Controlling Lipids AIDS in the Prevention of Type 2 Diabetes, Hypertension, and Cardiovascular Diseases

Abstract

Background: Abnormal lipid profiles are a characteristic feature of persons with chronic conditions in which the diabetic populations are recognized as the dominant group, regardless of gender and ethnicity worldwide. This study was conducted to identify and evaluate the abnormalities of serum lipid profiles in both nondiabetic and diabetic persons. Methods: This study was a case–control investigation conducted between 2013 and 2015. The study enrolled 266 patients from the North Central and South West Regional Health Authorities of Trinidad. Of the 266 patients recruited, 126 were diabetic and 140 were nondiabetic. Results: Our study observed that dyslipidemia was present among the nondiabetic populations as the nondiabetics had 55 women and 20 men with high cholesterol, 22 women and 14 men with high triglyceride (TG), 30 women and 25 men with low high-density lipoprotein cholesterol (HDL-C), 42 women and 21 men with high low-density level-cholesterol (LDL-C), 13 women and 8 men with high very low-density lipoprotein (VLDL), and also 30 women and 11 men with body mass index (BMI) over 30 kg/m². We also observed that diabetic patients had significantly lower TGs (P = 0.019) and higher HDL-C (P = 0.001) and LDL (P = 0.003) when compared with the diabetic men. In addition, the nondiabetic females also had higher HDL-C (P = 0.045) when compared to their male counterparts. Both diabetic and nondiabetic women exhibited significantly higher BMI of P = 0.000. A negative correlation was obtained among TGs and HDL (r = −0.356, n = 83, P = 0.001) and a positive correlation was observed among LDL and HDL (r = 0.230, n = 86, P = 0.035). Conclusions: This study observed the incidences in the abnormalities of serum lipid profiles in both nondiabetic and diabetic persons. It also presents the high occurrence of nondiabetic women with dyslipidemia as they presented with high cholesterol, high TG, low HDL-C, and high VLD-L with BMI over 30 kg/m².

Keywords: Abnormal lipid profile, middle-aged, nondiabetic, type 2 diabetic, weight

Introduction

Cholesterol is a steroid compound and it is transported in the blood attached to proteins called lipoproteins. The good lipoprotein is referred to as our high-density lipoprotein (HDL) as it protects our body from the risk of developing cardiovascular diseases (CVDs) and the bad lipoprotein is our low-density level (LDL) which increases our risk of CVD.

Our diet, physical activity, and even hereditary genes can determine the concentration changes in our cholesterol levels. A lipid profile gives the measure of cholesterol, HDL, LDL, very low-density lipoprotein (VLDL), and triglycerides (TGs) which accounts for the accumulation of fat deposits in our system.

Abnormalities in lipid profiles are considered one of the most contributing risk factors for atherosclerosis. It is also associated with an increased risk of smoking, alcohol consumption,[1] physical inactivity, obesity, and low fruit and vegetable intake.[2] Nutrition is another major risk factor as it determines the effect on lipoprotein metabolism in stimulating the production of VLDL.[3]

Abnormalities in lipid concentrations are commonly referred to as dyslipidemia and it is characterized by high TGs, low HDL, high serum VLDL – TGs, and an increase in LDL-C.[4][5]

Studies have revealed the estimated prevalence of dyslipidemia in 28 and 37 million Americans with a body mass index (BMI) over 30 and among 25–29.9, respectively. The prevalence rates were observed to be much lower among the age group of 20–39 years as compared to being higher between the age group of 40 and 79 years.[7]

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In addition, researches have shown that to decrease cardiac mortality, improvements in risk factors such as cholesterol levels and blood pressure has to be addressed, as according to the lipid guidelines presented by the Canadian Cardiovascular Society, abnormal lipid profiles can lead to serious CVDs. Furthermore ratios of total cholesterol (TC)/HDL-cholesterol (HDL-C) and LDL-C thresholds were observed to be preferential in identifying the high-risk patients when compared to just using LDL-C thresholds. It was also highlighted that the criteria for metabolic syndrome included at least three present identifications of abdominal obesity, TGs, blood pressure, and fasting blood glucose over or under a certain range. Dyslipidemia in Trinidad has been considered to be present once fasting TC levels >502 mM (200 mg/dL), TGs >2.26 mM (200 mg/dL), LDL >3.37 mM (130 mg/dL), HDL <0.91 mM (35 mg/dL), and TC/HDL >60 are obtained from biochemical testing. When a cholesterol screening was conducted in Trinidad, testing of 187 patients with a mean age of 42.8 ± 7.0 years showed an increase in body mass index and relative hyperlipidemia with CVD risk factors of increased body mass, increased blood cholesterol, TG, and LDL. The findings also showed the prevalence of men when compared to women with dyslipidemia for CVD regardless of ethnicity. Contrary to this, other researches have displayed that dyslipidemia was more pronounced in women.

Studies have shown and are continuing to show trends in persons globally with dyslipidemia. Trends have developed in Trinidad showing that serum lipid concentrations together with red blood cell membrane can help in the prediction of type 2 diabetes mellitus (T2DM). Abnormal lipid profiles have been observed to be more prominent among the diabetic group. The characteristics of the diabetic group which include high glucose levels, low insulin-resistant levels, and low creatinine were observed to have significant correlations with the abnormal lipid profiles in Trinidad.

The population of Trinidad keeps changing drastically and therefore the impact on the health-care system is as well. This study was conducted to identify and evaluate the abnormalities of serum lipid profiles in both nondiabetic and diabetic persons to decrease the risk for CVDs.

**Methods**

This was a case–control study conducted at the North Central Regional Health Authority (NCRHA) and South West Regional Health Authority (SWRHA) of Trinidad between August 2013 and December 2015. Ethical approval was granted from the Ethics Committee of the Faculty of Medical Sciences (FMS 12/02/13). The University of the West Indies. Inclusion criteria comprised patients over the age of 40 years who fasted overnight and both diabetic and nondiabetic regardless of gender and ethnicity. Exclusion criteria omitted patients presenting with complications of T2DM including nephropathy and retinopathy, immunological disorder, and any other coexisting infection(s).

This study comprised 266 patients from the NCRHA and SWRHA of Trinidad. Of the 266 patients recruited, 126 were diabetic and 140 were nondiabetic.

The suitable patients were informed thoroughly of the project and once interested they were asked to give consent. Upon this, the patients were issued a close-ended questionnaire requesting demographic data.

Clinical measurements of blood pressure, weight, and BMI were then recorded using established protocols. Blood samples were then obtained, processed, and stored for 6 months at −20°C for biochemical analysis.

The samples were then sent for testing at a diagnostic laboratory in which blood glucose, C-reactive protein (CRP), and lipid profile (cholesterol, TGs, HDL-C, and VLDL-C) were measured with a dry chemistry analyzer (Johnson & Johnson Vitros 250, Ortho-Clinical Diagnostics Inc., NY, USA).

**Statistical analysis**

Results obtained were analyzed using IBM Corp. Released 2012. IBM SPSS Statistics for Windows, version 21.0 (IBM Corp., Armonk, NY, USA). Descriptive statistics were calculated for the population selected. The mean and standard deviation among the categorical groups were also obtained using Chi-square test to obtain proper P value. No test for normality was done among the categorical group. Student’s unpaired t-test was used to compare between the male and female groups and among the diabetic and nondiabetic groups. The entire test was assessed as a 95% statistical level of significance.

**Results**

The sample population investigated consisted of 266 elderly participants with baseline characteristics as shown in Table 1. Nearly 47% of the population consisted of diabetic participants.

There was twice the number of female participants overall in the study when compared to males. Therefore, to balance the sample size for Table 2, the same number of women was randomly chosen to match the sample size of men using SPSS software.

The diabetic women had significantly lower TGs ($P = 0.019$) and higher HDL-C ($P = 0.001$) and LDL ($P = 0.003$) when compared to the diabetic men as shown in Table 2. In addition, the nondiabetic females also had higher HDL-C ($P = 0.045$) when compared to their male counterparts.
counterparts. Both diabetic and nondiabetic females exhibited significantly higher BMI of $P = 0.000$.

It was observed that dyslipidemia was present among the nondiabetic populations as the nondiabetics had 55 women and 20 men with high cholesterol, 22 women and 14 men with high TG, 30 women and 25 men with low HDL-C, 42 women and 21 men with high LDL-C, 13 women and 8 men with high VLDL, and also 30 women and 11 men with BMI over 30 kg/m² [Table 3].

Significant differences were observed among the diabetic and nondiabetic groups for cholesterol, TGs ($P = 0.004$), HDL ($P = 0.002$), and VLDL (0.040), regardless of their BMI and weight [Table 4].

The diabetics also showed a higher level of glucose as expected, higher levels of insulin, higher levels of cholesterol, higher levels of TGs, and lower levels of HDL. The systolic and diastolic pressure of the diabetic population was higher when compared to nondiabetic population ($P = 0.001$) [Figure 1].

**Discussion**

The occurrence of hypercholesterolemia for lipid determinants[11] and the prevalence of dyslipidemia among the diabetics have been previously identified within Trinidad,[14] which was clearly seen in our research among the diabetic population.

However, our major aim was to present the occurrence of dyslipidemia among the nondiabetic category. This was clearly observed in Table 3 as numerous patients, both male and female, showed the presence of high cholesterol, high TGs, low HDL, high VLDL, and high LDL. We also observed obese persons with a BMI over 30 kg/m² among the nondiabetic group.

A result like this is very crucial for the prevalence of atherosclerosis as abnormalities in serum cholesterol, TGs, and LDL can progress silently and can lead to more serious complications.

**Table 1: Characteristics of participants in the study population**

| Characteristics | Total ($n=266$) |
|-----------------|-----------------|
| Age, mean±SD    | 50.86±12.872    |
| BMI, mean±SD (kg/m²) | 26.85±6.023    |
| Weight, mean±SD (kg) | 70.82±16.511   |
| Diabetes (%)    | 47.4            |
| Smoking (%)     | 6               |
| Alcohol (%)     | 10.5            |
| Obesity (%)     | 30              |

SD=Standard deviation, BMI=Body mass index

**Table 2: Distribution of lipid profile and body mass index across gender for both diabetic and nondiabetic patients**

| Diabetic | Female ($n=42$) | Male ($n=42$) | $P$ |
|----------|-----------------|---------------|-----|
| Cholesterol (mg/dL) | 229.67±171.890 | 189.76±50.952 | 0.153 |
| TGs (mg/dL)      | 146.46±75.250  | 190.62±141.21 | 0.009** |
| HDL (mg/dL)      | 46.10±9.929    | 39.50±11.362  | 0.006* |
| LDL (mg/dL)      | 133.30±33.428  | 112.14±44.661 | 0.016* |
| VLDL (mg/dL)     | 29.10±16.610   | 25.65±28.721  | 0.368 |
| BMI (kg/m²)      | 32.49±3.978    | 26.12±5.830   | <0.001* |

| Nondiabetic | Female ($n=43$) | Male ($n=43$) | $P$ |
|-------------|-----------------|---------------|-----|
| Cholesterol (mg/dL) | 200.49±39.236 | 90.09±16.142 | 0.734 |
| TGs (mg/dL)      | 151.42±89.241  | 197.49±36.224 | 0.061 |
| HDL (mg/dL)      | 49.02±15.248   | 39.50±11.362  | 0.006* |
| LDL (mg/dL)      | 125.00±40.31   | 125.91±33.66  | 0.910 |
| VLDL (mg/dL)     | 23.59±18.661   | 25.88±11.812  | 0.613 |
| BMI (kg/m²)      | 32.30±4.545    | 25.80±5.491   | <0.001* |

*Statistically significant at $P=0.05$ CI, **Statistically significant at $P=0.001$ CI. HDL=High-density lipoprotein, LDL=Low-density lipoprotein, VLDL=Very low-density lipoprotein, BMI=Body mass index, CI=Confidence interval, TG=Triglycerides

Our study investigated abnormal lipid profiles among the mid-elderly. However, dyslipidemia at a young age if not controlled properly[15] can also lead to chronic noncommunicable diseases (CNCDs) such as diabetes, hypertension, and CVD. Table 4 clearly shows how the diabetic population had high systolic blood pressure which is a contributor to hypertension if not treated properly, and countless of researches have accepted that hypertension is a contributor to CVD.

Some of the major risk factors for CVDs include advancing age, high total serum cholesterol, high LDL-C, low HDL-C, type 2 diabetes, hypertension, cigarette smoking, and family history of CAD. Additional risk factors may also include obesity and inflammatory markers such as high-sensitivity CRP. Nevertheless, evidences have shown that an increase in insulin resistance causes plasma TGs and LDL-C to increase while decreasing HDL-C.[16]

Another major finding showed that the highest lipid abnormalities were observed among the female population in both diabetic and nondiabetic groups. We also observed that women, regardless of diabetic status, had higher BMI when
contrary to high cholesterol, LDL, and TGs, the female population showed much higher levels of HDL in our study. Women in their childbearing age tend to have higher HDL as high estrogenic levels contribute to high HDL, yet this was also observed senior women.

There were also significant differences in systolic pressure among the diabetic and nondiabetic groups. This was expected as persons with T2DM have high cholesterol and LDL-C which was proven in a previous study.[20]

For prevention of CNCDs, it is recommended that persons have their lipid profiles tested regular as diabetics tend to have abnormal lipid profiles when compared to the nondiabetics since associations between low insulin resistance and high cholesterol, TGs, and LDL-C have been observed in this study. These are the prevalent characteristics of the diabetic population globally,[16] and therefore regardless of gender or ethnicity, we highly recommend that special attention should be made to this.

The socioeconomic status, diet, level of exercise, and medications were some of the major limitations to this study which may have been able to give better insight in our population.

The health status of people is on an average determined by their environmental factors, personal behaviors and health practices, biological and genetic factors, and most importantly the health-care systems and services of the country and its availability.[21] It is advised that, as persons grow older, there will be fat deposits, especially in women when compared to men. Healthy diet, aerobic exercise, and regular clinical checkups should be implemented.[22]

Screening for lipid abnormalities should be endorsed among adults, regardless of ethnicity or gender.

**Conclusions**

This study the abnormalities of serum lipid profiles in both nondiabetic and diabetic persons. Screening for lipid abnormalities should be endorsed for all adults, regardless of ethnicity or gender.

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**Conflicts of interest**

There are no conflicts of interest.

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**References**

1. Crook ED, Thallapureddy A, Migdal S, Flack JM, Greene EL, Salahudeen A, et al. Lipid abnormalities and renal disease: Is

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**Table 3: The frequency of the target range values across gender**

|                      | Female (n=181) | Male (n=85) |
|----------------------|---------------|------------|
|                      | T2D (n=84)    | Non-T2D (n=97) | T2D (n=42) | Non-T2D (n=43) |
| Cholesterol (mg/dL)  |               |             |           |               |
| ≥200                 | 53            | 55          | 12        | 20            |
| <200                 | 31            | 42          | 30        | 23            |
| TG (mg/dL)           |               |             |           |               |
| ≥160                 | 27            | 22          | 20        | 14            |
| <160                 | 56            | 75          | 22        | 29            |
| HDL (mg/dL)          |               |             |           |               |
| ≥35                  | 38            | 67          | 10        | 18            |
| <35                  | 46            | 30          | 32        | 25            |
| LDL (mg/dL)          |               |             |           |               |
| ≥130                 | 48            | 42          | 13        | 21            |
| <130                 | 35            | 55          | 29        | 22            |
| VLDL (mg/dL)         |               |             |           |               |
| ≥30                  | 11            | 13          | 11        | 8             |
| <30                  | 25            | 35          | 15        | 17            |
| BMI (kg/m²)          |               |             |           |               |
| ≥30                  | 28            | 30          | 10        | 11            |
| <30                  | 56            | 67          | 32        | 32            |

HDL=High-density lipoprotein, LDL=Low-density lipoprotein, VLDL=Very low-density lipoprotein, BMI=Body mass index, T2D=Type 2 diabetes, TG=Triglyceride

**Table 4: Distribution of lipid profile, pressure, body mass index, and weight among the diabetic and nondiabetic groups**

|                      | Diabetic (n=126) | Nondiabetic (n=140) | P     |
|----------------------|-----------------|---------------------|-------|
| Cholesterol (mg/dL)  | 210.62±106.870  | 202.09±41.785       | 0.877 |
| TG (mg/dL)           | 160.91±101.344  | 133.25±82.568       | 0.004*|
| HDL (mg/dL)          | 44.46±12.231    | 49.48±15.023        | 0.002*|
| LDL (mg/dL)          | 126.56±39.499   | 126.38±36.170       | 0.990 |
| VLDL (mg/dL)         | 31.29±22.279    | 24.49±14.580        | 0.040*|
| Systolic pressure (mmHg) | 135.54±20.033  | 128.18±18.955       | 0.001*|
| Diastolic pressure (mmHg) | 80.63±9.831    | 79.22±11.186        | 0.349 |
| BMI (kg/m²)          | 27.32±5.901     | 26.43±6.120         | 0.181 |
| Weight (kg)          | 71.07±16.051    | 70.60±16.971        | 0.685 |

*Statistically significant at 5% CI. HDL=High-density lipoprotein, LDL=Low-density lipoprotein, VLDL=Very low-density lipoprotein, BMI=Body mass index, CI=Confidence interval

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compared to men. This was consistent with other studies which have shown that women generally have a higher percentage of body fat when compared to men.[17]

BMI accounts for the distribution of adiposity, and when a person crosses a BMI over 25 kg/m², his/her health becomes affected as now fat is having a negative effect on his/her body. The distribution of adipose tissue has been linked to elevated plasma levels[17,18] and more complicated diseases such as CVDs.[19]
dyslipidemia a predictor of progression of renal disease? Am J Med Sci 2003;325:340-8.

2. Chadee D, Seemungal T, Pinto Pereira LM, Chadee M, Maharaj R, Teelucksingh S. Prevalence of self-reported diabetes, hypertension and heart disease in individuals seeking state funding in Trinidad and Tobago, West Indies. J Epidemiol Glob Health 2013;3:95-103.

3. Grundy SM, Barnett JP. Metabolic and health complications of obesity. Dis Mon 1990;36:641-731.

4. Taskinen MR. Diabetic dyslipidemia. Atheroscler Suppl 2002;3:47-51.

5. Mooradian AD. Dyslipidemia in type 2 diabetes mellitus. Nat Clin Pract Endocrinol Metab 2009;5:150-9.

6. Goldberg IJ. Clinical review 124: Diabetic dyslipidemia: Causes and consequences. J Clin Endocrinol Metab 2001;86:965-71.

7. Brown CD, Higgins M, Donato KA, Rohde FC, Garrison R, Obarzanek E, et al. Body mass index and the prevalence of hypertension and dyslipidemia. Obes Res 2000;8:605-19.

8. Genest J, McPherson R, Frohlich J, Anderson T, Campbell N, Carpenter A, et al. 2009 Canadian Cardiovascular Society/Canadian guidelines for the diagnosis and treatment of dyslipidemia and prevention of cardiovascular disease in the adult-2009 recommendations. Can J Cardiol 2009;25:567-79.

9. Bersot TP, Pépin GM, Mahley RW. Risk determination of dyslipidemia in populations characterized by low levels of high-density lipoprotein cholesterol. Am Heart J 2003;146:1052-9.

10. McPherson R, Frohlich J, Fodor G, Genest J, Canadian Cardiovascular Society. Canadian Cardiovascular Society position statement – Recommendations for the diagnosis and treatment of dyslipidemia and prevention of cardiovascular disease. Can J Cardiol 2006;22:913-27.

11. Ezenwaka CE, Premanand N, Orrett FA. Studies on plasma lipids in industrial workers in central Trinidad and Tobago. J Natl Med Assoc 2000;92:375-81.

12. Nakhjavani M, Esteghamatim AR, EsfahaniAm F. Dyslipidaemia in type 2 diabetes mellitus: More atherogenic lipid profile in women. Acta medica Iranica 2006;44:11-118.

13. Nayak BS, Beharry VJ, Armoogan S, Nancoo M, Ramadhin K, Ramesar K, et al. Determination of RBC membrane and serum lipid composition in Trinidadian type 11 diabetics with and without nephropathy. Vasc Health Risk Manag 2008;4:893-9.

14. Nayak BS, Butcher DM, Bujhawan S, Chang D, Chang S, Cabral-Samaroo D, et al. Association of low serum creatinine, abnormal lipid profile, gender, age and ethnicity with type 2 diabetes mellitus in Trinidad and Tobago. Diabetes Res Clin Pract 2011;91:342-7.

15. Jellinger PS, Smith DA, Mehta AE, Ganda O, Handelsman Y, Röbard HW, et al. American Association of Clinical Endocrinologists’ guidelines for management of dyslipidemia and prevention of atherosclerosis. Endocr Pract 2012;18 Suppl 1:1-78.

16. Roger VL, Go AS, Lloyd-Jones DM, Adams RJ, Berry JD, Brown TM, et al. Heart disease and stroke statistics-2011 update: A report from the American Heart Association. Circulation 2011;123:e18-209.

17. Hubert HB. The importance of obesity in the development of coronary risk factors and disease: The epidemiologic evidence. Annu Rev Public Health 1986;7:493-502.

18. Anderson AJ, Sobocinski KA, Freedman DS, Barboriak JJ, Rimm AA, Gruchow HW. Body fat distribution, plasma lipids, and lipoproteins. Arteriosclerosis 1988;8:88-94.

19. Dagenais GR, Yi Q, Mann JF, Bosch J, Pogue J, Yusuf S. Prognostic impact of body weight and abdominal obesity in women and men with cardiovascular disease. Am Heart J 2005;149:54-60.

20. Nasri H, Behradmanesh S, Ahmadi A, Baradaran A, Nasri P, Rafieian-Kopaei M. Association of serum lipids with level of blood pressure in type 2 diabetic patients. J Renal Inj Prev 2013;3:43-6.

21. Ministry of Health. Health Reports Card for Trinidad and Tobago 2011. The Directorate of Health Policy, Research and Planning. Trinidad and Tobago. 2011. p. 1-12.

22. Grundy SM, Goodman DW, Rifkind BM, Cleeman JI. The place of HDL in cholesterol management. A perspective from the National Cholesterol Educational Program. Arch Intern Med 1989;149:505-10.