Characterization of lipid parameters in diabetic and non-diabetic atherosclerotic patients

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Abstract

Background & Objective The relationship between lipid profile perturbation and diabetes associated complications has long been an area of interest. Dyslipidemia is a potent predictor of cardiovascular morbidity and mortality in diabetic patients. The aim of present study was to investigate relationship between aging and lipid profiles in diabetic and non-diabetic atherosclerotic patients.

Methods Five hundred and seventy six individuals (45–75 year age) participated in this study. Among these, 192 were having history of diabetes mellitus and atherosclerosis. Individuals are categorized on the base of health (normal, non-diabetic atherosclerosis, diabetic atherosclerosis) and age (45–55 years, 56–65 years, and 66–75 years). All the participants were subjected to the procedures like a detailed history, biochemical analysis for fasting blood sugar, hemoglobin A1c, total cholesterol (TC), triglycerides (TG), low-density lipoprotein-(LDL), very low-density lipoprotein (VLDL) and high-density lipoprotein (HDL). All these parameters were compared between diabetic and non-diabetic atherosclerotic patients of all three age groups. TC/HDL and LDL/HDL were also calculated.

Results Diabetic atherosclerotic individuals (both males and females) had high level of TC, TG, LDL, VLDL and low level of HDL in comparison to non-diabetic atherosclerotic and normal control individuals. Among all three age groups, lipoprotein abnormality was observed to be more frequent in females than males. There was a significant increase in TC/HDL and LDL/HDL ratio in diabetic atherosclerotic subjects compared to age and sex matched non-diabetic atherosclerotic and normal control.

Conclusions Degree of dyslipidemia increases with increase in age in both genders. Female are more prone to diabetic dyslipidemia and hence have more risk of developing atherosclerosis with increasing age.

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Keywords: Aging; Atherosclerosis; Diabetes; Dyslipidemia

1 Introduction

Diabetes is associated with the development of many cardiovascular diseases (CVD). Diabetes impairs the utilization of lipids and lipoproteins which cause diabetes induced atherogenic dyslipidemia that, is one of the most important risk factor for the development of atherosclerosis in diabetic individuals. Atherosclerosis is one of the major causes in the development of CVD. Certain modifiable and non-modifiable risk factors contribute in the progression of atherosclerosis. Non-modifiable risk factors include age, gender and genetics, whereas modifiable risk factors include obesity, smoking, hypertension, diabetes and dyslipidemia. Diabetic dyslipidemia is characterized by increased serum low-density lipoprotein (LDL), triglycerides (TG) and decreased high-density lipoprotein (HDL). Diabetic individuals are more prone to dyslipidemia as compared to normal individuals, therefore, the chance of mortality and morbidity is high in diabetic individuals.

Geographic location, social and economic status of population affects the prevalence of dyslipidemia. Prevalence of dyslipidemia with respect to different risk factors is well studied. Age is an important risk factor in atherosclerosis. Cholesterol and lipoproteins levels increase with age in both genders, but the more pronounced increase has been reported in females than in males. The purpose of the study was to assess the effect of age and gender on dyslipidemia in diabetic versus non-diabetic atherosclerotic patients.

2 Methods

2.1 Subjects and study design

This case-control study included 576 subjects, out of...
which 192 subjects (96 males and 96 females) with normal blood glucose level were selected as control group (normal, N), 192 subjects (96 males and 96 females) with normal blood glucose level and atherosclerosis (non-diabetic and atherosclerotic, NA), 192 subjects (96 males and 96 females) with diabetes and atherosclerosis (diabetic and atherosclerotic, DA), visiting the outpatient clinic at Department of Cardiology at Fatima Memorial Hospital Shadman, Lahore, Pakistan. Diabetes was diagnosed by analyzing the level of glycosylated hemoglobin (HbA1c > 6.5%).[18] Diagnosed cases of diabetic and non-diabetic atherosclerotic patients were included after obtaining a written consent from their care takers to take part in the study. Questionnaires were duly filled in with bio-data of the patients, clinical presentation of the illness, complete blood count record, along with available additional investigative information. Blood samples were collected from Fatima Memorial Hospital Shadman, Lahore. This study was approved by the local Ethics Committee at the University of Lahore, Pakistan.

2.2 Inclusion and exclusion criteria

Inclusion criteria were male and female, aged 45–75 years, with the history of diabetes and atherosclerosis. Exclusion criteria were subjects with the history of smoking, alcoholism, renal diseases, thyroid disorders, pregnancy or any disease.

2.3 Collection of blood and isolation of serum

Blood samples were drawn from cubital vein in preprandial state from all individuals in morning into disposable syringes and then shifted in properly labeled EDTA vacutainer tubes. Blood samples were centrifuged at 2000 r/min for 10 min at 4°C to isolate serum. Sera were aliquoted and stored at −20°C for future analysis.

2.4 Estimation of biochemical parameters

All the biochemical analysis was performed at Center for Research in Molecular Medicine (CRIMM), the University of Lahore. Serum levels of TC, TG, and HDL were measured spectrophotometrically using commercial assay kits (Randox laboratories Ltd., United Kingdom). LDL and VLDL were calculated by using Friedewald formula.[19] Also, TC/HDL and LDL/HDL ratios were calculated in all groups.

2.5 Statistical analysis

The statistical analysis of data was performed using GraphPad Prism-5 for Windows (GraphPad Software, San Diego, CA, USA). The data was analyzed using One Way Analysis of variance (ANOVA) followed by Bonferroni testing. All data are expressed as a mean ± SE of the mean (SEM). *P < 0.05 was considered significant.

3 Results

3.1 Subjects demographic characteristics

The study subjects were divided: on the basis of health condition into normal, N group (192 subjects); non-diabetic and atherosclerotic, NA group (192 subjects); and diabetic and atherosclerotic, DA group (192 subjects). And according to age as 45–55 year (96 subjects); 56–65 year (96 subjects); and 66–75 year (96 subjects). Results of basic demographic characteristics studied are illustrated in Table 1.

3.2 Effect of gender on dyslipidemia

The correlation of age and diabetes which are two important risk factors in the contribution of atherosclerosis in males and females was evaluated (Figure 1). Figure 1A represents the 45–55 year age group data. Both males and females of DA group showed significant increase in TC (255 ± 1.23 mg/dL, 262 ± 1.24 mg/dL), TG (260 ± 1.10 mmol/L, 267 ± 1.28 mmol/L), HDL (4.2 ± 0.01, 4.5 ± 0.03), and LDL (5.6 ± 0.43, 6.1 ± 0.15), respectively. Gender differences were significant in TC, TG, and LDL levels in DA group.

Table 1. General demographic characteristics of the study population.

| Subjects Characteristics | Gender | Age groups |
|--------------------------|--------|------------|
|                          |        | 45–55 yrs  |
|                          |        | 56–65 yrs  |
|                          |        | 66–75 yrs  |
| N group (n = 192) M     |        | 50.06 ± 0.56 | 60.28 ± 0.51 | 70.21 ± 0.52 |
| (n = 96) F              |        | 49.93 ± 0.51 | 53.87 ± 1.04 | 70.34 ± 0.54 |
| NA group (n = 192) M    | Age    | 50.15 ± 0.54 | 60.06 ± 0.52 | 70.21 ± 0.52 |
| (n = 96) F              |        | 53.87 ± 1.04 | 60.09 ± 0.49 | 72.21 ± 0.52 |
| DA group (n = 192) M    |        | 50.15 ± 0.53 | 60.06 ± 0.52 | 70.21 ± 0.52 |
| (n = 96) F              |        | 50.00 ± 0.55 | 60.09 ± 0.50 | 71.21 ± 0.52 |
| N group (n = 192) M     |        | 4.2 ± 0.29  | 4.3 ± 0.07   | 4.4 ± 0.08   |
| (n = 96) F              |        | 3.8 ± 0.06  | 4.5 ± 0.07   | 5.1 ± 0.08   |
| NA group (n = 192) M    | FBG, mmol/L | 3.9 ± 0.03 | 4.0 ± 0.04  | 5.3 ± 0.18  |
| (n = 96) F              |        | 4.2 ± 0.01  | 4.5 ± 0.03   | 5.4 ± 0.16   |
| DA group (n = 192) M    |        | 7.1 ± 0.03  | 8.1 ± 0.02   | 8.8 ± 0.48   |
| (n = 96) F              |        | 7.3 ± 0.02  | 7.9 ± 0.03   | 8.9 ± 0.52   |
| N group (n = 192) M     |        | 3.9 ± 0.56  | 4.0 ± 0.15   | 4.1 ± 0.54   |
| (n = 96) F              |        | 3.6 ± 0.51  | 3.7 ± 0.15   | 4.0 ± 0.15   |
| NA group (n = 192) M    | HbA1c, % | 5.6 ± 0.43  | 6.1 ± 0.15   | 6.4 ± 0.15   |
| (n = 96) F              |        | 5.7 ± 0.42  | 6.6 ± 0.15   | 6.6 ± 0.52   |
| DA group (n = 192) M    |        | 7.9 ± 0.4   | 8.7 ± 0.15   | 9.9 ± 0.51   |
| (n = 96) F              |        | 7.8 ± 0.01  | 8.8 ± 0.02   | 10.1 ± 0.55  |

All values are presented as mean ± SE. DA: diabetic and atherosclerotic; F: female; FBG: fasting blood glucose; HbA1c: glycated hemoglobin; M: male; N: normal; NA: non-diabetic and atherosclerosis.
Figure 1. Correlation of dyslipidemia between males and females. (A): 45–55 year age group; (B): 56–65 year age group; (C): 65–75 year age group. Age wise comparison among different groups for all parameters. DAM vs. NAM, *P < 0.001; DAM vs. NM, #P < 0.001; DAF vs. NAF, ζP < 0.001; DAF vs. NF, ηP < 0.001; NM vs. NF, *P < 0.001; NAM vs. NAF, φP < 0.001; DAM vs. DAF, λP < 0.001. NM + NF: normal group males and females; NAM + NAF: non-diabetic and atherosclerosis group males and females; DAM + DAF: diabetic and atherosclerosis group males and females; HDL: high density lipoproteins; LDL: low density lipoproteins; VLDL: very low density lipoproteins.
mg/dL, 290 ± 1.18 mg/dL), LDL (210 ± 1.17 mg/dL, 250 ± 1.21 mg/dL), and VLDL (70 ± 0.80 mg/dL, 83 ± 0.82 mg/dL) as compared to that of NA group for TC (222 ± 3.57 mg/dL, 230 ± 1.15 mg/dL), TG (200 ± 1.21 mg/dL, 220 ± 1.20 mg/dL), LDL (190 ± 1.10 mg/dL, 220 ± 1.20 mg/dL), and VLDL (65 ± 0.92 mg/dL, 70 ± 0.96 mg/dL) and N group for TC (163 ± 1.04 mg/dL, 171 ± 1.15 mg/dL), TG (121 ± 1.10 mg/dL, 126 ± 1.19 mg/dL), LDL (123 ± 1.03 mg/dL, 140 ± 1.06 mg/dL), and VLDL (35 ± 0.59 mg/dL, 40 ± 0.78 mg/dL). Whereas DA group males and females have significant lower level of HDL in comparison to males as shown in Figure 1A. Male and female subjects of DA group showed significantly higher level of TC, TG, LDL, and VLDL and significantly lower level of HDL as compared to NA group for TC (246 ± 1.13, 250 ± 1.21), TG (250 ± 1.23, 260 ± 1.19), LDL (230 ± 1.16, 281 ± 1.15), and VLDL (85 ± 0.69, 90 ± 0.78) and N group for TC (195 ± 1.08, 200 ± 1.20), TG (195 ± 1.08, 149 ± 1.05), LDL (133 ± 1.07, 157 ± 1.13), and VLDL (43 ± 0.81 mg/dL, 50 ± 0.66 mg/dL). Whereas, DA group males and females showed significantly lower level of HDL (24 ± 0.46 mg/dL, 22 ± 0.30 mg/dL) in comparison to NA group (31 ± 0.67 mg/dL, 29 ± 0.44 mg/dL), and N group (37 ± 0.71 mg/dL, 40 ± 0.53 mg/dL). Among DA group subjects, females have significantly higher level of TC, TG, LDL, and VLDL and significantly lower level of HDL as compared to males as shown in Figure 1C.

3.3 Effect of age on dyslipidemia

Among the three age groups, levels of all the lipid constituents were high in 65–75 year age group in both males and females. Furthermore, within N, NA, and DA groups, females depicted high values of all lipid parameters in comparison to males in all age groups. The proportion of females with abnormal TC/HDL and LDL/HDL ratio was higher than that of males in NA and DA groups. Statistical difference was noted among all groups with more significance in 66–75 year age group as shown in Table 2.

4 Discussion

Diabetic individuals are at an increased risk of CVD compared to nondiabetic individuals, therefore diabetic subject have high mortality rate. Diabetic dyslipidemia is one of the major risk factors which contributes to atherosclerosis, one of the main forms of CVD. The study analyzes the pattern of modifiable risk factor i.e., diabetes and non-modifiable risk factors like age and gender in atherosclerotic patients. Our results showed that the FBG and HbA1c levels were higher particularly in DA subjects as compared to NA. This conforms the study of Ghazanfari, et al. who presented that FBG and HbA1c are used as diagnostic biomarker to separate diabetic from non-diabetic subjects. The study showed that age and gender have no effect on fasting glucose level and HbA1c. However it was found that the HbA1c value increases in DA as well as in NA male and female groups. Nicholas, et al. shows that increase in HbA1c level enhances the risk of diabetes induced mortality risk in subjects having CVD.

Diabetic dyslipidemia is also known as atherogenic dyslipidemia due to presence of high level of cholesterol, triglycerides and low level of HDL. To further explore this possibility, we evaluated the relationship of diabetes (another condition associated with development of atherosclerosis), with lipid profile parameters across three age groups (45–55, 56–65 and 66–75 year of age) in all groups (N, NA, DA) male and female subjects. Our results showed signifi-
Table 2. Effect of age on lipid values in all groups.

| Subjects | Parameters | Gender | Age groups | P value |
|----------|------------|--------|------------|---------|
|          |            |        | 45–55 Year | 56–65 Year | 66–75 Year |
| N group  | TC (mg/dL) | M (n = 96) | 163 ± 1.04 | 175 ± 1.21 | 195 ± 1.08 | P < 0.001** |
| (n = 192)|            | F (n = 96) | 171 ± 1.15 | 183 ± 1.10 | 200 ± 1.20 | P < 0.001** |
| NA group | TC (mg/dL) | M (n = 96) | 222 ± 3.57 | 239 ± 1.25 | 246 ± 1.13 | P < 0.001** |
| (n = 192)|            | F (n = 96) | 230 ± 1.15 | 242 ± 1.22 | 250 ± 1.21 | P < 0.001** |
| DA group | TC (mg/dL) | M (n = 96) | 255 ± 1.23 | 266 ± 1.13 | 271 ± 1.14 | P > 0.05*, P < 0.001* |
| (n = 192)|            | F (n = 96) | 262 ± 1.24 | 278 ± 1.20 | 290 ± 1.22 | P < 0.001** |
| N group  | TG (mg/dL) | M (n = 96) | 121 ± 1.10 | 175 ± 1.21 | 195 ± 1.08 | P > 0.05* |
| (n = 192)|            | F (n = 96) | 126 ± 1.19 | 140 ± 1.07 | 149 ± 1.05 | P < 0.001** |
| NA group | TG (mg/dL) | M (n = 96) | 200 ± 1.21 | 220 ± 1.25 | 250 ± 1.23 | P < 0.001** |
| (n = 192)|            | F (n = 96) | 210 ± 1.09 | 235 ± 1.16 | 260 ± 1.19 | P < 0.001** |
| DA group | TG (mg/dL) | M (n = 96) | 260 ± 1.10 | 278 ± 1.17 | 290 ± 1.03 | P > 0.05* |
| (n = 192)|            | F (n = 96) | 290 ± 1.18 | 315 ± 1.12 | 330 ± 1.07 | P > 0.05* |
| N group  | LDL (mg/dL)| M (n = 96) | 123 ± 1.03 | 130 ± 1.04 | 133 ± 1.07 | P > 0.05* |
| (n = 192)|            | F (n = 96) | 140 ± 1.06 | 151 ± 1.01 | 157 ± 1.13 | P > 0.05* |
| NA group | LDL (mg/dL)| M (n = 96) | 190 ± 1.10 | 210 ± 1.19 | 230 ± 1.16 | P > 0.05* |
| (n = 192)|            | F (n = 96) | 220 ± 1.20 | 245 ± 1.22 | 281 ± 1.15 | P < 0.001** |
| DA group | LDL (mg/dL)| M (n = 96) | 210 ± 1.17 | 240 ± 1.14 | 260 ± 1.09 | P < 0.001** |
| (n = 192)|            | F (n = 96) | 250 ± 1.21 | 263 ± 1.41 | 310 ± 1.17 | P < 0.001** |
| N group  | VLDL (mg/dL)| M (n = 96) | 35 ± 0.59  | 39 ± 0.53  | 43 ± 0.81  | P < 0.001** |
| (n = 192)|            | F (n = 96) | 40 ± 0.78  | 46 ± 0.78  | 50 ± 0.66  | P < 0.001** |
| NA group | VLDL (mg/dL)| M (n = 96) | 65 ± 0.92  | 80 ± 0.93  | 85 ± 0.69  | P > 0.05* |
| (n = 192)|            | F (n = 96) | 70 ± 0.96  | 85 ± 0.69  | 90 ± 0.78  | P < 0.001** |
| DA group | VLDL (mg/dL)| M (n = 96) | 70 ± 0.80  | 90 ± 0.79  | 100 ± 0.74 | P < 0.001** |
| (n = 192)|            | F (n = 96) | 83 ± 0.82  | 100 ± 0.78 | 120 ± 0.90 | P < 0.001** |
| N group  | HDL (mg/dL)| M (n = 96) | 42 ± 0.36  | 40 ± 0.41  | 37 ± 0.71  | P < 0.001** |
| (n = 192)|            | F (n = 96) | 29 ± 0.40  | 43 ± 0.48  | 40 ± 0.53  | P < 0.001** |
| NA group | HDL (mg/dL)| M (n = 96) | 34 ± 0.44  | 32 ± 0.70  | 31 ± 0.67  | P > 0.05* |
| (n = 192)|            | F (n = 96) | 33 ± 0.47  | 30 ± 0.59  | 29 ± 0.44  | P < 0.001** |
| DA group | HDL (mg/dL)| M (n = 96) | 29 ± 0.40  | 26 ± 0.51  | 24 ± 0.46  | P < 0.001** |
| (n = 192)|            | F (n = 96) | 45 ± 0.41  | 25 ± 0.49  | 22 ± 0.30  | P < 0.001** |
| N group  | TC/HDL   | M (n = 96) | 3.89 ± 0.04 | 4.34 ± 0.04 | 5.19 ± 0.09 | P < 0.001** |
| (n = 192)|            | F (n = 96) | 3.79 ± 0.04 | 4.28 ± 0.05 | 4.96 ± 0.07 | P < 0.001** |
| NA group | TC/HDL   | M (n = 96) | 6.56 ± 0.12 | 7.59 ± 0.17 | 8.07 ± 0.18 | P > 0.05* |
| (n = 192)|            | F (n = 96) | 6.96 ± 0.10 | 8.00 ± 0.14 | 8.73 ± 0.15 | P < 0.001** |
| DA group | TC/HDL   | M (n = 96) | 8.68 ± 0.129 | 10.05 ± 0.20 | 10.97 ± 0.19 | P < 0.001** |
| (n = 192)|            | F (n = 96) | 7.18 ± 0.11 | 10.84 ± 0.21 | 12.87 ± 0.19 | P < 0.001** |
| N group  | LDL/HDL  | M (n = 96) | 2.94 ± 0.03 | 3.23 ± 0.04 | 3.56 ± 0.08 | P < 0.001** |
| (n = 192)|            | F (n = 96) | 3.12 ± 0.02 | 3.53 ± 0.04 | 3.93 ± 0.06 | P > 0.05* |
| NA group | LDL/HDL  | M (n = 96) | 5.64 ± 0.09 | 6.70 ± 0.15 | 7.54 ± 0.16 | P < 0.001** |
| (n = 192)|            | F (n = 96) | 6.65 ± 0.10 | 8.10 ± 0.16 | 9.80 ± 0.15 | P < 0.001** |
| DA group | LDL/HDL  | M (n = 96) | 7.18 ± 0.11 | 9.08 ± 0.19 | 10.54 ± 0.18 | P > 0.05* |
| (n = 192)|            | F (n = 96) | 8.75 ± 0.12 | 10.26 ± 0.20 | 13.76 ± 0.20 | P < 0.001** |

All values are mean ± SE. *P value for 65–75 years vs. 45–55 years; †P value for 65–75 years vs. 55–65 years. DA: diabetic and atherosclerosis; HDL: high density lipoproteins; N: normal; NA: non-diabetic and atherosclerosis; TC: total cholesterol; TG: triglycerides; LDL: low density lipoproteins; VLDL: very low density lipoproteins.
cant increase in TC, TG, LDL, and VLDL levels in both genders in DA as compared to NA and N groups. Whereas DA group males and females have significant lower level of HDL in comparison to NA group and N group. In DA group, females have significantly higher level of TC, TG, LDL, and VLDL and significantly lower level of HDL as compared to males. This finding correlates with the previous study showing that diabetic dyslipidemia is more atherogenic than that of normal dyslipidemia,\(^{[25]}\) and female are more prone to it.\(^{[26]}\) It was found that in each age group, lipid parameters showed particular abnormal pattern in DA and NA groups and gender wise it was more in females as compared to males. This data is in agreement with other studies which show abnormal lipid pattern in diabetic and normal subjects although the cut off values slightly differed.\(^{[27]}\)

As aging is one of the risk factor for development of atherosclerosis,\(^{[28]}\) the relationship of aging on lipid profile of N, NA, and DA patients was evaluated. It was found that increasing age in both genders enhances the chance of atherosclerosis. This study also reveals that the prevalence of dyslipidemia by age appeared to be higher in the 66–75 years group in both male and female subjects in all groups. A high prevalence of atherogenic dyslipidemia in DAF, similar to that observed in DAM was observed. In diabetes, atherogenic dyslipidemia surpass the normal atherogenic dyslipidemic condition.\(^{[25,29]}\) Studies depicted that TC/HDL and LDL/HDL ratios can be used as markers of diabetic dyslipidemia in diabetic patients.\(^{[30,31]}\) The high value of TC/HDL and LDL/HDL ratios was found between 65–75 age group thereby affirming that increasing age causes the chance of atherosclerosis in diabetic subjects as compared to normal subjects. This result was consistent with previous study depicting that these ratios are associated with risk of future CVD.\(^{[32–34]}\)

The significance of this study is to highlight the effect of dyslipidemia in both males and female because it is the important parameter of atherosclerosis. The comparison of lipid profile in non-diabetic and diabetic atherosclerotic patients would enable us to maintain the health of patients by reducing cardiovascular risk. By correlating the effect of age and gender on lipid profile in these patients, it is concluded that in old age, diabetic females are more prone to dyslipidemia than diabetic males of the same age. This contributes to high risk of atherosclerosis and later on CVD in diabetic females compared to diabetic males. Considering the high prevalence of CVD in old age, morbidity rate can be reduced by controlling diabetes and hence, diabetes induced lipid abnormalities in these patients through different approaches, for example by changing lifestyle like dietary habits, by regular exercise and medical examination.

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### References

1. Grundy SM, Benjamin IJ, Burke GL, et al. Diabetes and cardiovascular disease: a statement for healthcare professionals from the American Heart Association. *Circulation* 1999; 100: 1134–1146.
2. Bartels DW, Davidson MH, Gong WC. Type 2 diabetes and cardiovascular disease: reducing the risk. *J Manag Care Pharm* 2007; (2 Suppl A): S2–S15.
3. Krauss RM. Lipids and lipoproteins in patients with type 2 diabetes. *Diabetes Care* 2004; 27: 1496–1504.
4. Farmer JA. Diabetic dyslipidemia and atherosclerosis: evidence from clinical trials. *Curr Atheroscler Rep* 2007; 9: 162–168.
5. Frostegard J. Immunity, atherosclerosis and cardiovascular disease. *BMC Med* 2013; 11: 117.
6. Third Report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (adult treatment panel III) final report. National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (adult treatment panel III). *Circulation* 2002; 106: 3143–3421.
7. Oyama N, Gona P, Salton CJ, et al. Differential impact of age, sex, and hypertension on aortic atherosclerosis: the Framingham Heart Study. *Arterioscler Thromb Vasc Biol* 2008; 28: 155–159.
8. American Diabetes Association (ADA). Standards of medical care in diabetes. *Diabetes Care* 2007; 30: 4–41.
9. Pathak R, Pathak A. Study of life style habits on risk of type 2 diabetes. *Int J Appl Basic Med Res* 2012; 2: 92–96.
10. Mooradian AD. Dyslipidemia in type 2 diabetes mellitus. *Nat Clin Pract Endocrinol Metab* 2009; 5: 150–159.
11. Goff DC Jr, Bertoni AG, Kramer H, et al. Dyslipidemia prevalence, treatment, and control in the Multi-Ethnic Study of Atherosclerosis (MESA): gender, ethnicity, and coronary artery calcium. *Circulation* 2006; 113: 647–656.
12. Aekplakorn W, Taneepanichskul S, Kessomboon P, et al. Prevalence of dyslipidemia and management in the Thai population, national health examination survey IV, 2009. *J Lipids* 2014; 249584.
13. Cabrera M, Sanchez-Chaparro MA, Valdivielso P, et al. Prevalence of atherogenic dyslipidemia: association with risk factors and cardiovascular risk in Spanish working population. “ICARIA” study. *Atherosclerosis* 2014; 235: 562–569.
14. Plana N, Ibarretxe D, Cabre A, et al. Prevalence of atherogenic dyslipidaemia in primary care patients at moderate-very high risk of cardiovascular disease. Cardiovascular risk perception. Clin Investig Arterioscler 2014; Published Online First: Jun 12. DOI: 10.1016/j.arteri.2014.04.002.

15. Zhang L, Connelly JJ, Peppel K, et al. Aging-related atherosclerosis is exacerbated by arterial expression of tumor necrosis factor receptor-1: evidence from mouse models and human association studies. Hum Mol Genet 2010; 19: 2754–2766.

16. Stamler J, Dyer AR, Shekelle RB, et al. Relationship of baseline major risk factors to coronary and all-cause mortality, and to longevity: findings from long-term follow-up of Chicago cohorts. Cardiology 1993; 82: 191–222.

17. Roeters van Lennep JE, Westerveld HT, Erkelens DW, et al. Risk factors for coronary heart disease: implications of gender. Cardiovasc Res 2002; 53: 538–549.

18. Standards of medical care in diabetes-2010. American Diabetes Association. Diabetes Care 2010; 33 (Suppl 1): S11–S61.

19. Friedewald WT, Levy RI, Fredrickson DS. Estimation of concentration of low density lipoprotein cholesterol in plasma without use of the preparative ultracentrifuge. Clin Chem 1972; 18: 449–502.

20. Howard BV, Robbins DC, Sievers ML, et al. LDL cholesterol as a strong predictor of coronary heart disease in diabetic individuals with insulin resistance and low LDL: The Strong Heart Study. Arterioscler Thromb Vasc Biol 2000; 20: 830–835.

21. Ghazanfari Z, Haghdoot AA, Alizadeh SM, et al. A Comparison of HbA1c and Fasting Blood Sugar Tests in General Population. Int J Prev Med 2010; 1: 187–194.

22. Nicholas J, Charlton J, Dregan A, et al. Recent HbA1c values and mortality risk in type 2 diabetes. Population-based case-control study. PLoS One 2013; 8: e68008.

23. Vijayaraghavan K. Treatment of dyslipidemia in patients with type 2 diabetes. Lipids Health Dis 2010; 9: 144.

24. Assmann G, Cullen P, Schulte H. Non-LDL-related dyslipidaemia and coronary risk: a case-control study. Diab Vasc Dis Res 2010; 7: 204–212.

25. Khan HA, Sobki SH, Khan SA. Association between glycaemic control and serum lipids profile in type 2 diabetic patients: HbA1c predicts dyslipidaemia. Clin Exp Med 2007; 7: 24–29.

26. Williams K, Tehnernof A, Hunt KJ, et al. Diabetes, abdominal adiposity, and atherogenic dyslipoproteinemia in women compared with men. Diabetes 2008; 57: 3289–3296.

27. Ogbera AO, Fasanmade OA, Chinanye S, et al. Characterization of lipid parameters in diabetes mellitus—a Nigerian report. Int Arch Med 2009; 2: 19.

28. Wang JC, Bennett M. Aging and atherosclerosis: mechanisms, functional consequences, and potential therapeutics for cellular senescence. Circ Res 2012; 111: 245–259.

29. Tenenbaum A, Fisman EZ. Fibrates are an essential part of modern anti-dyslipidemic arsenal: spotlight on atherogenic dyslipidemia and residual risk reduction. Cardiovasc Diabetol 2012; 11: 125.

30. Gimeno-Orna JA, Faure-Nogueras E, Sancho-Serrano MA. Usefulness of total cholesterol/HDL-cholesterol ratio in the management of diabetic dyslipidaemia. Diabet Med 2005; 22: 26–31.

31. Pandeya A, Sharma M, Regmi P, et al. Pattern of dyslipidemia and evaluation of non-HDL cholesterol as a marker of risk factor for cardiovascular disease in type 2 diabetes mellitus. Nepal Med Coll J 2012; 14: 278–282.

32. Lemieux I, Lamarche B, Couillard C, et al. Total cholesterol/HDL cholesterol ratio vs LDL cholesterol/HDL cholesterol ratio as indices of ischemic heart disease risk in men: the Quebec Cardiovascular Study. Arch Intern Med 2001; 161: 2685–2692.

33. Ridker PM, Stampfer MJ, Rifai N. Novel risk factors for systemic atherosclerosis: a comparison of C-reactive protein, fibrinogen, homocysteine, lipoprotein(a), and standard cholesterol screening as predictors of peripheral arterial disease. JAMA 2001; 285: 2481–2485.

34. Arsenault BJ, Rana JS, Stroes ES, et al. Beyond low-density lipoprotein cholesterol: respective contributions of non-high-density lipoprotein cholesterol levels, triglycerides, and the total cholesterol/high-density lipoprotein cholesterol ratio to coronary heart disease risk in apparently healthy men and women. J Am Coll Cardiol 2009; 55: 35–41.