Total Cholesterol

The mean ± SD serum total cholesterol levels were 194.96 ± 34.652 mg/dl in group A, 267.14 ±36.464 mg/dl in group B and 272.82 ± 73.374 mg/dl in group C. There was significant difference (p<.001) between group A and group B, group A and group C. But there was no significant difference (p>.05) between group B and group C.[Table I, Fig-1 & Fig-2]

Statistical analysis of the results of serum total cholesterol levels shown in Table II. Analysis between the groups done by One – way ANOVA (post Hoc Test):

Table II **:** Serum total cholesterol levels in group A (control), group B and group C (experimental)

Groups	Mean ± SD mg/dl Range (L- H) mg/dl	'p' value
A / B (n=50/ (n=50)	194.96 ± 34.652 / 267.14 ± 36.464 (128 - 280) / (155 - 461)	0.001***
A / C (n=50/ (n=50)	194.96 ± 34.652 / 272.82 ±73.374 (128 - 280) / (144 - 460)	0.001***
B / C (n=50/ (n=50)	267.14 ± 36.464 / 272.82 ± 73.374 (155 - 461) / (144 - 460)	0.927^{NS}

A= Apparently healthy subjects of non-tobacco chewer non-smoker (Control).
B= Apparently healthy subjects of tobacco chewer non-smoker (Experimental).
C= Apparently healthy subjects of tobacco chewer smoker (Experimental).
n= Number of subjects.
SD= Standard deviation.
***= p<0.001
NS= p>0.05
L= Lowest value.
H= Highest value
#= Normal range of serum total cholesterol level is < 200 mg/dl.[21]

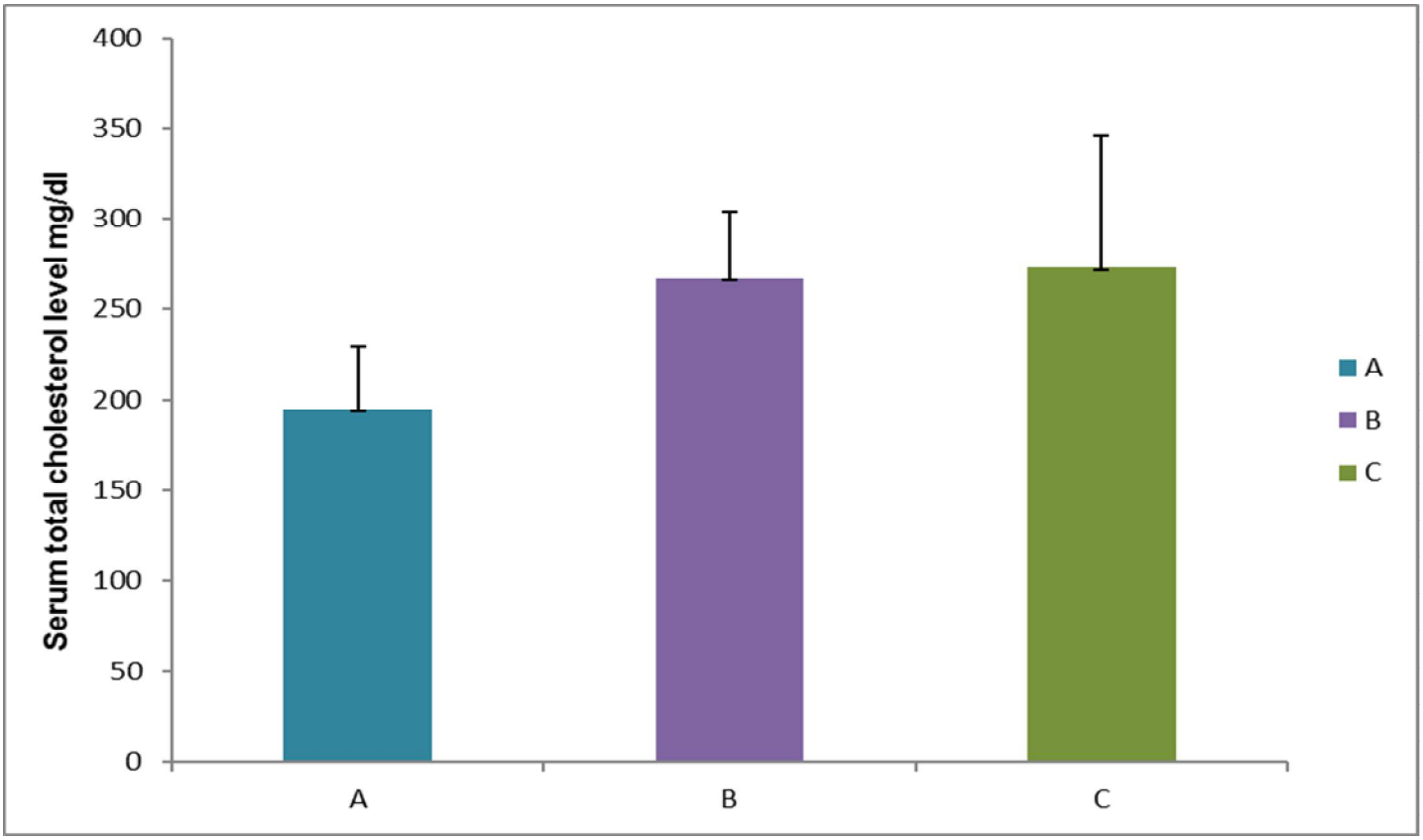
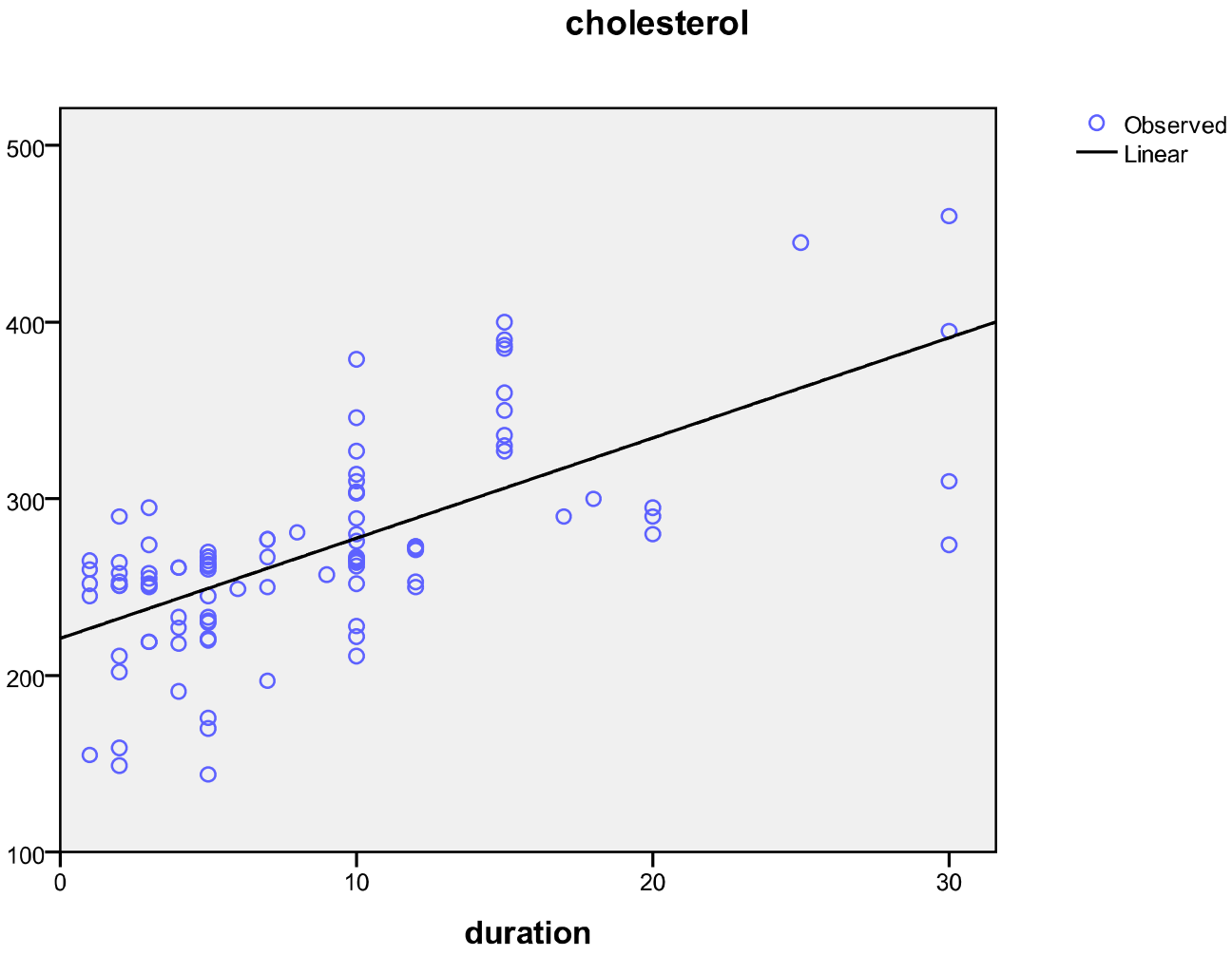


Figure 1. Bar diagram showing mean (± SD) serum total cholesterol levels in group A (control), group B and group C (experimental). Vertical lines indicate standard deviation (SD).



Statistical analysis was done by Pearson's Correlation Coefficient 'r' test.
(r value=.664; p value = .001)

Fig 2. Correlation of serum total cholesterol levels with duration of tobacco chewing & smoking in group B and group C (experimental).

Serum Triglyceride

Statistical analysis of the results of serum triglyceride levels shown in Table IlI. Analysis between the groups done by One – way ANOVA (post Hoc Test):
The mean ± SD serum triglyceride levels were 118.82 ± 27.743 mg/dl in group A, 174.00 ± 27.628 mg/dl in group B and 153.92 ± 38.223 mg/dl in group C. There was significant difference (p<.001) between group A and group B, group A and group C. There was also significant difference (p<.01) between group B and group C.

Table IlI: Serum total triglyceride levels in group A (control), group B and group C (experimental)

Groups	Mean ± SD mg/dl Range (L- H) mg/dl	'p' value
A / B (n=50) / (n=50)	118.82 ± 27.743 / 174.00± 27.628 (74 - 180) / (85 - 732)	0.001***
A / C (n=50) / (n=50)	118.82 ± 27.743/ 153.92 ± 38.223 (74 - 180) / (74 - 250)	0.001***
B / C (n=50) / (n=50)	174.00± 27.628 / 153.92 ± 38.223 (85 - 732) / (74 - 250)	0.006**

A= Apparently healthy subjects of non-tobacco chewer non-smoker (Control).
B= Apparently healthy subjects of tobacco chewer non-smoker (Experimental).
C= Apparently healthy subjects of tobacco chewer smoker (Experimental).
n= Number of subjects.
SD= Standard deviation.
***= p<0.001
**=p<0.01
L= Lowest value.
H= Highest value
#= Normal range of serum triglyceride level is < 150 mg/dl.[22]

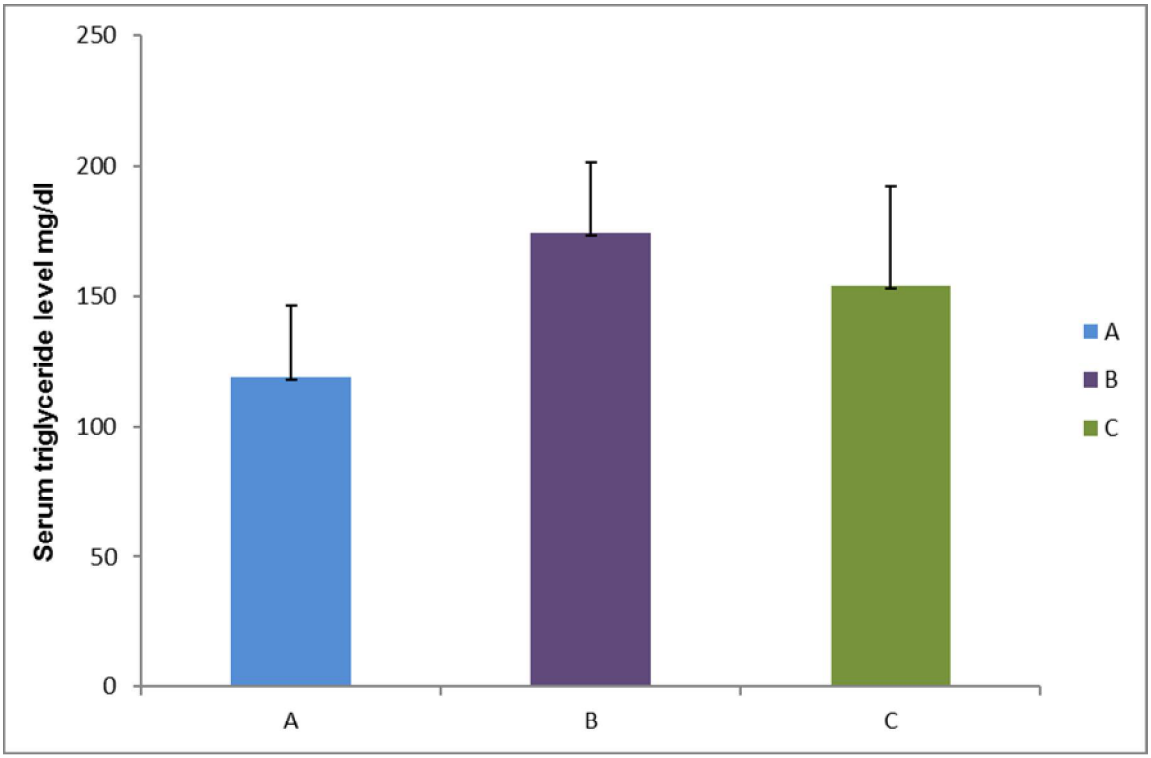


Figure 3. Bar diagram showing mean (± SD) triglyceride levels in group A (control), group B and group C (experimental). Vertical lines indicate standard deviation(SD).

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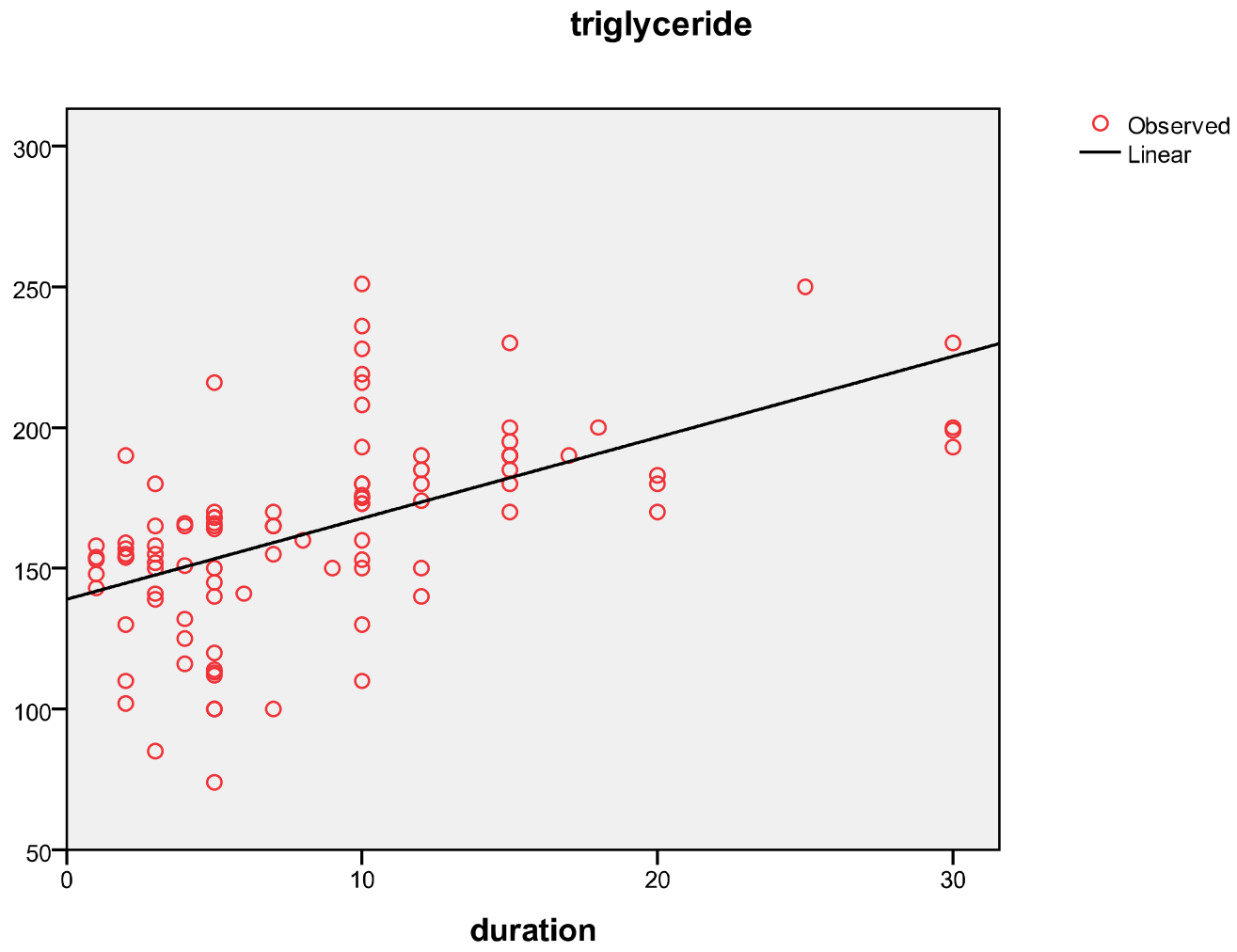


Statistical analysis was done by Pearson's Correlation Coefficient 'r' test. (r value =.561, p value =.001)

Fig 4. Correlation of serum triglyceride levels with duration of tobacco chewing & smoking in group B and group C (experimental).

Serum Low Density Lipoprotein Cholesterol

The mean ± SD serum low density lipoprotein cholesterol levels were 120.22 ± 30.756 mg/dl in group A, 199.02 ± 33.357 mg/dl in group B and 210.36 ±72.327 mg/dl in group C. The mean ± SD serum low density lipoprotein cholesterol levels were 120.22 ± 30.756 mg/dl in group A, 199.02 ± 33.357 mg/dl in group B and 210.36 ±72.327 mg/dl in group C. There was significant difference (p<.001) between group A and group B, group A and group C. But there was no significant difference (p>.05) between group B and group C.

Statistical analysis of the results of serum LDL-C levels shown in table III. Analysis between the groups done by one – way ANOVA (post Hoc Test):

Table IV: Serum LDL-C levels in group A (control), group B and group C (experimental)

Groups	Mean ± SD mg/dl Range (L- H) mg/dl	'p'value
A / B (n=50) / (n=50)	120.22 ± 30.756/ 199.02 ± 33.357 (59 - 210)/ (130 - 325)	0.001***
A / C (n=50) / (n=50)	120.22 ± 30.756/ 210.36 ± 72.327 (59 - 210) / (87 - 395)	0.001***
B / C (n=50) / (n=50)	199.02 ± 33.357 / 210.36 ± 72.327 (130 - 325) / (87- 395)	0.585^{NS}

A= Apparently healthy subjects of non-tobacco chewer non-smoker (Control).
B= Apparently healthy subjects of tobacco chewer non-smoker (Experimental).
C= Apparently healthy subjects of tobacco chewer smoker (Experimental).
n= Number of subjects.
SD= Standard deviation.
***= $p<0.001$
NS= $p>0.05$
L= Lowest value.
H= Highest value
#= Normal range of serum low density lipoprotein cholesterol level is <180 mg/dl.[23]

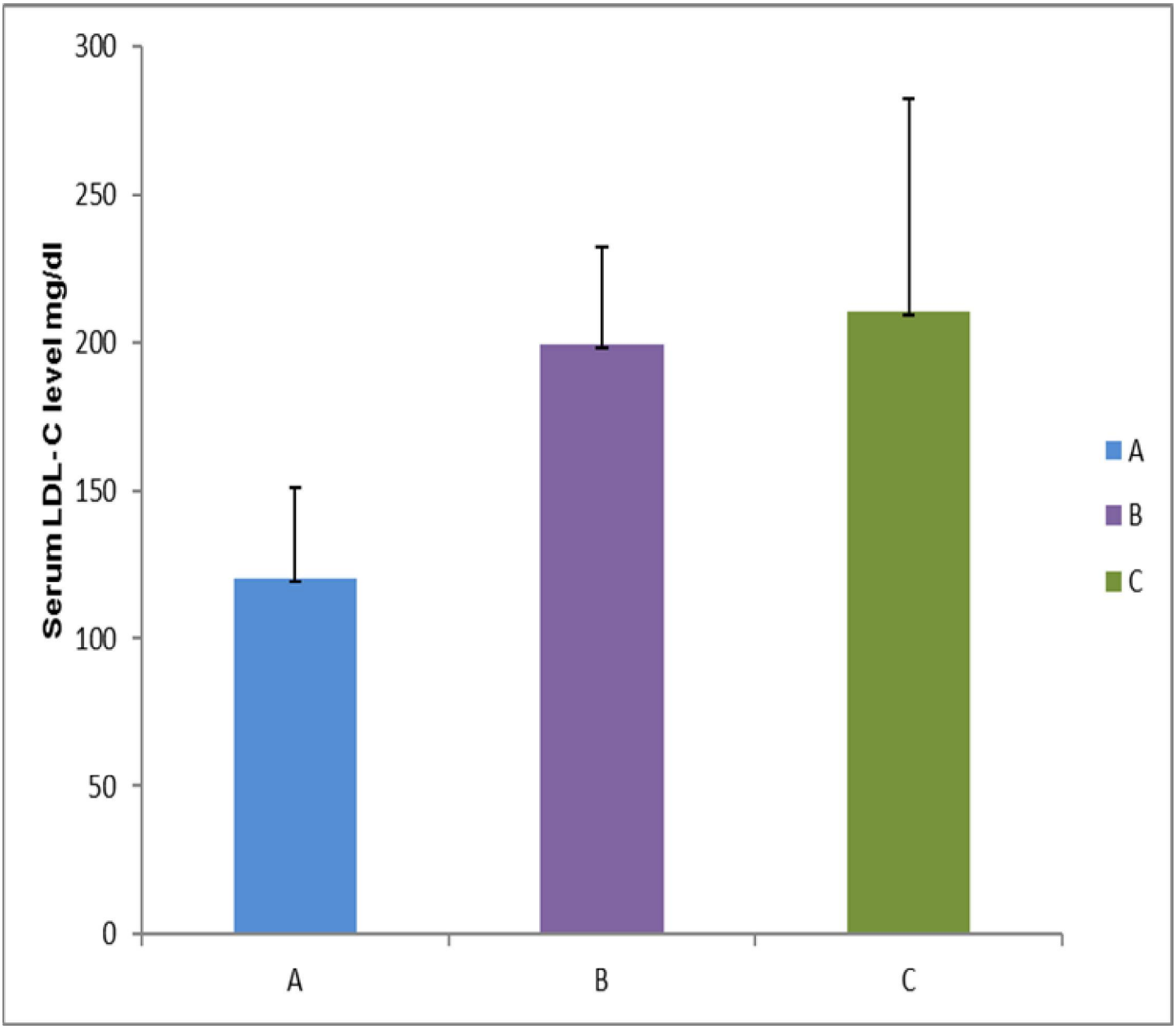
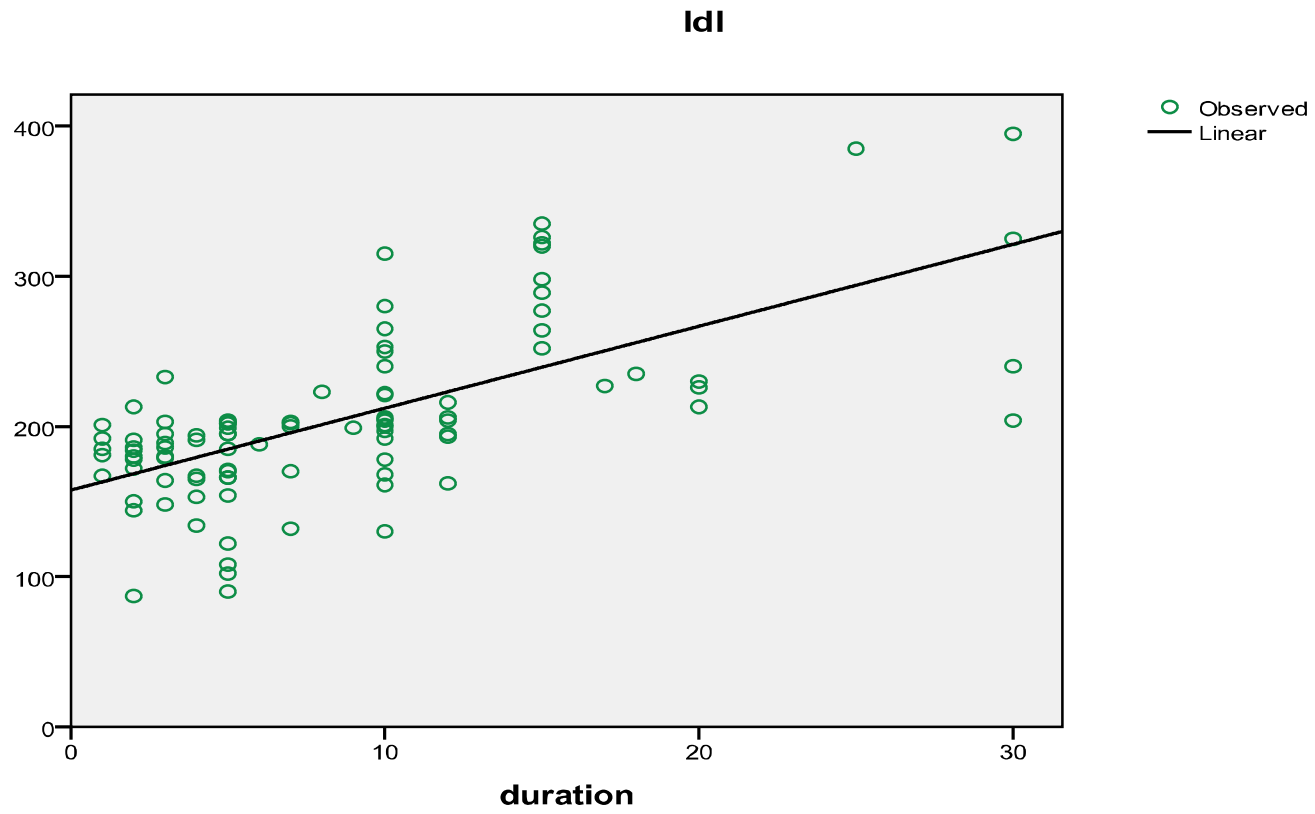


Figure 5. Bar diagram showing serum mean (± SD) LDL-C levels in group A (control), group B and group C (experimental). Vertical lines indicate standard deviation(SD).



Statistical analysis was done by Pearson's Correlation Coefficient 'r' test. (r value = .654, p value = .001)

Fig 6. Correlation of serum LDL-C levels with duration of tobacco chewing smoking in group B and group C (experimental).

Serum High Density Lipoprotein Cholesterol

The mean ± SD serum high density lipoprotein cholesterol levels were 48.72 ± 8.172 mg/dl in group A, 34.49 ± 6.548 mg/dl in group B and 31.36 ± 5.975 mg/dl in group C.

There was significant difference ($p<.001$) between group A and group B, group A and group C. Again, there was no significant difference ($p>.05$) between group B and group C.

Statistical analysis of the results of serum HDL-C levels shown in table V.

Analysis between the groups done by one – way ANOVA (post Hoc Test):

Table V: Serum HDL levels in group A (control), group B and group C (experimental)

Groups	Mean ± SD mg/dl Range (L- H) mg/dl	'p' value
A / B (n=50) / (n=50)	48.72 ± 8.172 / 34.49 ± 6.548 (24 - 66) / (22 - 49)	0.001***
A / C (n=50) / (n=50)	48.72 ± 8.172 /31.36 ± 5.975 (24 - 66) / (20 - 45)	0.001***
B / C (n=50) / (n=50)	34.49 ± 6.548 / 31.36 ± 5.975 (18 - 44) / (20 - 45)	0.078^{NS}

A= Apparently healthy subjects of non-tobacco chewer non-smoker (Control).
B= Apparently healthy subjects of tobacco chewer non-smoker (Experimental).
C= Apparently healthy subjects of tobacco chewer smoker (Experimental).
n= Number of subjects.
SD= Standard deviation.
***= $p<0.001$
NS= $p>0.05$
L= Lowest value.
H= Highest value
#= Normal range of serum high density lipoprotein cholesterol level is >45 mg/dl.[23]

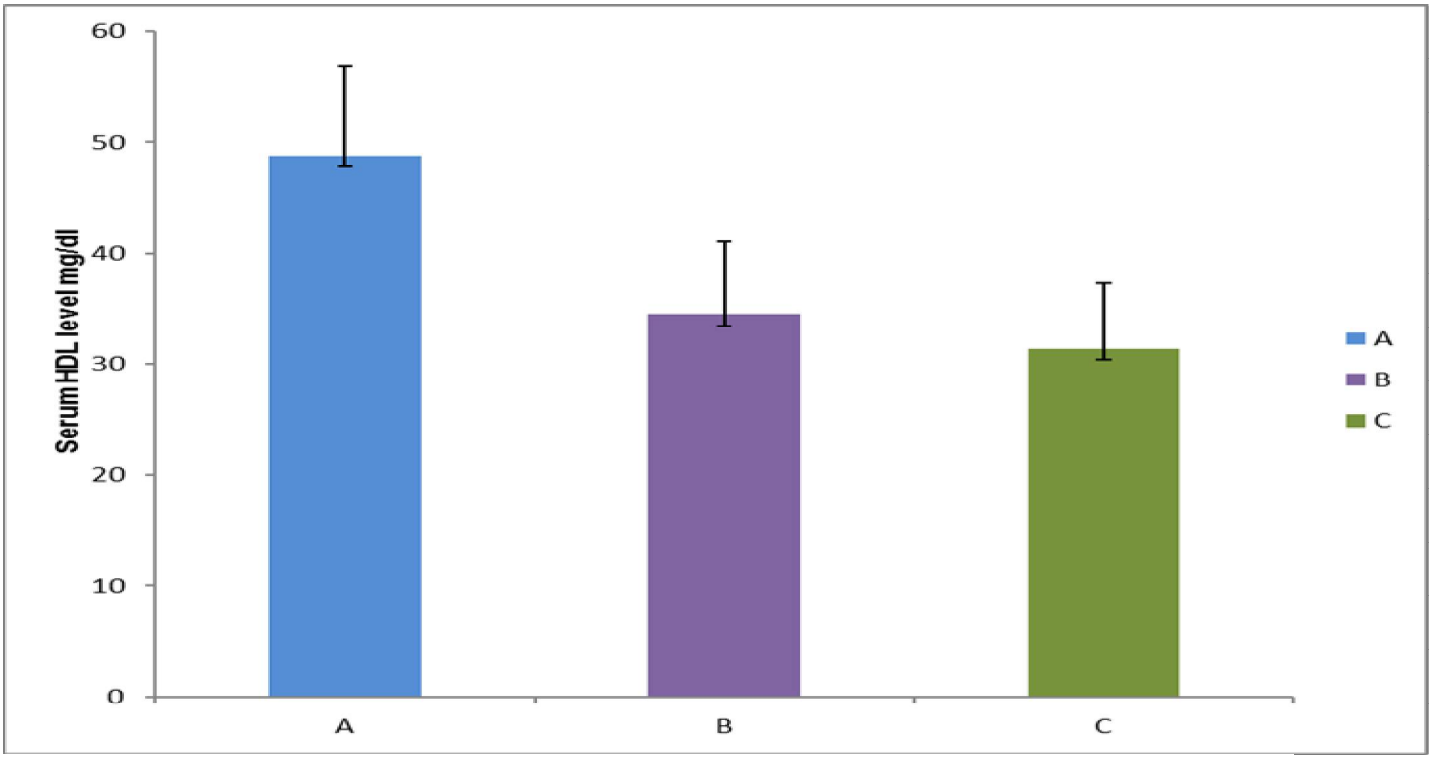
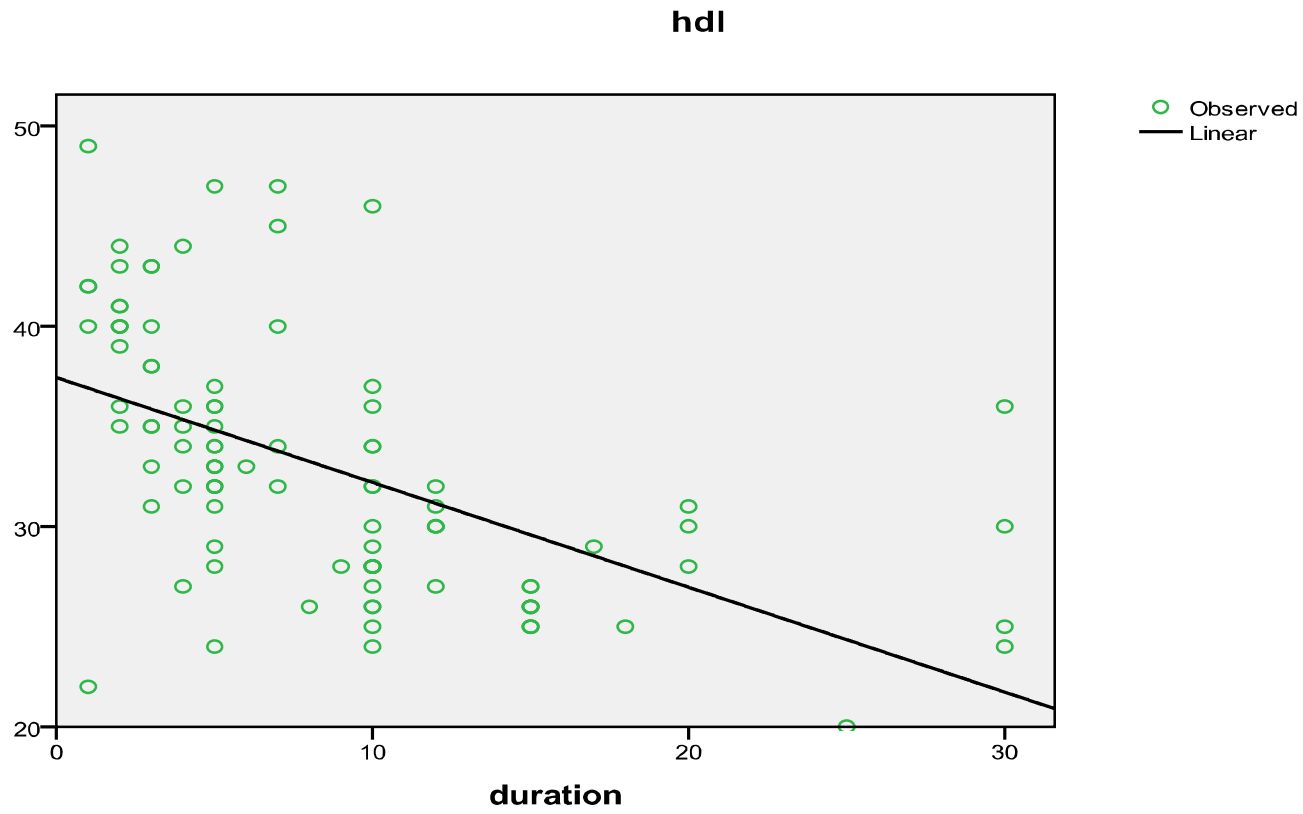


Figure 7. Bar diagram showing mean (± SD) serum HDL- C levels in group A (control), group B and group C (experimental). Vertical lines indicate standard deviation(SD).



Statistical analysis was done by Pearson's Correlation Coefficient 'r' test. (r value = .552 & p value = .001)

Fig 8. Correlation of serum HDL-C levels with duration of tobacco chewing & smoking in group B and group C (experimental).

Discussion

In this cross-sectional study, serum total cholesterol, serum triglyceride and serum low density lipoprotein cholesterol levels are significantly higher ($p<0.001$) and serum high density lipoprotein cholesterol level is significantly lower ($p< 0.001$) in tobacco chewer non-smoker and tobacco chewer smoker subjects than those of healthy control subjects.

In this study, the positive correlation between high level of serum total cholesterol, triglyceride, LDL-C and negative correlation between low level of HDL-C with duration of tobacco chewing and smoking in tobacco users may be due to consumption of tobacco for a prolong period of time which induced sustained rise of blood nicotine level as observed in other different researches. In addition, in Pearson's Correlation Coefficient 'r' test shows, serum total cholesterol, triglyceride, LDL-C levels are positively correlated but HDL-C level is negatively correlated with duration of tobacco chewing and smoking in tobacco users. Both of these are statistically significant in tobacco chewer non-smoker & tobacco chewer smoker subjects.

In this study, the mean serum total cholesterol level was significantly higher ($p<0.001$) in tobacco chewer smoker subjects than those of control subjects. This finding is in agreement with those reported by Gupta BK et al,[7] Kamble PH et al,[14] Shrivastava RK, Jha RK and Kumar R,[15] Mukherjee R and Chatterjee A,[16] Srinivasa RC and Emmanuel SY,[17] Guven A and Tolun F[18] and Yadav BK et al.[19] This finding of the present study reflects affecting of fat metabolism by tobacco consumption and smoking due to continued nicotine stimulated catecholamine secretion may have raised blood cholesterol level.

Siddique S et al,[9] Jaganmohan P and Sharma P,[11] Sharma SK and More UK[12] and Ahmed QR et al[13] observed that serum total cholesterol level was significantly higher in tobacco chewer non-smoker subjects which might be due to the sympathomimetic effect of nicotine and other minor alkaloids include nornicotine, anatabine, anabasine lead to release of adrenaline causes lipolysis in adipose tissues. This leads to increase in serum concentration of free fatty acids and cholesterol level.

In this study, the mean serum triglyceride level was significantly higher ($p<0.001$) in tobacco chewer non-smoker subjects & in tobacco chewer smoker subjects than those of control subjects. This finding is in agreement with those reported by Siddiqui S et al,[9] Dass BP, Jaganmohan P and sravanakumar P,[10] Sharma SK and More UK,[12] and Ahmed QR et al[13] which might be due to the sympathomimetic effect of nicotine and other minor alkaloids include nornicotine, anatabine, anabasine lead to release of adrenaline causes lipolysis in adipose tissues. This leads to increase in serum concentration of free fatty acids and triglycerides and may be due to tobacco induced stimulation on metabolism of free fatty acids in peripheral tissue. Also this may be due lipolysis caused by nicotine that increases free fatty acid concentration.

In this study, the mean serum low density lipoprotein cholesterol level was significantly higher ($p<0.001$) in tobacco chewer non-smoker subjects & in tobacco chewer smoker subjects than those of control subjects but the mean serum high density lipoprotein cholesterol level was significantly lower ($p<0.001$) in tobacco chewer non-smoker subjects and in tobacco chewer smoker subjects than those of control subjects. This finding is in agreement with those reported by Gupta BK et al,[7] Siddiqui S et al,[9] Dass BP, Jaganmohan P and sravanakumar P,[10] Sharma SK and More UK,[12] Ahmed QR et al,[13] Kamble PH et al,[14] Shrivastava RK, Jha RK and Kumar R,[15] Srinivasa RC and Emmanuel SY,[17] Guven A & Tolun F[18] and Yadav BK et al.[19] They found the correlation between consumption of tobacco and alteration of lipid profile which might be due to tobacco induced stimulation on metabolism of free fatty acids as well as increased cholesterol, LDL-C with decreased HDL-C. It may be due to the sympathomimetic effect of nicotine to release adrenaline that causes lipolysis in adipose tissues. This leads to increased concentration of free fatty acids and low density lipoprotein cholesterol.

From this above discussion it may be concluded that the higher serum total cholesterol, serum triglyceride, serum low density lipoprotein cholesterol in tobacco chewer non-smoker and tobacco chewer smoker subjects may be due to tobacco contains nicotine, nornicotine, anabasine and anatabine those stimulates release of adrenaline by the adrenal medulla. Adrenaline acts through the β_1 receptor leads to lipolysis in adipose tissues, leading to the increased serum concentration of free fatty acids, cholesterol, triglyceride and LDL-C.

Conclusion

This study may conclude that blood pressures are increased with tobacco use and related to duration of tobacco chewing & smoking in tobacco users. Results of this study may conclude that lipid profile is altered with tobacco use as evidenced by the mean serum total cholesterol, serum triglyceride and serum LDL-C levels were significantly higher ($p<0.001$) and HDL-C levels were significantly lower ($p<0.001$) in tobacco chewer non-smoker & tobacco chewer smoker subjects as compared with the healthy control subjects.

Acknowledgement

Authors of this study are thankful to the peoples for permission to collect samples. The authors also acknowledges partial financial support from research grant of DGHS of Bangladesh.

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