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Dyslipidemias and 10-years Cardiovascular Risk Scores in Adults in Asmara, Eritrea: Findings from a Community-based Cross-sectional study

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25 **Abstract**

26 **Background:** The objective of this study was to estimate the prevalence of dyslipidemias and associated
27 factors in adults (≥ 35 to ≤ 85 years) living in Asmara, Eritrea.

28 **Methods:** A total of 384 (144 (%) males and 242 (%) females, mean age $\pm$ SD, 68.06 ± 6.16 years)
29 respondents were randomly selected after stratified multistage sampling. The WHO NCD STEPS
30 instrument version 3.1 questionnaire was used to collect data. Measurements/or analysis including
31 anthropometric, lipid panel, fasting plasma glucose (FPG), and blood pressure (BP) were also
32 undertaken.

33 **Results:** The frequency of dyslipidemia in this population was disproportionately high (87.4%) with the
34 worst affected subgroup in the 51-60 age band. The level of awareness was also low. In terms of
35 individual lipid markers, the proportion were as follows: HDL-C (40 mg/dL men and 50 mg/dL females)
36 (55.2%); TC ≥ 200 mg/d (49.7%); LDL ≥ 130 mg/dL (44.8%); TG ≥ 150 mg/dL (38.1%). The mean $\pm$ SD,
37 for HDL-C, TC, LDL-C, non-HDL-C, and TG were 45.28 ± 9.60 ; 205.24 ± 45.77 ; 130.77 ± 36.15 ;
38 160.22 ± 42.09 and 144.5 ± 61.26 mg/dl, respectively. Regarding NCEP ATP III risk criteria, 17.6%,
39 19.4%, 16.3%, 19.7%, and 54.7% were in high or very high-risk categories for TC, Non-HDL-C, TG,
40 LDL-C, and HDL-C, respectively. Among all respondents, 59.6% had mixed dyslipidemias with
41 TC+TG+LDL-C dominating. In addition, 27.3%, 28.04%, 23.0%, and 8.6% had abnormalities in 1, 2, 3
42 and 4 lipid abnormalities, respectively. In terms of Framingham CVD Risk scores, 12.7%, 2.8% were in
43 the high risk and very high-risk strata. Further, the high burden of dyslipidemia coexisted with an equally
44 high burden of abdominal obesity (71.8%), BMI ≥ 25 kg/m² (44.6%), dysglycemia (24.7%), hypertension
45 (24.4%), and physical inactivity. Dyslipidemia was associated with employment status (ref: unemployed
46 vs. employed, aOR 0.48, 95% CI 0.24–0.97, p=0.015) and self-employed (aOR 0.41, 95% CI 0.17–1.00,
47 p=0.018); marital status (ref: not married vs married (aOR 2.35, 95% CI 1.19–4.66, p=0.009); increasing

48 DBP (aOR 1.04 mmHg (1.00-1.09)=0.001) and increasing FPG (aOR 1.02 per 1 mg/dL, 95% CI 1.00–
49 1.05, p=0.001).

50 **Conclusion:** High frequency of poor lipid health may be a prominent contributor to the high burden of
51 CVDs – related mortality and morbidity in Asmara, Eritrea. Consequently, efforts directed at early
52 detection, and evidence-based interventions are warranted.

53 **Keywords:** Dyslipidemia, HDL-cholesterol, LDL-cholesterol, Lipid metabolism, Triglycerides.

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68 **Introduction**

69 Cardiovascular disease (CVD) is a leading cause of mortality and morbidity worldwide. Apart from the
70 high morbimortality, the disease is associated with increases in years of life lost (YLLs), years lived with
71 disability (YLDs) and disability-adjusted life-years (DALY) ^{1,2}. To illustrate, the 2013 global burden of
72 disease (GBD) estimate suggested that CVD caused ~17.8 million deaths globally, corresponding to 330
73 million YLLs and another 35.6 million years YLDs ³. In 2019, ischaemic heart disease (IHD) and
74 cerebrovascular accidents (stroke) were the top-ranked causes of DALYs in persons above 50 years ^{2,3}. A
75 notable aspect of the ongoing carnage is that low and medium-income countries (LMIC) are
76 disproportionately impacted ⁴. For example, data suggests that sub-Saharan Africa (SSA) bears the
77 highest burden of stroke globally (age-standardized stroke incidence rates of ~316 per 100000) ^{5,6}.
78 Overall, and in absolute numbers, CVD causes four to five times as many deaths in LMICs as in high-
79 income countries (HICs). The clinical spectrum of CVD in SSA is further expanded by cardiac
80 complications associated with infectious diseases (e.g. TB - related pericardial effusion and HIV-
81 cardiomyopathy, and combined antiretroviral (cART) related treatment complications) ⁷.

82 The disproportionate impact of CVD in LMIC is largely driven by a complex interplay between
83 population-wide changes in socio-demographic, economic, and lifestyle factors ^{6,8}. Another readily
84 detectable and treatable driver of CVD in SSA is dyslipidemia (Total cholesterol (TC) $\geq$ 200 mg/dl,
85 Triglycerides $\geq$ 150 mg /dl, LDL – C $\geq$ 130 mg/dl, HDL-C $\leq$ 40 mg/dl; male, HDL–C $\leq$ 50 mg/dl; female)
86 ^{4,6}. At present, dyslipidemia, hypertension (HTN), and obesity are considered to be the most important
87 CHD drivers. Worldwide, TC causes about 18% of strokes and 56% of ischemic heart disease (IHD),
88 accounting for 4.4 million deaths annually and 93.8 million DALYs ². More importantly, the highly
89 regarded INTERHEART study demonstrated that dyslipidemia is one of the leading population-level risk

90 factors for Ischemic Heart Disease (IHD) in SSA ⁹. Mechanistically, dyslipidemia triggers endothelial
91 dysfunction – a process that is central to the pathogenesis of atherosclerosis, thrombosis, and
92 hypertension.

93 Despite the contribution of dyslipidemia to the high and rising burden of CVD in SSA ^{4,6}; the condition is
94 under-diagnosed, under-treated and under-described. This represents a missed opportunity given the fact
95 that targeting risk drivers of CVD at the population level by a combination of simple, low cost, efforts
96 could avert more than 50% of the associated morbidity and mortality ¹⁰. This, therefore, cannot be
97 overemphasized: poor lipid health in adult populations in SSA is now a true epidemic and public health
98 crisis that both patients and clinicians must face. Previously identified barriers to addressing this problem
99 include lack of awareness (among the public and health-care professionals); high cost of diagnosis and
100 treatment; a dearth of well-trained health personnel; lack of local clinical practice guidelines; under-
101 treatment and a limited understanding of its epidemiology ⁴. Overall, the lack of reliable health
102 information/statistics and a severe lack of community-based epidemiological data is a source of serious
103 concern as it handicaps public health strategies directed at prevention and treatment/or management of
104 dyslipidemia in the region.

105 Much of the description in the foregoing paragraph applies to Eritrea. At present, very little is known
106 about the risk factors associated with CVD in any population in Eritrea. The lack of data is extremely
107 concerning given the fact that World Health Organization (WHO) fact-sheets have consistently shown
108 that CVD-related mortality in the country is disproportionately high (388.1 vs. 282.2 per 100 000 in
109 males and females, respectively) ¹¹. Interestingly, the country has one of the lowest prevalence of
110 overweight/obesity (mean BMI = 20.5 (95% CI: 19.9-21.1 and BMI $\geq$ 25 kg/m² = 17.7% (95% CI: 14.7%
111 - 20.2%) in SSA ^{11,12}. The prevalence of other known drivers such as tobacco use, irresponsible alcohol
112 consumption, hypertension, and Diabetes mellitus are modest or low ³. One facet of this problem that has

received little attention is the burden of dyslipidemia and its possible contribution to the excess burden of CVD in the country.

Therefore, this study was designed to generate population-level data on the burden of dyslipidemia in the adult population in Asmara, Eritrea. Beyond the focus on dyslipidemia, using multivariable risk scores/prediction algorithms to identify persons at higher risk is a well-established intervention strategy and has proved to be cost-effective in multiple jurisdictions^{13,14}. Therefore, we computed Framingham CHD risk scores (FCRS) of the participants. Data on these markers can be crucial in designing evidence-based, context-specific community-level and/or individualized interventions. The information can also be leveraged in the future to evaluate whether CVD risk is declining, stagnating, or even increasing. Further, estimating CVD risk using FCRS presupposes the collection of data on an expanded list of risk factors. To this end, data regarding the prevalence and distribution of individual risk factors such as obesity, fasting blood sugar (FPG), blood pressure (BP), among others, are also presented.

Methods and Design

Study design, setting and participants

This population-based cross-sectional study was conducted in Asmara, Eritrea, from October 2020 to November 2020. Individuals aged between 35 – 75 years living in the study area were targeted. Located in the central region (Zoba Maekel), Asmara serves as the capital city and has the largest population cluster in the country (approximately 658,516 persons). Administratively, the city is divided into 13 *Zobas* (sub-zones) (Mai-Temenay, Edaga-Hamus, Akria, Paradizo, Aba-Shawel, Arbaete-Asmara, Maekel-Ketema, Tsetserat, Tiravelo, Sembel, Godaef, Gejeret and Geza-banda).

Sample size determination and sampling procedure

“A single population-proportion formula was used to determine the sample size¹⁵.” Using a proportion of 50% (dyslipidemia) (we used 50% prevalence because there were no credible data on the prevalence of

dyslipidemia in Eritrea – this resulted in the highest sample size), 95% confidence interval (CI), 5% type I error level, 80% power and adjustment for response rate; the sample size was calculated to be 384 participants. To identify eligible study participants, a stratified multistage sampling design was followed. Briefly, the total sample size was proportionally allocated to the 13 sub-zones. After eliminating sparsely populated Zip-codes within each Sub-zone, one Zip code (enumeration area (EA)) was selected using a simple random sampling technique (computer-based random number generator). The appropriate number of households (HH) per EA was subsequently selected using random sampling. Households were excluded from the study if all members of the HH were outside the required age range (≥ 35 to ≤ 85 years). If an abandoned house was encountered during the random selection, it was replaced by the next inhabited household. Where appropriate, eligible participants per HH were selected using the Kish method (a random selection of eligible individuals at the HH level). Potential participants were excluded if they were bedridden, pregnant, breastfeeding, on specific medications (Steroid, β -adrenergic blockers, Thiazide diuretics, Anti-HIV medication among others), or had Diabetes Mellitus (DM). In the absence of eligible participants during the visit, a second visit was offered to grant potential participants the opportunity to participate in the survey. In case of the participant's absence during the entire study period, replacement by the current person who was 35 and above was undertaken. All eligible individuals who provided written informed consent were enrolled.

Data collection, measurements, and definitions

Data were collected using a modified version of the WHO NCD STEPS instrument version 3.1¹⁶. To accommodate unlettered participants, the questionnaire was translated from English to Tigrigna (a local Language) by a language expert. Overall, the instrument incorporates queries on a range of well-established cardio-metabolic risk factors and is separated into four sections/Steps. Step 1, includes questions on socio-demographic characteristics (sex, age, the highest level of education, occupation, marital status, ethnicity as well as family history of DM (diabetes mellitus); Step 2 explores lifestyle

160 factors (exercising, sedentarism, smoking, alcohol consumption, and history of hypertension); Step 3
161 explores physical measurement (anthropomorphic measurements, blood pressure (BP) measurements,
162 among others); and Step 4 describes biochemical measurements including Fasting plasma glucose (FPG),
163 TG, TC, HDL.

164 **Anthropometry and Blood Pressure Measurement**

165 **Anthropometric measurements:** Standardized techniques following WHO-STEPS surveillance manual
166 and calibrated equipments were used for anthropometric measurements. All anthropometric
167 measurements were performed by well-trained investigators. Weight, Height, Hip circumference
168 (measured at the widest part of the buttocks), and WC (measured at the iliac crest) were measured as per
169 established protocols using standardized instruments/equipments. Abdominal obesity was defined as per
170 IDF specification ($WC \geq 94$ cm in males and ≥ 80 cm in females). For population-level comparisons,
171 overweight and obesity were defined using body mass index (BMI) – (where $BMI = \text{weight (kg)} \div$
172 $(\text{height (m)})^2$). A per WHO BMI specification, a $BMI < 18.5 \text{ kg/m}^2$ was categorized as underweight; $\geq$
173 $18.5 - 24.9 \text{ kg/m}^2$ was classified as normal weight; $BMI \geq 25 - 29.9 \text{ kg/m}^2$ as being overweight and a
174 $BMI \geq 30 \text{ kg/m}^2$ was classified as obese. The waist-to-hip ratio (WHR) and the waist-to-height ratio
175 (WHtR) were also calculated. For purposes of analysis, a $WHR \geq 0.90$ for men and ≥ 0.85 for women
176 were considered abnormal as per IDF guidelines.

177 **Blood pressure (BP):** Sitting BP was measured as per 1999 WHO/International Society of Hypertension
178 guidelines for the management of hypertension protocol, on the first day of contact, using a standard
179 adult arm cuff of a well-calibrated Omron Digital Blood Pressure machine (Omron M6, Omron, Kyoto,
180 Japan). After 10 minutes of rest, 3 measurements were taken after an interval of ~5 minutes. The
181 participant BP was computed from the average of the 2nd and 3rd measurements. Systemic hypertension
182 (HTN) was defined as systolic blood pressure (SBP) ≥ 140 mmHg or a diastolic blood pressure (DBP) $\geq$
183 90 mmHg ¹⁷, or previous diagnosis of HTN or self-reports of antihypertensive medication use.

184 **Biochemical testing**

185 **Specimen collection and analysis**

186 Participants who consented to participate in the study were requested to fast for 8 hours overnight before
187 blood sample collection. As per established protocols, 5 ml of blood was obtained from the median
188 cubital vein, after more than 9 hours of fasting. The samples were transported in an icebox within 2 hours
189 of collection. Total Cholesterol (TC), Triacylglycerol (TG), and High-density lipoprotein cholesterol
190 (HDL-C), Fasting Plasma Glucose (FPG) were analyzed, as per manufactures instructional guidelines,
191 using Beckman Coulter (AU480 Chemistry System). In addition, quality control measures were
192 performed according to the manufacturer's recommendation and laboratory guidelines.

193 **Lipid panel:** As highlighted above, TC, TG, HDL - C were evaluated. The lipid panel components were
194 categorized using the National Cholesterol Education Program Adult Treatment Panel III (NCEP-ATP
195 III) and ADA guidelines. Separately, Friedewald formula ($\text{LDL [mg/dL]} = \text{total cholesterol [mg/dL]} -$
196 $\text{HDL [mg/dL]} - \text{triglycerides [mg/dL]}/5$) was used to estimate LDL-C concentration (participants with
197 TG level > 400 mg/dL were excluded in this analysis). In addition, non-HDL-C was computed as TC-
198 HDL-C. We calculated four lipid ratios: TG/HDL; TC/HDL; LDL/HDL and non-HDL/HDL ratio. A
199 TG/HDL ratio ≥ 5 was regarded as abnormal. Dyslipidaemia was defined as any of the following
200 abnormalities: TC ≥ 200 mg/dL (≥ 5.2 mmol/l); LDL-C ≥ 130 mg/dL (≥ 3.4 mmol/l); TG ≥ 150 mg/dL
201 (≥ 1.7 mmol/l); HDL-C (≤ 40 mg/dL (< 1.04 mmol/l) in male and ≤ 50 mg/dl (< 1.3 mmol/l) in female) ¹⁸
202 or reported use of anti-lipid medication. Further, mixed-dyslipidemia was defined as the concurrent
203 presence of 2 or more lipoprotein abnormalities.

204 **Fasting plasma glucose and Glycated hemoglobin (HbA_{1C}):** FPG was analyzed in all consenting
205 patients. Accordingly, the enrollees were classified as normal (FPG < 100 mg/dL) (< 5.6 mmol/L);
206 Prediabetes (FPG ≥ 100 and ≤ 125 mg/dL) ($< 5.6 - \leq 6.9$ mmol/L); undiagnosed DM (FPG ≥ 125 mg/dL)

207 (≥ 7 mmol/L). Taking into account the cost of the study and the level of sensitivity and specificity
208 required for presumptive diagnosis of DM, HbA_{1c} analysis was restricted to FPG ≥ 125 mg/dL. As per
209 ADA criteria, HbA_{1c} $< 5.7\%$ was regarded as normal; HbA_{1c} between $5.7\% - 6.4\%$ was classified as pre-
210 diabetes and HbA_{1c} $> 6.5\%$ was classified as undiagnosed DM.

211 **Framingham CVD Risk Score**

212 The Framingham CVD Risk Score Calculator (10-years, general cardiovascular disease: Framingham,
213 2008 paper) was used to estimate 10-year CVD risk. The calculator incorporates a range of traditional
214 CVD risk markers including HDL, age range, hypertension treatment, smoking, and TC. Similar, to
215 Reiger *et al.*, Framingham CVD Risk Score was ascribed to participants based upon the low risk ($< 3\%$);
216 moderate ($\leq 3\%$ to $< 15\%$); high (≥ 15 to $< 30\%$), and very high ($\geq 30\%$) ¹⁹.

217 **Data analysis**

218 The completed questionnaires were entered on CSPro software (version 7.0). Keying errors were handled
219 by the double entry of data. The data was analyzed using Statistical Package for Social Sciences version
220 20.0 for Windows (SPSS Inc., Chicago, Illinois, USA.). Enrollee characteristics were summarized using
221 frequencies and percentages. Depending on the distribution, continuous data were presented as mean $\pm$
222 standard deviation (SD) or median $\pm$ interquartile range (IQR). Data normality and homoscedasticity, and
223 multicollinearity were tested using suitable statistics. Unadjusted statistical comparisons between
224 categorical variables and categorical outcomes were made using the Chi-square (χ^2) test or Fisher exact
225 test. Depending on data distribution, the t-test and one-way analysis of variance (ANOVA) or their non-
226 parametric equivalents were employed. To identify factors that were associated with specific lipid
227 abnormalities or dyslipidemia, Binomial logistic regression with backward variable removal (backward:
228 conditional) was developed. Subsequently, crude (COR) and adjusted odds ratios (aOR) and associated
229 95% confidence (95% CI) were reported. To correct for the impact of multiple comparisons, the

230 Bonferroni correction was applied. All p -values were 2-sided and the level of significance was set at $p <$
231 0.05 for all analyses. Missing values or refusals to answer questions were handled by exclusion from
232 analysis.

233 **Ethical consideration**

234 Administrative and ethical approval was granted by the Eritrean Ministry of Health (EMOH) research
235 proposal review and ethical clearance committee. Written informed consent was obtained from each
236 participant in the local language (Tigrigna) as per the procedures approved by the EMOH ethical
237 committee. Importantly, enrollees were duly informed of their non-negotiable right to instantly terminate
238 their participation in the study. Strict adherence to approved laboratory protocols was observed during
239 specimen collection, processing, and testing. All methods were performed in accordance with the
240 national guidelines and regulations.

241 **Result**

242 **Demographic characteristics, patient history, anthropometry, and clinical measurements**

243 Altogether, 533 individuals were approached for participation in the study. Of these, 33 potential
244 participants were excluded due to a previous diagnosis of diabetes mellitus (DM), 4 due to pregnancy,
245 and 24 were on medication. In addition, 69 failed to grant consent, and 17 were non-respondents. The
246 final samples comprised 384 respondents (144 (37.5%) males and 242 (63%) females). The mean $\pm$ SD
247 age of the respondents was 52.17 ± 13.29 years (male, 54.85 ± 14.81 vs. female, 50.57 ± 12.04 years; p -value
248 = 0.004). The BMI ranged from 15.81 – 39.56 kg/m^2 , with a mean of 24.82 ± 4.09 kg/m^2 (95% CI: 24.4-
249 25.2). Females compared to males had a lower level of education (No formal education: 79.2% in
250 females vs 20.8% in males); more likely to be single (62.5%), divorced (84.6%), or widowed (84.6%);
251 more likely to be unemployed (88.1%) and were less likely to smoke (15.41%) or drink alcohol. See
252 Table 1 for additional information.

The relationship between specific demographic characteristics, anthropometry, clinical measurements, and specific lipid markers was also evaluated. Overall, women had significantly higher TC (209.9 ± 49.32 vs 197.39 ± 37.96 mg/dL, $p=0.009$), HDL-C (47.53 ± 9.7 vs 41.5 ± 8.09 mg/dL, $p = 0.02$) and LDL-C (133.98 ± 38.31 vs 125.35 ± 31.56 mg/dL, $p = 0.02$) compared to men. In contrast, men had higher mean values for TGs (153.5 ± 62.59 vs 139 ± 59 mg/dL, $p = 0.026$) and TC/HDL ratio (4.84 ± 0.91 vs 4.50 ± 1.00 , $p < 0.001$). In addition, respondents with prediabetes and undiagnosed DM had higher values in TG compared to respondents with normal FPG; lower HDL-C, and higher TC/HDL ratio. Finally, increasing age (to ≤ 60 years) was characterized by higher values in TC, LDL-C, non-HDL-C, and TC/HDL ratio. See Table 2 for additional information.

Prevalence of dyslipidemia and other lipid panel abnormalities

At least one dyslipidemia was present in 87.4% (95% CI: 84% - 90.8%) of the study respondents. The most prevalent lipid abnormality was low HDL-C (55.2%) followed by high TC [192(49.7%)]; high LDL 173 [(44.8%)] and hypertriglyceridemia [147 (38.1%)]. As seen in Figure 1, the prevalence of high TC, high TG, low HDL, and high LDL increased with age.

Participants Lipid Profiles as per the ATP III, Adult Treatment Panel III Risk Schema

These analyses revealed that women were disproportionately affected across all TC risk strata: borderline (71 (57.3%) in females vs 53(42.7) in males) and high risk (52 (76.5%) female's vs 16 (23.5%) in males). Similarly, the pattern was significant across non-HDL-C risk bands (borderline high (60 (62.6%) in female's vs 46(37.4) in males), high risk (32 (66.7%) female's vs 16 (33.3%) in males) and very high (32 (88.9%) in female's vs 3(11.1%) in males. The same pattern was observed across LDL-C and HDL-C risk categories. Mean values for TC, TG, LDL-C, HDL-C, non-HDL-C, and TC/HDL ratio are also presented. See table 3 for additional information.

Prevalence of mixed Dyslipidemias

277 As seen in Table 4, mixed dyslipidemia, defined as the presence of ≥ 2 lipid abnormalities, was also
278 analyzed. Among all respondents, 59.6% (95% CI 54.6% - 64.6%) had mixed dyslipidemias. Most
279 notably, respondents with abnormalities in two lipid variables presented either with elevated TG plus
280 low HDL-C (39(10.2%) or high TC plus high LDL-C, (51(13.4%). High TC, TG, and LDL-C was the
281 most common presentation (80 (20.9%) in respondents presenting with 3 lipid abnormalities. All four
282 dyslipidemias occurred in 33(8.6%) of the respondents. See table 4 for further information.

283 **Logistic regression analysis of factors associated with lipid levels**

284 **Factors associated with elevated Non-HDL-C, TG, and TC**

285 We summarize here the results of the multivariate models in Table 6. In this analysis, alcohol
286 consumption (aOR 2.24, 95% CI 1.34-3.74, $p = 0.002$), Family history of DM (aOR 1.04, 95% CI 1.02–
287 1.07, $p = 0.001$), FPG (aOR 1.02 1 mg/dl, 95% CI 1.00 – 1.04, $p = 0.008$) had a strong independent
288 association with abnormal non-HDL-C. In a separate analysis, abnormal TG was independently
289 associated with female gender (aOR 1.71, 95% CI 1.10 – 2.65, $p = 0.008$); Alcohol consumption (yes,
290 aOR 1.76, 95% CI 1.11 – 2.79, $p = 0.014$); WC (aOR 1.03 per 1 cm, 95% CI 1.01 – 1.05, $p = 0.002$).
291 Although present in the adjusted model, the association between TG and family history of DM (aOR
292 0.536, 95% CI 0.264 – 1.09, $p = 0.086$) or FPG (aOR 1.01 per 1 mg/dl, 95% CI 0.998 – 1.02, $p = 0.114$)
293 was not significant. Relatedly, abnormal TC was associated with alcohol consumption (aOR 2.01, 95%
294 CI 1.30 – 3.11, $p = 0.002$); Presence of hypertension (aOR 2.19, 95% CI 1.35 – 3.53, $p = 0.001$) and
295 increasing concentration of FPG (aOR 1.01 per 1 mg/dl, 95% CI 1.00 – 1.02, $p = 0.001$).

296

297 See table 5 for further information

298 **Factors associated with abnormalities in HDL-C, LDL-C, and TC/HDL-C**

299 In this analysis, the data demonstrates that respondents who were employed or self-employed
 300 were less likely to present with low HDL – employed (aOR 0.51, 95% CI 0.32 – 0.83, p = 0.001)
 301 and (aOR 0.52, 95% CI 0.27 – 0.975, p = 0.034) compared to those who are unemployed (mostly
 302 housewives). Further, low HDL-C was associated with increasing FPG (aOR 2.10, 95% CI 1.25
 303 – 3.52, p = 0.001). Separately, analysis of the factors associated with abnormal LDL (>130
 304 mg/dl) implicated a connection with alcohol consumption (aOR 1.80, 95% CI 1.16 – 2.81, p =
 305 0.001); increasing WC (aOR 1.03 per 1 cm, 95% CI 1.00 – 1.05, p = 0.001) and presence of
 306 hypertension (aOR 2.06, 95% CI 1.26 – 3.37, p = 0.001). Although BMI ≥ 25 kg/m² was
 307 associated with abnormal LDL in the crude model, the relationship was attenuated in the
 308 adjusted model. Finally, presence of dyslipidemia (at least 1 lipid abnormality) was associated
 309 with employment status (ref: unemployed vs. employed, aOR 0.48, 95% CI 0.24 – 0.97, p =
 310 0.015) and self-employed (aOR 0.41, 95% CI 0.17 – 1.00, p = 0.018); marital status (ref: not
 311 married vs married (aOR 2.35, 95% CI 1.19 – 4.66, p = 0.009); increasing DBP (aOR 1.04
 312 mmHg (1.00-1.09) = 0.001) and increasing FPG (aOR 1.02 per 1 mg/dL, 95% CI 1.00 – 1.05, p
 313 = 0.001). See Table 6 for additional information.

Framingham Risk Scores: Magnitude and Relationships

Framingham risk scores for all the respondents were computed as highlighted in the previous
 section. In the overall sample (N = 384), 166(43%) of the respondents were in the low-risk strata;
 160(41.5%) in the moderate risk strata; 49 (12.7%) in the high-risk strata and 11(2.8%) were in
 the very high-risk strata. Figure 2A. Disaggregation of the data for selected lipid abnormalities is
 shown in Figure 2B. Briefly, 23.1% of the respondents with elevated LDL and 21.4% of patients
 with high TC/HDL ratio were in the high-risk category.

Socioeconomic factors may play differential underpinning roles in CVD. Consistent with the
 data presented above, a large proportion of patients with No formal education or

divorced/widowed had a high 10-year risk of CVD – 27.3% and 30.8% respectively. As expected, a connection between Age, FPG, and FRS was also demonstrated. See additional information in Table 7.

Discussion

Although more than 80% of the global burden of CVD in LMIC, knowledge of the importance of risk factors is largely based on extrapolations from high-income countries (HIC) ⁴. Furthermore, country-level analysis reveals important differences in the combination of CVD risk factors (age, gender, tobacco smoking, diabetes mellitus (T2DM), lipid abnormalities/dyslipidemia, hypertension, obesity, and a family history of CVDs) operating at any one time ²⁰ within or between countries is highly variable. For this reason, updated, context-specific data, on the burden or factors associated with CVD incidence, prevalence, morbidity, or mortality has been emphasized. In this study, the first of its kind in Eritrea, we evaluated the magnitude/burden, risk factors, and patterns of dyslipidemia in the adult population in Asmara, Eritrea.

Overall, 87.4% of the study respondents had at least 1 lipid abnormality as per the NCEP ATP III guideline with a significant difference between males and females. Methodological differences notwithstanding, the estimate reported in this study is disproportionately high. However, comparable results have been reported by some investigators in the region. A study conducted in Nigeria reported a frequency of 85.9% ⁶, 67.3% in South Africa ¹⁹, United Arab Emirates 72.5% ²¹, Makelle city, Northern Ethiopia (66.7%) ²², India (ICMR-INDIAB study) (79%) ²³, Northern China (31.2%) ²⁴, and USA (52%) ²⁵. Separately, some estimates of the prevalence of dyslipidemia among adults in Africa place the value between 15% to about 50% ^{4,26}. In contrast, the Africa Middle East Cardiovascular Epidemiological (ACE) Study reported a

high prevalence of 70% ²⁷. In other words, the frequency of dyslipidemia in some parts of Africa is similar or even higher than what has been reported in some high-income countries (HIC) – (33–75%) ²⁸. These estimates should raise concern considering the well-documented association between dyslipidemia and CVD.

Another important finding was the fact that the average values of TC, LDL, non-HDL-C; were above the cutoffs for abnormal values. Historically, multiple authors have argued that the mean values of lipid and lipoprotein biomarkers for populations in SSA are lower. For example, in the widely regarded INTERHEART study, the highest mean TC concentrations (> 200 mg/dL) were observed in Europeans and other Asians, intermediate levels (180–190 mg/dL) were observed for most of the regions, and the lowest means (< 160 mg/dL) was observed in SSA ²⁸. High mean values for multiple lipid markers have been observed by multiple investigators in the region ⁴. Therefore, the presumption that the mean values for lipids are low in SSA warrants additional scrutiny.

In terms of individual lipid and lipoprotein markers, the most predominant abnormality was low-HDL (55.2%), followed by hypercholesterolemia (49.7%), high LDL-C (44.8%), and Hypertriglyceridemia (38.1%), respectively. Similar patterns, with notable exceptions, have been reported in other settings in the region. For example, a study from Uganda reported that low HDL-C was the predominant lipid sub-fraction abnormality (71.3%), then high TC (6.0%), elevated LDL-C (5.2%), and elevated TG (5.0%) ²⁹. In a study conducted in South Africa (Soweto) ⁷, the patterns among participants of Africa descent were as follows: low HDL-C (63%), high LDL-C (44%), high TC (39%), and high TG (23%). In more recent years, a prominent meta-analysis estimated that the frequency of low HDL-C was 37.4% (95% CI: 29.4 – 45.7). Other dyslipidemias were in the following order: high LDL-C, 28.6% (95% CI: 15.8–43.5)

< Total Cholesterol, 25.5% (95% CI: 20.0 – 31.4) < high TG 17.0%, 95% CI: 11.9-22.7) ⁴. Overall, but with notable exceptions ^{22,30}, HDL-C was the most common isolated lipid abnormality ⁸. Emphasizing this point, some investigators have suggested that over the last 50 years, a decline in HDL-C concentration has been gradually occurring in Africa ³¹. Separately, the Heart of Soweto Registry Study reported that low HDL-C is a common presentation in individuals with non-communicable heart disease ⁸. Although causality was not inferred, and extrapolation of the data to this setting may be dubious; the research raised pertinent issues which should be explored in this setting.

Although the high frequency of low-HDL in populations in SSA is well documented; the importance of this phenomenon is still elusive. Part of the problem is attributable to the ambiguity concerning the relationship between HDL-C and CVD. For example, genetic studies using Mendelian randomization and longitudinal studies present contrasting results ³². Nevertheless, and considering the role of HDL-C in reverse TC transport, its anti-oxidant, anti-thrombotic, and anti-inflammatory properties; its cardioprotective role is presumed to be substantial. At present, research implicates a strong genetic contribution (~50%) to the observed variability of serum HDL-C at the population level ³³. Presumably, 50% of the observed variation has been linked to a diverse range of modifiable risk factors including insulin resistance/type 2 diabetes mellitus (T2DM), BMI > 25 Kg/m², sedentarism/physical inactivity, β -blockers, progestational agents, cigarette smoking, and very high carbohydrate intakes ³⁴. Other inputs include infection and inflammation ³⁵.

In our study, we established a positive association between low HDL-C, elevated FPG, and employment status (predominance in the unemployed portion of the population). Another notable outcome was the weak association between low HDL-C and BMI categories. Similar to other

studies ^{34,36}, consumption of alcohol was associated with increased concentration of HDL. In general, the mechanisms underpinning the positive correlation between alcohol consumption and HDL-C concentrations are not known, although it has been hypothesized that alcohol may increase HDL-C by mediating the transport of apolipoprotein A1 (Apo-A1). Even though, an investigation on the potential link between low HDL and genetical differences in the Eritrean population has not been undertaken (such studies are warranted); a potential contribution should not be overlooked. This notwithstanding, we believe that a large part of the observed outcome can be explained by physical inactivity and dietary factors (low - fat diet and/or carbohydrate over nutrition – include high intake of fructose-containing sugars). Research in runners and the Framingham study demonstrated a strong inverse relationship between HDL-C concentration and physical activity ³⁷. Likewise, carbohydrate over-nutrition can also lead to enhanced *de novo* lipogenesis and subsequent induction of ectopic lipid accumulation or dyslipidemia. In all, differences in lipid concentrations around the world have been attributed to dietary differences ³⁴. Unfortunately, individual-level data on nutrition was not documented in this study. Therefore, this explanation, although plausible, is speculative at best. Regardless, much work remains to be done before the mechanisms associated with low HDL-C in SSA populations are fully elucidated.

Genetic, histopathologic, observational, and interventional studies, have established the important role of dyslipidemias in the development of CVD and coronary heart disease. The well-respected Multiple Risk Factor Intervention Trial (MRFIT) demonstrated a J-shaped curvilinear relationship between TC and CVD mortality ³⁸. Likewise, TC/HDL-C and ApoB/apoA1 ratios provide equivalent information about CVD risk. Further, the evidence that LDL – C prospectively predicts hard CVD events (coronary death, MI, and stroke) is

unequivocal. However, whether or not TG levels are a causal risk factor for CVD remains unclear. Regardless, we have to note that TG, TC, and LDL-C existed, rarely, in isolation in this setting. Therefore, the high proportions and, to some extent, mean values of TC, LDL-C, TG, TC/HDL-C, and non-HDL-C observed in this setting should raise concern. To this end, dissecting the factors associated with abnormalities in relevant lipid parameters can provide useful data for targeted public health intervention.

To a large extent, most associations of TG, TC, TC/HDL-C, LDL-C, and Non-HDL-C were in the expected direction. High TC was independently associated with alcohol consumption, hypertension, and FPG. Similar to other studies ³⁹, elevated LDL-C was associated with alcohol consumption, WC, and hypertension. These risk factors were also associated with non-HDL-C and TG (add sex) in this study. Remarkably, a large proportion of participants with elevated LDL-C were in the Framingham risk scores high-risk category. Another interesting relationship was the observed association between TC/HDL-C ≥ 5 ratio and sex (higher in males); WC, hypertension, and FPG. Despite the broad agreement between this study and other studies, notable exceptions were observed. For example, BMI ≥ 25 kg/m² had only one association (LDL-C). This was in contrast to studies that have uncovered a significant relationship between elevated BMI, high TG, and low HDL-C ⁴⁰. We are unable to provide definitive explanations why BMI is a poor marker of dyslipidemias in this setting. However, we found a significant relationship between WC and multiple dyslipidemias in the multivariate analysis – Non-HDL-C, LDL-C, TC/HDL, and TG. As previously noted ⁴¹, the use of WC for public health screening or clinical evaluation of patients is still limited in Eritrea. In this regard, the current study merely adds to the evidence on its utility within in setting.

Furthermore, troubling associations and patterns were apparent in this population. The high frequency of dyslipidemia in women (high regardless of menopausal status), the unemployed, those who are divorced/or widowed, and those with lower levels of education points to a gathering problem among a vulnerable subgroup of the population. The same pattern was observed when FRS was computed. The clustering of CVD risk factors among the unemployed, in populations of low socioeconomic status, or among the less educated strata of the society is well documented ³⁴. According to some authors, education mediates the risk of CVD through urbanization, unemployment, access to information/awareness, food, social support and cohesion, and individual health behaviors. In any case, our data support the possibility that physical inactivity/sedentarism and excess weight gain (particularly abdominal obesity) are significant contributors to dyslipidemia in this setting.

Another important finding was the fact that combined dyslipidemia is more common in this population (68.6%) compared to single lipid abnormalities. For example, 28.04%, 23%, and 8.6% of the study participants had abnormalities in two, three, and four lipid components, respectively. The most common combination was high TC + TG + LDL-C (20.9%). The proportion of participants with high TC+ low HDL-C and high TG + low HDL-C was also substantial – 13.4% vs. 10.2%, respectively. Overall, this result points to the importance of mixed dyslipidemia in addition to the single lipid abnormalities in this population. Importantly, the simultaneous presence of elevated TC + LDL-C concentrations is particularly notable given their proatherogenic role. Further, evidence from epidemiologic studies suggests that the co-occurrence of high TG + low HDL-C concentrations (atherogenic dyslipidemia) is a strong risk factor for coronary Heart Disease (CHD), with *post hoc* analyses of several studies suggesting that these individuals have the highest rate of hard coronary events ^{42,43}. In this setting, the

picture is unclear, particularly when mixed dyslipidemia is superimposed upon other predisposing factors (genetic, cultural, and behavioral, among others). Nevertheless, there is little doubt that these combinations, by themselves can accentuate CVD ²⁰. Crucially, the magnitudes of mixed dyslipidemias reported in this study are much higher than what has been reported in some HIC ^{20,44}. Unfortunately, we could not locate comparable community-based studies on the epidemiology of mixed dyslipidemias from the region. Therefore, rigorous population-based prospective investigations are necessary to determine the risk associated with the co-presence of specific lipid abnormalities.

Strengths and Limitations

This study is not without limitations. First, the cross-sectional nature of the study limits the dissection of causality. In addition, the fact that the population was mostly composed of urban residents limits the generalizability of our findings. The use of a researcher-administered questionnaire to capture data on specific variables may be affected by the recall, social desirability, and outcome misclassification biases. Lastly, the Framingham risk score and Friedewald equation for LDL-C estimation have not been validated in this population; hence the results should be used with caution. Despite the above limitations, and in the absence of longitudinal studies, this investigation represents a major first step towards getting baseline on lipid profiles in Asmara, Eritrea. Attempt to analyze the frequency of mixed dyslipidemia along with Framingham risk score also adds to its uniqueness.

Conclusion

This study uncovered many important findings. First, the frequency of dyslipidemia in this population was disproportionately high (87.4%). In terms of relative frequencies, the most prevalent lipid abnormality was low HDL-C (55.2%) followed by high TC (49.7%); high LDL (44.8%), and hypertriglyceridemia (38.1%). Regarding the NCEP ATP III risk category, 68(17.6%), 75(19.4%), 63(16.3%), 76(19.7%), and 211(54.7%) were in high or very high-risk categories for TC, Non-HDL-C, TG, LDL-C, and HDL-C, respectively. Among all respondents, 59.6% had mixed dyslipidemias and the most predominant mixed dyslipidemia was the TC+TG+LDL-C combination. In Addition, 106 (27.3%), 107(28.04%), 88(23.0%), and 33(8.6%) had abnormalities in 1, 2, 3 and 4 lipid abnormalities, respectively. In terms of Framingham risk scores, 166(43%), 160(41.5%), 49 (12.7%), 11(2.8%) were in the low-risk, moderate risk, high-risk, and very high-risk strata. Altogether, it's our submission that dyslipidemia is a principle contributor to CVD risk in this setting. The level of awareness is low and most study participants were not on any type of treatment. Interestingly, binomial logistic regression demonstrated that presence of dyslipidemia was associated with employment status (ref: unemployed vs. employed, aOR 0.48, 95% CI 0.24 – 0.97, p = 0.015) and self-employed (aOR 0.41, 95% CI 0.17 –1.00, p = 0.018); marital status (ref: not married vs married (aOR 2.35, 95% CI 1.19 – 4.66, p = 0.009); increasing DBP (aOR 1.04 mmHg (1.00-1.09) = 0.001) and increasing FPG (aOR 1.02 per 1 mg/dL, 95% CI 1.00 – 1.05, p = 0.001). Taken together, these observations call for concerted, effort directed at scaling up early recognition and treatment, including optimal pharmacological and non-pharmacological therapy at all levels of care.

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Authors' contributions

OOA, NF, NTH, WYW, FTZ, HNL, TAW, THT, EYG conceived of the study and participated in the design. NTH, WYW, FTZ, HNL, TAW, THT collected data and performed laboratory experiments. OOA, NF, supervised the data collection process. OOA, STM performed the statistical analysis, and wrote the initial draft. OOA, STM, NF, EYG reviewed/edited the manuscript. All authors read and approved the final manuscript.

Additional Information

The corresponding author is responsible for submitting a competing interests statement on behalf of all authors of the paper. The authors have no conflict of interest to declare on this study. Material support was obtained from the Eritrean Ministry of Health.

Supporting Documents

The dataset supporting the conclusions of this article are available from the corresponding author on reasonable request.

Ethics approval and consent to participate

Ethical approval for the study and experimental protocols used was obtained from Eritrean Ministry of Health (MOH) research ethical committee. Informed consent was obtained from all participants. During the study, strict adherence to approved laboratory protocols was observed.

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Figure 1. Prevalence of dyslipidemia within specific age strata

Figure 2A. Framingham risk score

Figure 2B: Relationship between specific lipid abnormalities and Framingham risk categories

Table 1. Demographic characteristics, patient history, anthropometry, and clinical measurements at inclusion in the study

Variables	Male, n(%)	Female, n(%)	P-value (χ^2) or t-test	Total n (%)
Age (years)				
≤40	27 (32.2)	57 (67.9)	0.132(5.607)	84 (21.8)
40-50	43 (33.6)	85 (66.4)		128 (33.2)
51-59	28 (36.8)	48 (63.2)		76 (19.7)
≥60	46 (46.9)	52 (21.5)		98 (25.4)
Educational level				
No formal education/elementary	16 (20.8)	61 (79.2)	<0.001(41.46)	77 (19.9)
Junior	27 (34.2)	52 (65.8)		79 (20.5)

High school	47 (31.5)	102 (68.5)		149 (38.6)
Higher Education	54 (66.7)	27 (33.3)		81(21.0)
Marital Status				
Single	12 (37.5)	20 (62.5)		32 (8.3)
Married	124 (41.1)	178 (58.9)	<0.001(12.50)	302 (78.2)
Divorced	2 (15.4)	11 (84.6)		13 (3.4)
Widowed	6 (15.4)	33 (84.6)		39 (10.1)
Occupation				
Unemployed	24 (11.9)	178 (88.1)		202 (52.33)
Self-employed	40 (72.7)	15 (27.3)		55 (14.24)
Government employee	65 (45.1)	36 (14.9)	<0.001(162.46)	101 (26.16)
Privately employed	15 (53.6)	13 (46.4)		28 (7.25)
Smoking history				
Yes	11(84.6)	2(015.41)	0.001(12.87)	13(3.4)
No	133(35.7)	240(64.3)		373(96.6)
Alcohol Consumption				
Yes	101(40.7)	147(59.3)	0.079(3.50)	248(64.2)
No	43(31.2)	95(68.8)		138(35.8)
Previous Diagnosis of hypertension				
Yes	25(36.2)	44(63.8)	0.041(0.891)	69(17.9)
No	119(37.5)	198(62.5)		317(82.1)
BP (mmHg)				
SBP>130	48(39.3)	74(60.7)	0.317(0.574)	122(31.6)
DBP> 85	45(42.5)	61(57.5)	0.238(1.66)	106(27.5)
Elevated BP (>140/90)	35(37.2)	59(62.8)	0.544(0.000)	94(24.4)
WC (abnormal)	67(24.2)	210(75.80)	< 0.001(72.17)	277(71.8)
WHR (≥ 0.95 (men)/≥0.80 (women))				
WHtR (>0.50)	115(79.9)	218(90.1%)	0.006(7.96)	0.57±0.7
BMI (kg/m2)				24.82±4.01
<18.5	4(23.5)	13(76.5)		17(4.4)
18.5-24.9	91(46.2)	106(53.8)	< 0.001(19.33)	197(51.0)
25-29.9	43(33.6)	85(66.4)		128(33.2)
>30	6(13.6)	38(86.4)		44(11.4)
FPG (mg/dl)				96.17±22.96
Normal < 100	106(36.7)	183(63.3)	0.599(1.025)	289(75.3)
IFP (≥100-125)	31(41.3)	44(58.7)		75(19.5)
DM (>125)	6(30.0)	14(70.0)		20(5.2)

P values (2 tailed): Frequencies of specific demographic and clinical variables between males and females and associated Chi square/ Fishers exact test values or student t-test values. Abbreviation - BP: Blood pressure; SBP: systolic blood pressure; DBP: diastolic blood pressure; WC: waist circumference; WHR: Waist/Hip ratio; WHR: Waist/height Ratio; BMI: Body Mass Index; FPG: Fasting Plasma Glucose.

Table 2. Relationship between specific demographic characteristics, anthropometry, clinical measurements and specific lipid markers

Variables	TC (Mean $\pm$ SD)	TG (Mean $\pm$ SD)	HDL-C (Mean $\pm$ SD)	LDL-C (Mean $\pm$ SD)	Non-HDL-C (Mean $\pm$ SD)	TC/HDL-C (Mean $\pm$ SD)
Gender						
Male	197.39 $\pm$ 37.96	153.5 $\pm$ 62.59	41.5 $\pm$ 8.09	125.35 $\pm$ 31.56	155.89 $\pm$ 34.62	4.84 $\pm$ 0.91
Female	209.9 $\pm$ 49.32	139 $\pm$ 59.9	47.53 $\pm$ 9.7	133.98 $\pm$ 38.31	162.79 $\pm$ 45.83	4.50 $\pm$ 1.00
p-value for difference	0.009	0.026	<0.001	0.02	0.119	0.001
Age (years)						
<40 years	191.58 $\pm$ 36.2	131 $\pm$ 57.97	44.8 $\pm$ 7.37	120.46 $\pm$ 29.9	146.77 $\pm$ 34.40	4.34 $\pm$ 0.87
40-50 years	205.89 $\pm$ 49.6	146.48 $\pm$ 67.0	44.95 $\pm$ 10.18	130.12 $\pm$ 35.6	161.32 $\pm$ 45.04	4.67 $\pm$ 0.95
51-60 years	213.9 $\pm$ 40.67	154.7 $\pm$ 60.58	45.25 $\pm$ 9.9	137.7 $\pm$ 33.17	168.67 $\pm$ 37.26	4.86 $\pm$ 1.12

>60 years	209.3±49.5	145.3±54.9	46.1±10.28	135±41.9	163.74±45.41	4.63±0.98
p-value for difference	0.01	0.100	0.760	0.011	0.006	0.009
Educational Level						
No formal education	210.48±48.54	136.1±40.14	46.5±10.9	137±45.2	165.55±46.75	4.62±1.04
Elementary	198±46.9	140.2±58.1	46.4±9.8	124.4±37.36	151.59±43.06	4.34±0.93
Junior	211±49.1	148.6±62.5	45.9±8.8	132.68±31.78	165.13±45.49	4.67±0.97
Secondary	208.6±44.0	145.41±63.9	45.7±10.0	134.4±37.1	162.95±40.21	4.68±1.03
Tertiary	195±42.6	144.2 ±64.2	42.7±8.37	122.94±32.5	152.40±38.57	4.64±0.91
p-value for difference	0.100	0.88	0.100	0.099	0.142	0.359
Marital status						
Married	204.81±44.5	145.95±63.0	44.58±9.37	130.5±33.8	160.38±40.69	4.68±0.98
Single	201.8±53.7	137.6±51.9	46.2±10	128.1±45	155.56±47.39	4.40±0.82
Divorced	195.8±34.8	129.1±64.5	47.6±13.15	122.4±32.69	148.23±32.24	4.33±1.18
Widowed)	214.46±51	144.25±53.3	49.1±8.8	137.38±45.9	166.74±50.57	4.44±1.05
p-value for difference	0.500	0.700	0.03	0.54	0.497	0.147
Alcohol Consumption						
No	196.6±49.4	135.02±56.64	44.05±9.65	133.67±35.04	152.59±45.98	4.56±1.12
Yes	210.0±42.97	149.60±63.14	45.97±9.51	125.7±37.63	162.46±39.21	4.66±0.90
p-value for difference	0.006	0.023	0.061	0.042	0.011	0.401
Waist Circumference (cm)						
Normal	191.79±39.7	138.28±61	43±8.88	122.19±33.9	148.77±37.64	4.57±1.06
Increased*	210.5±46.9	146.9±61.29	46.1±9.7	134.15±36.48	164.72±42.95	4.65±0.95
p-value for difference	<0.001	0.200	0.004	0.003	0.001	0.524
BMI (kg/m²)						
<18.5	186.6±42	95.6±39.7	47.88±10.9	119±30.45	138.76±37.78	3.99±0.95
18.5-24.9	205.3±46.5	140.8±59.8	45.3±9.09	131.7±36.17	160.28±44.09	4.63±1.08
25-29.9	206.3±47	154.36±64.7	44.89±10.7	129.9±37.44	161.82±40.81	4.68±0.84
>30	208.8±39	149.5±54.7	45.29±7.79	133.6±34.38	163.55±36.69	4.68±0.88
p-value for difference	0.369	0.002	0.69	0.51	0.181	0.055
WHR						
Normal	195.3±45.12	128.4±53.1	46.1±10.59	124.9±37.9	149.24±40.54	4.34±1.02
Abnormal	207.3±45.69	147.9±62.4	45.1±9.37	132±35.7	162.57±42.10	4.68±0.97
p-value for difference	0.049	0.018	0.430	0.140	0.016	0.012
Fasting plasma glucose (mg/dL)						
<100	203.2±45.9	140.35±61.15	45.99±9.4	128.82±34.8	157.37±42.65	4.49±0.92
100-124.9	210.56±45.3	152.3±57.5	43.48±10.27	136.35±39.05	167.57±39.67	4.96±1.07
>125	214.95±44.3	173.35±68.4	41.6±8.32	138.68±42.17	173.35±40.45	5.25±1.01
p-value for difference	0.290	0.03	0.027	0.170	0.063	0.001
Total	205.24±45.77	144.5±61.26	45.28±9.60	130.77±36.15	160.22±42.09	4.62±0.98

Data are mean (standard deviation) unless otherwise indicated

Abbreviations: BMI body mass index, HDL-C high-density lipoprotein cholesterol, LDL-C low-density lipoprotein cholesterol, TC Total cholesterol, WHR Waist to hip ratio, TG triglyceride, SBP: Systolic Blood Pressure; DBP: Diastolic blood pressure; FPG: Fasting plasma glucose.

P values (2 tailed) for student t- test: Comparisons of the mean values of specific lipid markers across specific demographic, anthropomorphic, and clinical variables

Table 3. Gender comparisons and NCEP ATP III risk categories

Variable	Male n (%)	Female n (%)	P-value (χ^2) or t-test	Total n (%)
TC	197.40±37.96	209.9±49.32	0.005	205.24±45.77
Optimal	75(38.7)	119(61.3)	0.027(7.24)	194(50.3)
Borderline	53(42.7)	71(57.3)		124(32.1)
High Risk	16(23.5)	52(76.5)		68(17.6)
Non-HDL-C	155.89±34.62	162.79±45.83	0.029	160.22±42.09
Optimal	34(41.0)	49(59.0)	0.039(10.10)	83(21.5)
Near-Optimal	46(37.4)	77(62.6)		123(31.9)
Borderline High	45(42.9)	60(57.1)		105(27.2)

High	16(33.3)	32(66.7)		48(12.4)
Very High	3(11.1)	32(88.9)		27(7.0)
TG	153.5±62.60	139.1±59.92	0.095	144.50±61.26
Normal	79(33.1)	160(66.9)		239(61.9)
Borderline High	37(44.0)	47(56.0)	0.088(4.85)	84(21.8)
High	28(44.4)	35(55.6)		63(16.3)
LDL-C	125.35±31.56	133.98±38.31	0.018	130.77±35.15
Optimal	43(50.6)	42(49.4)		85(22.0)
Near-Optimal	38(30.2)	88(69.8)		126(32.6)
Borderline High	44(44.4)	55(55.6)	0.003(16.31)	99(25.6)
High	14(25.9)	40(74.1)		54(14.0)
Very High	5(22.7)	17(77.3)		22(5.7)
HDL-C	41.50±8.10	47.54±9.72	0.001	45.28±9.60
Optimal	5(15.2)	28(84.8)		33(8.5)
Borderline	75(52.8)	67(47.2)	0.001(25.92)	142(36.8)
High Risk	64(30.3)	147(69.7)		211(54.7)

Table 4: Mixed Dyslipidemia

Lipid Abnormality	Male N (%)	Female N (%)	Difference %	Total Frequency N (%)
No Lipid abnormality	22(45.8)	26(54.2)	-8.4	48(12.6)
Isolated dyslipidemia				
One abnormality				
TC	1(12.5)	7(87.50)	-75.0	8(2.1)
TG	4(80.1)	1(20.0)	60.1	5(1.3)
HDL-C	28(31.1)	62(68.9)	-37.8	90(23.6)
LDL-C	1(33.3)	2(66.7)	-33.4	3(0.3)
Total				106(27.3)
Mixed Dyslipidemia				
Two abnormalities				
TG + Low-HDL-C	18(46.2)	21(53.8)	-7.6	39(10.2)
LDL + Low-HL-C	1(20.0)	4(80.0)	-60.0	5(1.3)
TC+TG	7(63.6)	4(36.4)	+27.2	12(3.14)
TC+LDL-C	19(37.3)	32(62.7)	-25.4	51(13.4)
Total				107(28.04)
Three Abnormalities				
TG+TC+HDL-C	1(12.5)	7(87.5)	-75.0	8(2.1)
TC+TG+LDL	30(6)	50(94)	-88.0	80(20.9)
Total				88(23.0)
Four Abnormalities				
TG+TC+HDL-C+LDL-C	10(30.3)	23(62.8)	-32.5	33(8.6)
Dyslipidemia	142(37.2)	240(62.8)	-25.6	334(87.4)

Abbreviation: TG: Triacylglycerol; TC: Total Cholesterol; HDL-C; High-Density Lipoprotein Cholesterol; LDL-C; Low-Density Lipoprotein Cholesterol.

Table 5. Association between LDL, TC, TG, HDL, Non-HDL and TG/GDL ratio with key risk factors: results from logistic models

Stratification Variables	OR of Non-HDL ≥ 130 mg/dL		OR of TG ≥ 150 mg/dL		OR of TC ≥ 200 mg/dL	
	Crude OR (95% CI)	Adjusted OR (95% CI)	Crude OR (95% CI)	Adjusted OR (95% CI)	Crude OR (95% CI)	Adjusted OR (95% CI)
Age (years)	1.01(0.985-1.034)		0.99(0.97-1.01)		1.01(0.99-1.03)	
Sex						
Male	1		1	1	1	
Female	0.46(0.23-0.92)		1.61(0.91-2.88)	1.71(1.10-2.65)	0.81(0.46-1.42)	
Education						
> High School	1		1		1	
< High School	1.13(0.625-2.04)		1.03(0.63-1.68)		0.84(0.52-1.36)	
Employment Status						
Unemployed	1		1		1	
Employed	1.77(0.86-3.65)		1.40(0.77-2.54)		0.80(0.45-1.43)	
Self-employed	1.55(0.62-3.90)		1.00(0.40-2.10)		0.77(0.38-1.58)	
Alcohol Consumption						
No	1	1	1	1	1	1
Yes	2.22(1.30-3.80)	2.24(1.34-3.74)	1.73(1.07-2.28)	1.76(1.11-2.79)	2.05(1.30-3.23)	2.04(1.33-3.15)
Marital Status						
Not Married	1		1		1	
Married	1.49(0.79-2.82)		0.53(0.26-1.06)		1.38(0.80-2.37)	
Family History of DM						
No	1		1	1	1	
Yes	0.83(0.39-1.77)		0.49(0.23-1.01)	0.53(0.262-1.06)	0.713(0.37-1.37)	
WC (cm)	1.06(1.02-1.09)	1.04(1.02-1.07)	1.04(1.02-1.06)	1.04(1.02-1.06)	1.01(0.98-1.04)	
BMI (kg/m²)						
<25	1		1		1	
>25	1.37(0.70-2.69)		0.89(0.51-1.55)		1.25(0.72-2.16)	
Hypertension						
No	1		1		1	1
Yes	1.56(0.685-3.56)		0.88(0.46-1.66)		1.54(0.83-2.87)	2.19(1.35-3.53)
DBP (mmHg)	1.00(0.961-1.04)		1.02(0.99-1.06)		1.03(0.99-1.06)	
SBP (mmHg)	0.995(0.98-1.02)		1.00(0.99-1.02)		1.00(0.986-1.02)	
FPG	1.02(0.998-1.04)	1.02(1.00-1.04)	1.01(0.998-1.02)	1.01(1.00-1.02)	1.01(0.996-1.02)	1.01(1.00-1.02)

Abbreviation: TG: Triacylglycerol; TC: Total Cholesterol; HDL-C; High-Density Lipoprotein Cholesterol; LDL-C; Low-Density Lipoprotein Cholesterol.

Table 6. Association between LDL, TC, TG, HDL, Non-HDL and TG/GDL ratio with key risk factors: result from logistic models

Stratification Variables	OR of Low HDL mg/dL		OR of LDL $\geq$ 130 mg/dL		OR of TC/HDL > 5		Dyslipidemia	
	Crude OR (95% CI)	Adjusted OR (95% CI)	Crude OR (95% CI)	Adjusted OR (95% CI)	Crude OR (95% CI)	Adjusted OR (95% CI)	Crude OR 95% (CI)	Adjusted OR 95% (CI)
Age (years)	0.99(0.67-1.01)	0.98(0.97-1.00)	1.01(0.99-1.03)		1.00(0.97-1.02)		1.01(0.98-1.04)	
Sex								
Female	1		1	1	1	1	1	
Male	0.824(0.47-1.44)		0.696(0.39-1.24)	0.68(0.44-1.08)	1.86(0.99-3.50)	1.84(1.00-3.38)	0.76(0.35-1.65)	
Education								
>High School	1		1		1		1	
<High School	1.14(0.70-1.85)		0.76(0.47-1.23)		0.74(0.44-1.26)		0.89(0.42-1.86)	
Employment Status								
Unemployed	1	1	1		1		1	1
Employed	0.58(0.32-1.03)	0.51(0.32-0.83)	0.85(0.47-1.52)		0.714(0.37-1.37)		0.52(0.22-1.23)	0.48(0.24-0.97)
Self-employed	0.61(0.30-1.25)	0.52(0.27-0.975)	0.79(0.38-1.63)		0.765(0.35-1.68)		0.47(0.17-1.30)	0.41(0.17-1.00)
Alcohol Consumption								
No	1	1	1	1	1		1	
Yes	0.42(0.267-0.68)	0.419(0.265-0.66)	1.83(1.16-2.90)	1.80(1.16-2.81)	1.17(0.711-1.93)		0.96(0.48-1.92)	
Marital Status								
Not Married	1		1	1	1	1	1	1
Married	1.32(0.77-2.26)		1.64(0.94-2.87)	1.56(0.92-2.65)	1.90(0.97-3.740)	1.84(1.00-3.38)	2.40(1.18-4.89)	2.35(1.19-4.66)
Family History of DM								
No	1.45(0.73-2.88)		1		1		1	
Yes			0.69(0.354-1.34)		1.13(0.56-2.27)		1.41(0.50-3.97)	
WC (cm)	1.00(0.97-1.02)		1.02(0.997-1.05)	1.03(1.00-1.05)	1.04(1.00-1.07)	1.03(1.00-1.05)	0.98(0.93-1.03)	
BMI (kg/m²)							1.09(0.95-1.26)	
<25	1	1	1	1	1			
>25	0.63(0.36-1.10)	0.65(0.42-1.02)	1.72(0.99-3.01)	1.70(0.99-2.92)	1.27(0.69-2.34)			
Hypertension								
No	1		1	1	1	1	1	
Yes	0.78(0.42-1.47)		1.74(0.933-3.25)	2.06(1.26-3.37)	1.60(0.83-3.10)	1.72(1.03-2.88)	1.52(0.54-4.29)	
DBP (mmHg)	1.01(0.98-1.05)		1.01(0.91-1.04)		1.03(0.99-1.07)		1.05(0.99-1.11)	1.04(1.00-1.09)
SBP (mmHg)	0.99(0.97-1.01)	0.99(0.98-1.00)	1.01(0.99-1.02)		1.00(0.98-1.01)		0.89(0.42-1.86)	
FPG (mg/dL)	1.98(1.16-3.36)	2.10(1.25-3.52)	1.00(0.99-1.01)		1.01(1.00-1.023)	1.012(1.00-1.022)	1.02(0.99-1.05)	1.02(1.00-1.05)

Abbreviation: TG: Triacylglycerol; TC: Total Cholesterol; HDL-C; High-Density Lipoprotein Cholesterol; LDL-C; Low-Density Lipoprotein Cholesterol

Table 7. Relationship between FRS and selected variables

Variables		Low (95% CI)	Moderate (95% CI)	High (95% CI)	P - value
Age (years)		46.41 ± 8.9	61.81 ± 11.5	72.86 ± 9.2	< 0.001
WC (cm)	Abnormal - N (%)	128(46.2)	108(39.0)	41(14.8)	0.127(4.12)
	WC (Mean±SD)	93.0±11.5	93.31±11.42	95.8±8.6	0.229
BMI (kg/m²)	>25 Kg/m2	81(47.1)	71(41.3)	20(11.6)	0.109(4.43)
	Mean±SD	25.19±4.44	24.76±3.93	23.96±3.35	0.131
FPG (mg/dL)	> 100 mg/dl	28(29.8)	47(50)	19(20.2)	0.011(8.98)
	FPG (Mean±SD)	93.13±23.38	97.17±21.20	102.02±25.27	0.029
Educational Level					
No Formal Education		15(45.5)	9(27.3)	9(27.3)	0.005(18.6)
Primary		84(68.3)	26(21.1)	13(10.6)	
Secondary		116(77.9)	24(16.1)	9(6)	
Higher Education		57(70.4)	13(16)	11(13.6)	
Marital Status					
Married		219(72.5)	54(17.9)	29(9.6)	<0.001(36.3)
Single		40(88.9)	4(8.9)	1(2.2)	
Divorced/Widowed		13(33.3)	14(35.9)	12(30.8)	

Abbreviation: WC: Waist circumference, BMI: Body mass index; FPG: Fasting plasma glucose.

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