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ORIGINAL RESEARCH article

The burden of dyslipidaemia in an urban Libyan population: Epidemiological assessment and a review of modern therapy

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Abstract: Dyslipidemia is a major modifiable risk factor for cardiovascular disease, yet data from Libyan populations remain limited. This study aims to investigate the prevalence, patterns, and demographic determinants of dyslipidemia among residents of Zawia City, Libya, and to provide a concise review of lipid-lowering therapies, including emerging ones. It is a cross-sectional study of 508 individuals of all ages and both sexes registered at many clinical laboratory centers across Zawia City for lipid profile testing from January 1, 2026, to May 31, 2026. Dyslipidemia was defined according to the National Cholesterol Education Program (NCEP) and ATPIII criteria. Statistical analysis was performed on all lipid parameters and expressed as mean ± SD, frequency, and percentage. The prevalence of all dyslipidemia patterns was assessed across age and gender groups. The findings showed that 81.6% of participants had at least one form of dyslipidemia. High LDL-C was the most common lipid abnormality (61.6%), followed by hypertriglyceridemia (36.2%), low HDL-C (36.0%), and hypercholesterolemia (27.8%). Clear gender differences were observed: Females had significantly higher rates of hypercholesterolemia (30.6%/24.3% for female/male, $p < 0.05$) and high LDL-C (65.1/57.4% for female/male, $p < 0.01$). By contrast, males exhibited significantly higher rates of low HDL-C (54.8/20.5% for male/female, $p < 0.001$). The high-risk ratio was twofold higher in males than in females (43.0/19.8% for male/female, $p < 0.001$). Age-specific analysis showed that all lipid abnormalities peaked in the 41–50-year age group, where the prevalence of hypercholesterolemia, hypertriglyceridemia, high LDL-C, and low HDL-C (36.4%, 45.7%,74.3%, and 44.3%, respectively) was greatest. These results demonstrate that dyslipidemia is highly prevalent among the population in Zawia City, with LDL-C abnormalities predominating and midlife individuals bearing the greatest burden. The presence of gender-specific patterns underscores the need for targeted prevention and control strategies. Routine screening and tailored interventions are urgently required to reduce cardiovascular disease risk in the Libyan population.

Introduction

Dyslipidemia, characterized by abnormal blood lipid profiles, is a significant risk factor for the development of cardiovascular disease (CVD), which is the leading cause of mortality worldwide [1]. This condition encompasses elevated total cholesterol, triglycerides, and low-density lipoprotein cholesterol (LDL-C), as well as reduced high-density lipoprotein cholesterol (HDL-C), and can manifest individually or concurrently [2]. Approximately 39.0% of the global adult population experiences elevated blood cholesterol levels, with a

higher prevalence observed among women than men [3]. Furthermore, elevated LDL-C contributed to 4.32 million deaths in 2017, accounting for 7.7% of total mortality that year [4].

The etiology of dyslipidemia is complex and multifactorial, as genetic predispositions, dietary habits, and lifestyle behaviors such as physical inactivity, stress, and smoking can all contribute to the development and progression of this condition [5-7]. Additionally, underlying metabolic processes, including insulin resistance and thyroid dysfunction, can disrupt the body's ability to maintain normal lipid profiles, leading to the manifestation of dyslipidemia [8-10]. Dyslipidemia can lead to severe complications, such as atherosclerosis, which can restrict blood flow and increase the risk of coronary artery disease, heart attack, and stroke [11, 12]. Elevated triglycerides can also increase the risk of acute pancreatitis, and dyslipidemia can contribute to peripheral artery disease and the development of xanthomas [7, 13]. Managing dyslipidemia through lifestyle changes and medication is crucial to mitigate these risks.

Determining the prevalence and characteristics of dyslipidemia in a population is essential for designing targeted, evidence-based public health strategies to address this modifiable cardiovascular risk factor and improve population health [1, 14, 15]. In this context, this study aims to identify the overall prevalence of dyslipidemia and its various types among residents of Zawia city; investigate dyslipidemia patterns (hypercholesterolemia, hypertriglyceridemia, high LDL-C, and low HDL-C) in relation to demographic characteristics; identify which age-gender subpopulations are most affected by dyslipidemia; identify the high-risk group for developing cardiovascular diseases by estimating the cholesterol/HDL-C ratio; and provide a brief review of lipid-lowering therapies, highlighting novel agents. By providing locally relevant empirical data, this study seeks to inform tailored screening, prevention, and intervention strategies aimed at reducing cardiovascular risk within the Zawia community. Additionally, the findings may serve as a reference for similar urban Libyan settings undergoing rapid behavioral and epidemiologic transitions, especially given that Libyan studies on this burden of non-communicable disease (NCD) are limited.

Materials and methods

Study design and setting: This research was conducted as a cross-sectional observational study to evaluate the distribution of lipid profile abnormalities among residents of Zawia City, Libya. The study aimed to assess the prevalence of dyslipidemia and its association with demographic variables such as age and gender. Data were collected over five months, from January 1, 2026, to May 31, 2026. Participants were recruited from multiple clinical laboratories across the city, ensuring representation from a wide cross-section of the local population. These laboratories were equipped with standardized testing facilities and followed uniform protocols for biochemical analysis. The study was conducted in collaboration with these centers, which served as the clinical and institutional settings for participant recruitment and sample processing.

Participants: A total of 508 individuals were included in the study. The target population comprised residents (aged 10 to 90 years) who had undergone lipid profile testing during the study period, regardless of their medical conditions. There were no specific exclusion criteria, so the participant pool included apparently healthy individuals and those with known metabolic or cardiovascular conditions. Participants were not actively recruited; instead, individuals who sought laboratory lipid profiling for routine check-ups or medical referrals during the study period were consecutively included. Basic demographic characteristics, such as age and gender, were recorded, enabling further subgroup analysis. The diverse composition of the participant group allowed for a more accurate assessment of dyslipidemia across various population segments within Zawia City.

Data collection and measurements: Lipid profile data were obtained from venous blood samples collected in red-top tubes, following standard phlebotomy protocols. All samples were processed within one hour of collection to preserve sample integrity and ensure accuracy. Analyses were performed on the Atellica Solution,

an integrated, fully automated biochemistry and immunoassay analyzer from Siemens. Before testing, quality control procedures were strictly followed, with quality control sera run using BIO-RAD standards. Measured parameters included total cholesterol, triglycerides, HDL-C, and LDL-C. For samples with triglyceride levels below 400 mg/dL, LDL-C was calculated using the Friedewald formula. For samples with triglyceride levels of 400 mg/dL or higher, direct LDL-C measurement was performed on the analyzer. All results were digitally recorded and anonymized to maintain confidentiality.

The classification of dyslipidemia was based on the guidelines established by the National Cholesterol Education Program (NCEP) and the Adult Treatment Panel III (ATP III) [16, 17]. According to these standards, the following thresholds were used to identify lipid abnormalities: Hypercholesterolemia: Total cholesterol ≥ 200 mg/dL, Hypertriglyceridemia: Triglycerides ≥ 150 mg/dL, Low HDL-C: HDL-C ≤ 40 mg/dL, High LDL-C: LDL-C ≥ 100 mg/dL, and Elevated cholesterol/HDL-C Ratio: ratio ≥ 4.5. These clinical cutoffs were selected for their widespread acceptance in epidemiological and clinical practice settings. They also provide a standardized framework for comparing dyslipidemia prevalence across populations and studies.

Ethical issues: This study received ethical approval from the Scientific Research Ethics Committee of the Libyan Medical Research Center/Zawia-Libya (registered under reference number NBC:018.H.26.132) before the research began.

Statistical analysis: All data were analyzed using the Statistical Package for the Social Sciences (SPSS), version 23.0. Descriptive statistics were used to summarize the demographic and biochemical data, including frequency, percentage, mean, and standard deviation. Categorical variables, such as gender and the presence or absence of specific lipid abnormalities, were compared using the chi-squared (χ^2) test. This allowed for the determination of statistically significant differences in dyslipidemia prevalence between male and female participants. A p-value of less than 0.05 was considered statistically significant for all comparisons. Continuous variables such as lipid levels were expressed as means with standard deviations, and any outliers or inconsistencies were reviewed for possible data entry or analytical errors. The statistical approach provided a clear basis for assessing relationships between lipid parameters and demographic variables.

Results

Participant characteristics: The demographic data in **Table 1** indicate that the 508 study participants had a mean age of 49.8 ± 15.2 years. Of these, 230 (45.3%) were male, with a mean age of 50.6 ± 13.4 years, and 278 (54.7%) were female, with a mean age of 49.2 ± 16.5 years.

Table 1: Demographic characteristics of patients

Gender	Frequency (%)	Age (mean ± SD)
Male	230 (45.3)	50.6 ± 13.4
Female	278 (54.7)	49.2 ± 16.5
Total	**508**	**49.8 ± 15.2**

Lipid profile overview: **Table 2** presents the mean values of blood lipid measurements in the study population. The mean total cholesterol level was 175.3 ± 45.9 mg/dL, the mean triglycerides were 144.8 ± 76.4 mg/dL, the mean LDL-C was 113.7 ± 39.9 mg/dL, and the mean HDL-C was 45.8 ± 11.8 mg/dL.

Table 2: Mean values of different blood lipid types among patients

Blood lipid type	Mean ± SD (mg/dl)
Cholesterol	175.3 ± 45.9
Triglycerides	144.8 ± 76.4
LDL-C	113.7 ± 39.9
HDL-C	45.8 ± 11.8

Lipid profile variation by age groups: **Table 3** illustrates the distribution of patients' lipid profiles across different age groups. The data indicate that mean concentrations of total cholesterol, triglycerides, and LDL-C generally increase with age, reaching their peak values in the 41 - 50 years age group before declining with further increases in age. Specifically, this age group exhibited the highest mean values for cholesterol (184.0 ± 44.9 mg/dl), triglycerides (160.6 ± 86.5 mg/dl), and LDL-C (123.1 ± 39.9 mg/dl). In contrast, mean HDL-C levels remained relatively consistent across all age groups.

Table 3: Distribution of patients' lipid profiles according to age groups

Age (years)	Cholesterol (mg/dl) Mean ± SD	Triglycerides (mg/dl) Mean ± SD	LDL-C (mg/dl) Mean ± SD	HDL-C (mg/dl) Mean ± SD
≤ 20	145.0 ± 28.5	96.9 ± 62.6	93.3 ± 23.9	45.5 ± 9.5
21 - 30	162.9 ± 24.7	107.5 ± 47.1	104.6 ± 27.5	44.5 ± 11.4
31 - 40	178.3 ± 36.2	150.1 ± 82.6	117.4 ± 29.3	45.1 ± 11.5
41 - 50	184.0 ± 44.9	160.6 ± 86.5	123.1 ± 39.9	44.0 ± 10.4
51 - 60	180.9 ± 48.6	150.9 ± 76.8	120.3 ± 41.7	48.1 ± 13.2
61 - 70	171.2 ± 50.0	146.7 ± 65.7	106.4 ± 42.3	47.7 ± 12.8
> 70	148.9 ± 49.2	117.3 ± 48.2	89.4 ± 45.7	44.2 ± 11.3

Prevalence of dyslipidemia and its patterns: The overall prevalence of dyslipidemia, encompassing at least one lipid abnormality, was 81.6% among 508 participants. **Table 4** shows the prevalence of various dyslipidemia patterns among the study patients. Hypercholesterolemia was observed in 141 (27.8%) patients, and its prevalence in female patients (30.6%) was significantly higher than that of male patients (24.3%) ($p < 0.05$). Hypertriglyceridemia was present in 184 (36.2%) patients, comprising 87 (37.8%) males and 97 (34.9%) females, with no statistically significant difference between genders ($p > 0.05$). High LDL-C was the most common form of dyslipidemia, found in 313 (61.6%) patients, with a significantly higher rate in females (65.1%) than males (57.4%) ($p < 0.01$). On the other hand, low HDL-C was observed in 183 (36.0%) patients, with a prevalence significantly more than double in males (54.8%) compared to females (20.5%) ($p < 0.001$).

Table 4: Prevalence of various patterns of dyslipidemia in the study population

Dyslipidemia type	All patients Frequency (%)	Male patients Frequency (%)	Female patients Frequency (%)	Chi square	P value
Hypercholesterolemia (Cholesterol ≥ 200 mg/dl)	141 (27.8)	56 (24.3)	85 (30.6)	5.965	**< 0.05**
Hypertriglyceridemia (Triglycerides ≥ 150 mg/dl)	184 (36.2)	87 (37.8)	97 (34.9)	0.543	> 0.05
High LDL-C (LDL-C ≥ 100 mg/dl)	313 (61.6)	132 (57.4)	181 (65.1)	7.671	**< 0.01**
Low HDL-C (HDL-C ≤ 40 mg/dl)	183 (36.0)	126 (54.8)	57 (20.5)	26.016	**< 0.001**

High-risk ratio analysis: The cholesterol-to-HDL-C ratio, a clinically important index of cardiovascular risk, was calculated for all participants. A ratio equal to or greater than 4.5 was considered high risk. A high-risk ratio was observed in 154 (30.3%) patients, of whom 99 (43.0%) were males, and 55 (19.8%) were females. Therefore, the prevalence of the high-risk ratio was approximately twice as high among male patients compared to female patients, and the difference was statistically significant ($p < 0.001$), as shown in **Table 5**. This finding highlights a substantially higher cardiovascular risk burden among the male participants compared to their female counterparts, driven mainly by lower HDL-C levels among men.

Table 5: Prevalence of high-risk ratio in the study patients

Groups	High risk ratio (≥ 4.5) Frequency (%)	Chi square	P value
In all patients	154 (30.3)		
In male patients	99 (43.0)	12.571	**< 0.001**
In female patients	55 (19.8)		

Age-based distribution of dyslipidemia patterns: **Table 6** presents the age-based distribution of dyslipidemia types in the study population. The data indicate that the frequencies of hypercholesterolemia, hypertriglyceridemia, elevated LDL-C, low HDL-C, and high-risk ratio generally increased with age, peaking in the 41-50-year-old cohort before declining with further increases in age. In other words, this age group exhibited the highest rates for hypercholesterolemia (36.4%), hypertriglyceridemia (45.7%), elevated LDL-C (74.3%), low HDL-C (44.3%), and high-risk ratio (42.1%). This suggests that individuals in the 41-50 age range are the most vulnerable to these dyslipidemia conditions. Notably, elevated LDL-C remained the most common form of dyslipidemia in every age group, underscoring its dominant contribution to cardiovascular risk in the population.

Table 6: Distribution of dyslipidemia patterns in different age groups of patients

Age (years)	High cholesterol (≥ 200 mg/dl) Frequency (%)	High triglycerides (≥ 150 mg/dl) Frequency (%)	High LDL-C (≥ 100 mg/dl) Frequency (%)	Low HDL-C (≤ 40 mg/dl) Frequency (%)	High Risk ratio (≥ 4.5) Frequency (%)
≤ 20	01 (3.7)	04 (14.8)	08 (29.6)	08 (29.6)	02 (7.4)
21 - 30	02 (6.5)	03 (9.7)	18 (58.1)	11 (35.5)	08 (25.8)
31 - 40	13 (20.9)	20 (32.3)	45 (72.6)	26 (41.9)	20 (32.3)
41 - 50	51 (36.4)	64 (45.7)	104 (74.3)	62 (44.3)	59 (42.1)
51 - 60	42 (35.0)	49 (40.8)	81 (67.5)	38 (31.7)	39 (32.5)
61 - 70	25 (27.2)	36 (39.1)	41 (44.6)	27 (29.3)	19 (20.7)
> 70	07 (19.4)	08 (22.2)	16 (44.4)	11 (30.6)	07 (19.4)

Discussion

In this cross-sectional study, 508 participants were enrolled and tested for lipid profiles at different clinical laboratory centers in Zawia City. The mean age of patients was 49.8 years (SD: 15.2), and 54.7% were females and 45.3% were males. The current data reveal a clear age-related trend in lipid profiles, with mean total cholesterol, triglycerides, and LDL-C increasing with age, peaking in the 41 - 50 age group before declining (for this age group, the mean ± SD (mg/dl) of total cholesterol, triglycerides, and LDL-C were 184.0 ± 44.9, 160.6 ± 86.5, and 123.1 ± 39.9, respectively). Furthermore, recent research showed that the prevalence of all types of dyslipidemia peaked in this age group, too (the prevalence of hypercholesterolemia, hypertriglyceridemia, high LDL-C, and low HDL-C was 36.4%, 45.7%, 74.3%, and 44.3%, respectively, in the 41-50-year age group). This trend aligns with general physiological changes associated with aging, as lipid metabolism can be influenced by hormonal changes, dietary habits, and physical activity levels, all of which may shift with age [1, 18, 19]. The hormonal shifts that occur with aging can impact how the body processes lipids, resulting in changes in total cholesterol, triglycerides, and LDL-C [19-21]. Additionally, decreased physical activity and poorer dietary choices, such as increased consumption of saturated fats and added sugars, can further exacerbate the lipid profiles observed in older adults [1, 18, 22, 23]. This age-related pattern in lipid profiles and types of dyslipidemia has been observed in other studies, with either similar or different age ranges for peak lipid levels and for the highest prevalence of dyslipidemia compared with the current research. Partial overlap has been documented in China, where the peak prevalence of hypertriglyceridemia was among

middle-aged men (45 - 54 years) and, conversely, the highest rate of elevated LDL-C was observed in the older age group (55 - 64) [22]. Consistently, Hawaldar et al. in India revealed that the highest levels of cholesterol, triglycerides, and LDL-C were in the 41-50 years age group, mirroring the trend reported in this study [24]. On the other hand, Abujbara et al. in Jordan documented the highest prevalence of hypercholesterolemia and hypertriglyceridemia in the older age bracket (50 - 59), whereas the highest rate of increased LDL-C was reported in the 40-49 age group, therefore showing some similarity with the recent research finding [25]. U.S. National Health and Nutrition Examination Survey (NHANES) data indicate that individuals aged 40 to 59 have a higher prevalence of hypercholesterolemia than those under 39 or over 60 years old. This finding is consistent with current research, which also showed the highest prevalence of hypercholesterolemia in the combined 41 - 50 and 51 - 60 age groups. The NHANES study attributed this age-related disparity to several factors, including differences in physical activity levels, dietary habits, and other sociocultural and socioeconomic factors [26].

Furthermore, this study showed a high burden of dyslipidemia among populations in Zawia City, Libya, with over three-fourths (81.6%) of patients included in this study having at least one lipid abnormality. This overall prevalence of dyslipidemia aligns with findings from India (79.0%) [14], Russia (76.1%) [27], and Egypt (76.0%) [28]. However, the burden documented in the current research exceeds that reported in several countries, including China (48.2%) [1], the USA (53.0%) [29], Nigeria (58.1%) [30], and northern Ethiopia (66.7%) [31]. Moreover, the recent finding demonstrated that the high LDL-C (61.6%) was the most common pattern of dyslipidemia, followed by hypertriglyceridemia (36.2%), low HDL-C (36.0%), and hypercholesterolemia (27.8%). Additionally, our data showed that females had significantly higher rates of hypercholesterolemia (30.6% vs. 24.3%) and high LDL-C (65.1% vs. 57.4%) than males. Conversely, the recent study found that males are more than twice as likely to have low HDL-C than females (54.8% vs 20.5%). However, our data showed a higher prevalence of hypertriglyceridemia in males compared to females; the difference was not statistically significant (37.8/34.9% for males/females, $p > 0.05$). The prevalence of elevated LDL-C (61.6%) in this study is higher than the previous finding reported in Nigeria (1.7%) [30], China (5.8%) [1], India (11.8%) [14], USA (27.0%) [29], Iran (35.5%) [32], the United Arab Emirates population (UAE) (38.6%) [33], Turkey (44.5%) [34] and Ethiopia (49.5%) [31]. The findings are almost similar to those reported in Iraq (57.8%) [35], Russia (60.3%) [27], and Ghana (61.0%) [36]. However, this was lower than the findings reported in Jordan (75.9%) [25]. Furthermore, the females in the current study were significantly more affected than males by elevated LDL-C, and this result was in line with studies conducted in India, northern Ethiopia, Iran, and Ghana [14, 31, 32, 36]. However, this contradicts the findings reported in China, Russia, and the UAE, where males were more affected [1, 27, 33].

The prevalence of hypertriglyceridemia (36.2%) in the recent study is higher than the previous finding reported in Italy (19.2%) [37], Oman (20.7%) [38], Nigeria (21.4%) [30], Russia (25.5%) [27], Portugal (26.0%) [39], UAE (29%) [33], Tunisia (29.2%) [40], Malaysia (29.3%) [41], India (29.5%) [14], USA (30.0%) [29], and Turkey (30.4%) [34]. However, it is almost consistent with the prevalence reported in China (33.3%) [1]. It is lower than that documented in Saudi Arabia (40.3%) [42], Iraq (41.6%) [35], Jordan (41.9%) [25], Iran (46.0%) [32], and South Africa (59.3%) [43]. Recent research has found a higher rate of elevated triglycerides in males than females, a finding consistent with that in most other countries [1, 14, 25, 27, 32, 33, 35, 40]. However, the difference was not statistically significant in our cohort, unlike in other studies. This may be due to our limited sample size and/or unique population-specific factors, such as local lifestyle or genetic predispositions, which may attenuate sex-based differences observed elsewhere.

The prevalence of low HDL-C (36.0%) reported in this study aligns with a report from Malaysia (37.2%) [41]. However, it is lower than figures from the UAE (42.5%) [33], Nigeria (42.8%) [30], Iran (43.9%) [32], Iraq (49.9%) [35], Jordan (59.5%) [25], and India (72.3%) [14]. Conversely, it exceeds the rates reported in studies from various countries worldwide, including China (14.5%) [1], Ethiopia (16.5%) [31], Ghana (17.0%) [36],

Russia (18.5%) [27], Turkey (21.1%) [34], Tunisia (21.2%) [40], the USA (23.0%) [29], and Senegal (26.5%) [44]. Notably, the present study revealed that males have more than twice the prevalence of low HDL-C compared to females, with a difference of 34.3 percentage points, highlighting a significantly higher risk among males. While many international studies [1, 35, 40, 44] confirm that males typically have higher rates of low HDL-C than females, consistent with this research, the most comparable data come from Tunisia, where males are approximately three times more likely to be affected by low HDL-C than females [40]. On the other hand, some studies conducted in Russia, Ethiopia, Iran, the UAE, and Ghana [27, 31-33, 36] reported that females are more affected, which contradicts these findings.

Hypercholesterolemia (27.8%) is the least prevalent component of dyslipidemia in the recent study. This finding is comparable to data from Ethiopia (30.8%) [31]. However, it is lower than the prevalence reported in many previous studies, including Turkey (37.5%) [34], Tunisia (40.8%) [40], Iran (41.6%) [32], UAE (42.8%) [33], Jordan (44.3%) [25], Saudi Arabia (54.0%) [42], Russia (58.2%) [27], the United Kingdom (59.0%) [45], and Ghana (60.0%) [36]. Conversely, it exceeds the figures documented in Nigeria (2.6%) [30], India (13.9%) [14], and China (22.4%) [1]. Additionally, the current results showed that hypercholesterolemia is significantly higher in females than in males. While this aligns with many reports from India, Russia, Iran, Ghana, and Tunisia [14, 27, 32, 36, 40], it conflicts with findings from China, Nigeria, and the UAE [1, 30, 33] that show males are more affected by elevated cholesterol. A significant finding of this research was the dramatic reduction in all types of lipid abnormalities observed after the age of 60, with a further considerable decline occurring past 70 years. This was consistent with Jordan's findings [25] and supported by Streja et al. in a chapter about dyslipidemia management in the elderly [46]. One potential explanation for this trend is that many individuals with dyslipidemia may not survive past 60, leading to a diminished prevalence of these conditions in older populations [46].

Another key finding of this study is that 30.3% of patients had a high-risk ratio (Cholesterol-to-HDL-C ratio ≥ 4.5). However, it aligns with a report from Jordan (33.2%) [25]. It is lower than that documented in the UAE (72.3%) [33]. Furthermore, this research demonstrated that the prevalence of an elevated risk ratio was approximately twofold higher among males than females (43.0% vs 19.8%, $p < 0.001$), thereby increasing their risk of developing atherosclerotic cardiovascular disease (ASCVD). This may be explained by the more than twofold higher prevalence of low HDL-C among males compared with females, as shown in the current study. This is almost consistent with a report from Jordan, in which the male risk ratio is twice that of females [25]. Moreover, recent data revealed that the highest rate of elevated risk ratio occurred in the 41-50-year-old cohort (42.1%), which was the most affected by all types of dyslipidemia recorded in this study. Lastly, these variations across studies may stem from lifestyle factors, such as an unhealthy diet and physical inactivity, as well as economic and sociocultural influences, genetic and hormonal factors, preexisting comorbidities, and potential discrepancies in study design, diagnostic criteria (cutoff values), and participant characteristics, including age, gender, and living region (urban, rural, or both).

Study limitations and future research directions: The current study, however, has some limitations. The cross-sectional design precludes inference of causality. Participants were individuals who presented for laboratory testing, which may have introduced selection bias toward those with higher health awareness or pre-existing conditions. Additionally, the absence of data on physical activity, dietary intake, smoking, Body Mass Index, and comorbidities such as diabetes, hypertension, or cardiovascular disease limits assessment of their direct contributions. Moreover, no data on treatment, whether the patient is receiving any lipid-lowering agents or not, can affect data analysis. Future research should adopt longitudinal designs with comprehensive risk factor assessment to better understand causal pathways and evaluate intervention effectiveness. Despite these limitations, this study provides robust baseline data for Zawia City and can inform policy and clinical decision-making in Libya. Given the country's epidemiological transition and rising CVD burden, addressing dyslipidemia through early identification and intervention can avert CVD.

Modern pharmacotherapy for dyslipidemia: A concise review

Hyperlipidemia is a critical, modifiable risk factor for ASCVD; its high burden in the current study population underscores the urgent need for effective management in Libya. While established agents provide a robust therapeutic foundation, the advent of novel, targeted therapies has significantly expanded treatment options, enabling more personalized and effective lipid-lowering strategies. This concise review synthesizes current evidence to present an integrated perspective on foundational and modern pharmacotherapy for hyperlipidemia.

Established antihyperlipidemic agents: The therapeutic foundation: These agents form the cornerstone of lipid management, supported by extensive long-term cardiovascular outcome data.

Statins (HMG-CoA reductase inhibitors): Statins competitively inhibit hepatic HMG-CoA reductase, increasing hepatic LDL receptor (LDLR) expression and enhancing clearance of LDL-C from the circulation [47-49]. They are the first-line therapy for ASCVD because they reduce LDL-C, and high-intensity statin therapy can achieve ≥ 50% LDL-C reduction, resulting in a significant decrease in major adverse cardiovascular events and mortality [48-50]. Common adverse effects of statin therapy include headache, myalgia, dizziness, and gastrointestinal upset. These manifestations are often dose-dependent and may be ameliorated by substituting an alternative statin [51, 52]. More serious adverse reactions include elevated serum transaminase levels, myopathy, and rhabdomyolysis. Statins have also been linked to renal impairment and may confer an increased risk of cardiomyopathy [51, 53]. Contemporary studies indicate a potential association between statin use and the development of type 2 diabetes mellitus [54].

Ezetimibe: Ezetimibe inhibits intestinal cholesterol absorption by blocking the sterol transporter Niemann-Pick C1-Like 1 (NPC1L1) protein in the small intestine [55, 56]. As monotherapy, it lowers LDL-C by 18.0% - 25.0%, and when combined with a statin, it provides an additional LDL-C reduction of up to 70.0% [57]. This combination increases cardiovascular benefits in patients with acute coronary syndrome (ACS) and chronic kidney disease [58, 59].

Fibrates (PPAR-α agonists): Fibrates activate peroxisome proliferator-activated receptor-alpha (PPAR-α), which increases lipoprotein lipase activity. This results in enhanced triglyceride lipolysis and clearance, as well as an increase in HDL-C level [51]. They mainly reduce serum triglycerides by 30.0% - 45.0% and, to a lesser extent, raise HDL-C and lower LDL-C levels [60]. Their main use is to prevent pancreatitis in severe hypertriglyceridemia; however, their impact on significant cardiovascular outcomes is less certain [61, 62].

Niacin (Nicotinic acid): It effectively reduces triglycerides, LDL-C, and total cholesterol levels. Moreover, niacin is the most effective option for increasing HDL-C [63]. However, its side effects-including cutaneous flushing, impaired glucose tolerance, and liver toxicity-have led to its withdrawal from clinical use [60].

Bile acid sequestrants: Agents such as colesevelam bind to bile acids in the intestine, disrupting the enterohepatic circulation. This prompts the liver to convert more cholesterol into new bile acids, reducing hepatic cholesterol stores and upregulating LDLR activity [51, 55, 64]. These agents decrease LDL-C by 15.0 - 30.0% and raise HDL-C by 3.0% - 5.0%, but their use is often limited by gastrointestinal side effects, the potential for triglyceride (TG) elevation, and dosing inconvenience [64, 65].

Omega-3 fatty acids (icosapent ethyl): ω-3 fatty acids are found primarily in marine animals and plant oils, and they include eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA). They act by inhibiting de novo lipogenesis by suppressing the cholesterol regulatory element binding protein gene. Furthermore, they decrease triglyceride levels by boosting fatty acid oxidation and triglyceride breakdown through activation of peroxisome proliferator gene family members [66]. ω-3 fatty acids reduce triglyceride levels by 25.0% - 30.0%; therefore, they are primarily used to treat hypertriglyceridemia [64]. It has been reported that icosapent ethyl (IPE), which contains only EPA, is effective in reducing cardiovascular events [67]. Although ω-3 fatty acids containing EPA and DHA also decrease cardiovascular events, their effects are less pronounced than with IPE [68].

Modern, targeted pharmacotherapy: Expanding the armamentarium: These new therapeutic strategies offer high potency, novel mechanisms, and personalized applications for complex lipid disorders.

PCSK9 (proprotein convertase subtilisin/kexin type 9) inhibitors: PCSK9 is synthesized by the liver and promotes lysosomal degradation of hepatic LDLRs. Consequently, its inhibition increases LDLR availability and enhances the clearance of LDL-C from the blood [69, 70]. Currently, PCSK9 inhibitors mainly encompass the PCSK9 monoclonal antibody and the PCSK9 small interfering RNA (siRNA) [55].

PCSK9 monoclonal antibodies: They mainly include Evolocumab and Alirocumab, which are approved for treating dyslipidemia in many countries [55]. They are administered subcutaneously every 2 - 4 weeks. These agents reduce LDL-C by 45% to 60% regardless of whether statin therapy is co-administered, and have been shown to reduce cardiovascular events (FOURIER, ODYSSEY OUTCOMES trials) [64, 71, 72].

PCSK9 small-interfering RNA: Inclisiran is a long-acting siRNA that suppresses expression of PCSK9 mRNA in hepatocytes, leading to inhibition of PCSK9 synthesis [73]. It is administered subcutaneously twice yearly and provides a sustained reduction in LDL-C of approximately 50.0% [74].

Bempedoic acid (ATP-citrate lyase inhibitor): Bempedoic acid is an oral prodrug that inhibits ATP-citrate lyase, an enzyme upstream of HMG-CoA reductase, the target of statins, in the cholesterol synthesis pathway. Inhibiting cholesterol synthesis upregulates hepatic LDLR, increasing LDL-C uptake and lowering circulating LDL-C levels [75, 76]. It is activated specifically in the liver but not in most peripheral tissues, including skeletal muscle. Therefore, hepatic-specific activation of bempedoic acid reduces muscular side effects compared with statins [77, 78]. Evidence from the CLEAR Wisdom trial shows that adding bempedoic acid to statin therapy significantly lowers LDL-C by 15.1% in patients with ASCVD or heterozygous familial hypercholesterolemia [78]. The CLEAR Outcomes trial revealed that bempedoic acid reduces the risk of major cardiovascular events compared with placebo in statin-intolerant patients at high risk or with established ASCVD [79]. Coadministration of bempedoic acid with ezetimibe yields a more pronounced LDL-C reduction of approximately 38.0% [80]. A meta-analysis of aggregate clinical trial data found that bempedoic acid, when used as monotherapy, lowered LDL-C levels by 24.0% [81]. Moreover, bempedoic acid has a favorable safety profile alongside its effectiveness, making it a valuable alternative to statins in statin-intolerant, high-risk patients [82].

Therapies for severe and genetic dyslipidemias

- *ANGPTL3 inhibitors:* Angiopoietin-like protein 3 is a liver-derived secretory protein that regulates plasma lipid metabolism by inhibiting lipoprotein lipase and endothelial lipase, thereby limiting the hydrolysis of triglycerides and phospholipids [47, 83]. Pharmacological inhibition of ANGPTL3, whether via monoclonal antibodies (Evinacumab), antisense oligonucleotides (Vupanorsen), or siRNAs (Zodasiran), reduces circulating levels of triglycerides, LDL-C, non-HDL-C, and HDL-C, inducing panhypolipoproteinemia [55]. This lipid-lowering effect is mechanistically distinct from that of statins and PCSK9 inhibitors because it operates independently of LDLR function, a property that makes it particularly effective in homozygous familial hypercholesterolemia (HoFH), where LDLR activity is absent or severely impaired [47, 84]. Evinacumab reduces LDL-C by approximately 50.0% in patients with HoFH [85]
- *ApoC3 inhibitors:* Apolipoprotein C-III (ApoC3) inhibitors represent a novel therapeutic class that targets ApoC3, a key regulator of triglyceride metabolism. ApoC3 elevates plasma triglyceride levels by reducing lipoprotein lipase activity, inhibiting clearance of triglyceride-rich lipoproteins from the circulation, and promoting hepatic VLDL secretion into plasma [55, 86]. The primary mechanism of

action of ApoC3 inhibitors involves antisense oligonucleotides or siRNA molecules that bind ApoC3 mRNA, thereby disrupting protein translation and lowering circulating ApoC3 levels. This, in turn, enhances lipoprotein lipase-mediated lipolysis and accelerates plasma triglyceride clearance [55, 86]. Key agents in this class include volanesorsen (the first approved drug for familial chylomicronemia syndrome), olezarsen, and plozasiran, all of which have shown substantial reductions in triglycerides, ranging from 49.0% to 77.0%, in phase II and III clinical trials [55].

- *Apo(a)/Lp(a) inhibitors:* Apolipoprotein(a) [apo(a)] is the unique protein component of Lipoprotein(a) [Lp(a)], an LDL-C variant that represents an independent genetic risk factor for ASCVD and calcific aortic valve stenosis [55]. Novel inhibitors targeting apo(a)/Lp(a) synthesis offer a promising strategy to reduce cardiovascular risk through several mechanisms [55]. Among these are the antisense oligonucleotide pelacarsen, which reduces Lp(a) by up to 80.0% in a dose-dependent manner by inhibiting hepatic apo(a) mRNA [87], and the siRNAs olpasiran and zerlasiran, which lower Lp(a) by > 95% and > 80%, respectively [88, 89]. These therapies are indicated for patients with established CVD and elevated Lp(a) levels who remain at high residual cardiovascular risk despite optimal lipid-lowering therapy, with ongoing phase 3 trials evaluating their impact on cardiovascular outcomes [55].

Gene-editing therapy: VERVE-101 is a first-in-class CRISPR-based gene-editing agent that permanently inactivates the PCSK9 gene, offering a durable, single-infusion strategy for LDL-C reduction. Early-phase clinical data demonstrate substantial efficacy, with mean LDL-C reductions of up to 69.0% at higher doses and a favorable short-term safety profile [90]. While these findings are highly promising, this approach introduces genomic mutations, necessitating rigorous long-term safety surveillance. Furthermore, the durable efficacy of VERVE-101 positions it as a potentially transformative therapeutic option, particularly for young patients with early-stage disease [48].

Conclusion: This study demonstrates that dyslipidemia is highly prevalent among residents of Zawia City, Libya, affecting approximately four out of every five individuals. High LDL-C was the most common lipid abnormality, reported in about two-thirds of the study population, followed by hypertriglyceridemia and low HDL-C. Hypercholesterolemia was the least common lipid abnormality in the current study. Gender-specific patterns were evident, with females more frequently affected by hypercholesterolemia and high LDL-C, whereas males had more than twice the rate of low HDL-C. In addition, the high-risk ratio was twofold higher in men. Age-related analysis revealed that all types of dyslipidemia peaked in the 41 - 50 age group, and the elevated risk ratio was also most prevalent in this age group. Therefore, this age group is the most vulnerable stage for lipid-related metabolic disturbances and for developing CVD. The coexistence of high LDL-C and low HDL-C in this population signals a distinctly atherogenic lipid profile that may substantially elevate the risk of CVD, particularly among men, who also displayed a higher risk ratio. These findings highlight an urgent need for population-level interventions, including routine lipid screening, lifestyle modification programs focusing on diet and physical activity, and risk-based pharmacological management. Targeted strategies addressing age and gender disparities will be essential to mitigate the growing CVD burden in Libya.

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