ABSTRACT

Background and Aims: Attaining guideline-recommended low-density lipoprotein cholesterol (LDL-C) goals (<70 mg/dl or ≥ 50% reduction) with statin therapy remains suboptimal after an acute coronary syndrome (ACS). This study aimed to assess the level of lipid-lowering therapy (LLT) utilization and achievement of LDL-C targets after ACS hospitalization in the United Arab Emirates (UAE).

Methods: A retrospective, observational, longitudinal database analysis of Emirati patients with ACS or stable coronary heart disease was evaluated from January 2015 to June 2018. Patients were divided based on whether or not they were treated with LLT at index hospitalization with ACS. LDL-C target level achievement was assessed according to the 2013 American College of Cardiology/American Heart Association and European Society of Cardiology/European Atherosclerosis Society guidelines.

Results: A total of 3,066 patients (mean age 65.5 ± 14 years) met the inclusion criteria. Overall, 58.1% (n = 1782) of the patients in the cohort were on LLT during the ACS hospitalization. At discharge, the mean LDL-C level was 84.8 ± 39.0 mg/dl, and 28%, 21%, and 9% received high-, moderate-, and low-intensity statins, respectively. At 6 months (n = 2046; 66.7%), 27.7% and 16.7% achieved an LDL-C of <70 mg/dl and 70–100 mg/dl, respectively. The highest level of LDL-C reduction by 50% within 6 months was observed among patients using moderate-intensity statin (37.2%).

Conclusion: A large proportion of Emirati patients were not on LLT after ACS, and the rate of LDL-C target value attainment was extremely poor (27.7%). Optimal statin utilization by closely implementing the guidelines in the UAE is recommended.

Key words: Acute coronary syndrome, cardiovascular diseases, low-density lipoprotein cholesterol goal attainment, real-world evidence, statin therapy, United Arab Emirates

INTRODUCTION

Atherosclerotic cardiovascular disease (ASCVD) is a leading contributor to morbidity and mortality in both developing and developed countries.[1-6] In the United Arab Emirates (UAE), cardiovascular disease (CVD) is the leading cause of death in Abu Dhabi, with 37.1% of fatalities.[6] Acute coronary syndrome (ACS) is a severe and life-threatening clinical manifestation of CVD. It is characterized by a sudden reduction in blood flow to the heart as a result of plaque rupture. Decreasing low-density lipoprotein cholesterol (LDL-C) with statins has been shown to reduce all-cause and CV-related mortality in patients with and without ASCVD.[7-10] Previous studies reported that a 22% relative reduction in major CV events is
achieved for each 1 mmol/L (38.7 mg/dl) reduction in LDL-C level.\textsuperscript{[11-13]}

The benefits of lowering LDL-C with statin therapy, particularly early after an ACS, have been established. Despite the benefits of statin therapy after an ACS event, attaining guideline-recommended LDL-C goals (<70 mg/dl or ≥50% reduction) is still suboptimal.\textsuperscript{[14-18]}

Previous studies showed that less than one-third (28% women vs. 32% men) of the Arabian Gulf patients with ASCVD achieved their LDL-C targets.\textsuperscript{[16,19-21]}

However, little is known regarding the therapeutic management and achievement of guideline-recommended LDL-C goals in the UAE. The present study aimed to assess the real-world evidence of lipid-lowering therapy (LLT) utilization and achievement of LDL-C targets after ACS in the region of Abu Dhabi, UAE.

**PATIENTS AND METHODS**

A retrospective, observational, longitudinal database analysis of Emirati patients with ACS or with stable coronary heart disease (CHD) was conducted using data from the Cerner® electronic database. The Cerner database consisted of anonymized electronic medical records (EMRs) of patients treated under Abu Dhabi health services (SEHA) in the UAE from 49 health centers across the region of Abu Dhabi from January 1, 2015, to April 31, 2018.

**Operational definitions**

- ACS was defined as an ST-segment elevation myocardial infarction or left bundle branch block myocardial infarction, a non-ST-segment-elevation myocardial infarction, or unstable angina
- Stable CHD was defined as one or more of the following: coronary stenosis of >50% as assessed by cardiac computed tomography or coronary angiography, prior percutaneous coronary intervention or prior coronary artery bypass grafting, or history of ACS (>3 months from the index date)
- Obesity was defined as the body mass index >30 kg/m\(^2\) according to the World Health Organization guidelines
- Diabetes was defined as the current treatment for diabetes, a previous diagnosis of diabetes, or having blood pressure >140/90 mmHg
- The lipid profile included measurement of serum levels of total cholesterol, high-density lipoprotein cholesterol (HDL-C), LDL-C, and triglycerides as per the local practice
- LLT includes any category of statins, ezetimibe, fibrates, bile acid, and niacin
- Patients on LLT treatment were considered if there was any evidence of medication coverage on or within 30 days or any recorded prescription of medication days supplied before the index hospitalization date (baseline)
- Patients with no evidence for LLT over 30 days before the index date but used LLT for at least 90 days after ACS hospitalization were considered as “LLT treatment”
- Patients with no evidence of a prescription for LLT on or during the 2 years before ACS hospitalization were considered as not on LLT treatment
- LDL-C target level achievement was defined as <70 mg/dl, according to the 2013 American College of Cardiology/American Heart Association (ACC/AHA) and European Society of Cardiology/European Atherosclerosis Society (ESC/EAS) guidelines.\textsuperscript{[1,2]}

**Inclusion criteria**

The following inclusion criteria were used:

a. Adult Emirati patients discharged after an ACS event from January 2015 to April 2018
b. Patients were followed up at acute care centers, and ambulatory treatment of secondary prevention was considered
c. Patients treated with LLT at prior hospitalization and on admission for the ACS or at the time of the physician follow-up visit
d. Presence of ≥1 lipid profile in 2015 (the last LDL-C measurement in 2015 was considered the index date)
e. Evidence of at least one CV risk condition before the index hospitalization, for which statins would be recommended as per the 2013 ACC/AHA and ESC/EAS guidelines\textsuperscript{[1,2]}
f. At least 3 months of follow-up from the index date with lipid parameters and LLT
g. Patients were identified using the International Classification of Diseases, Tenth Revision diagnosis, or procedure codes.

**Data variables**

Patient demographic data and clinical variables at discharge including age, gender, body mass index, and comorbidities such as hypertension, type 2 diabetes mellitus, chronic heart failure or chronic renal failure, prior myocardial infarction or angina, and chronic obstructive pulmonary disease were collected. Clinical parameters, lipid profile, and outcomes at 6 months of follow-up were collected. Patients with ACS who did not survive until discharge were excluded from the study.
Patients were divided into two subgroups based on whether or not they were treated with LLT at the baseline. The following mutually exclusive classes of LLT were considered based on the statin potency: high-, moderate-, and low-intensity statins.\textsuperscript{[1]} The statins assessed were atorvastatin, rosuvastatin, simvastatin, and pravastatin. The CV health outcomes were assessed at weeks 12 and 24 after the index hospitalization. These outcomes included rehospitalization, recurrent myocardial infarction, percutaneous coronary intervention, coronary artery bypass grafting, and stroke. Any lipid profiles and LLT data available within the follow-up period were collected.

Ethical clearance
The study protocol was approved by the Abu Dhabi regional human ethics committees, and the research was performed in accordance with the Declaration of Helsinki. Patients’ informed consent was required for the retrospective study.

Statistical analysis
Categorical variables were presented as numbers and/or percentages and continuous variables as means and standard deviations. Demographic and clinical variables at baseline were initially compared between patients on LLT and those not on LLT using Chi-square or Mann–Whitney U-test. LDL-C target attainment was assessed using pre-ACS risk classification and also according to the time point (3 months and 6 months of follow-up). Lipid profiles and type of LLT used at the baseline and follow-up were assessed descriptively. Multivariate logistic regression analysis was performed to evaluate the odds ratio (OR) with 95% confidence interval (CI) to predict the patient factors associated with LDL-C goal attainment. \( P < 0.05 \) was considered statistically significant.

RESULTS

Patient characteristics
A total of 3066 Emirati adult patients were identified with documented ACS from January 2015 to June 2018. The mean (standard deviation) age of the patients with ACS was 65.5±14 years, and 64.4% were male [Table 1]. Overall, 58.1% (n = 1782) of the patients in the cohort were on LLT prior to index hospitalization with ACS, and these patients were younger compared with those not on LLT (\( P < 0.001 \)). The majority of the patients not on LLT had hypertension (31.7% vs. 22.1%) or diabetes (31% vs. 22.7%). Thirty-six patients (1.2%) died during the follow-up period.

Lipid profile
At discharge, the mean LDL-C level was 84.8±39.0 mg/dl, with a total cholesterol of 152.3±45.3 mg/dl [Table 1].

| Table 1: Demographic and clinical characteristics of acute coronary syndrome cohort (n = 3066) |
|-------------------------------------------------|-------------------------------------------------|-------------------------------------------------|-----------------|
| All patients | Patients on LLT (n=1782; 58.1%), n (%) | Patients not on LLT (n=1284; 41.8%), n (%) | \( P \)          |
| Age (years), mean±SD | 65.5±14 | 64.3±13.4 | 67.1±13.9 | \textless 0.001 |
| Male | 1976 (64.4) | 1188 (38.7) | 788 (25.7) | 0.003 |
| BMI | 29.3±6.8 | 29.3±5.7 | 29.4±7.2 | 0.668 |
| HbA1c | 7.1±1.7 | 7.1±1.8 | 7.3±1.8 | 0.351 |
| SBP | 134.8±23.8 | 137.3±23.5 | 130.8±22.6 | \textless 0.001 |
| DBP | 73.8±14.5 | 76±14.4 | 71.8±13.9 | 0.001 |
| Comorbidities | | | | |
| Heart rate | 77.5±17.6 | 78.6±19 | 78±16.6 | 0.419 |
| Hypertension | 1653 (54) | 680 (22.1) | 973 (31.7) | 0.368 |
| Diabetes | 1649 (53.7) | 698 (22.7) | 951 (31) | 0.586 |
| Heart failure | 496 (16.1) | 269 (8.77) | 227 (7.4) | 0.055 |
| Chronic kidney disease | 772 (23.5) | 415 (13.5) | 357 (11.6) | 0.004 |
| Prior angina | 476 (15.5) | 282 (9.2) | 194 (6.3%) | 0.589 |
| COPD | 76 (2.4) | 40 (1.3%) | 36 (1.1%) | 0.326 |
| Primary diagnosis | | | | |
| Acute coronary syndrome | 2449 (79.8) | 1449 (47.2) | 1000 (32.6) | 0.349 |
| Lipid levels (mg/dl) | | | | |
| Total cholesterol | 152.3±45.3 | 154.9±47.7 | 148.7±41.4 | 0.694 |
| LDL-C | 84.8±39.0 | 87.4±41.7 | 81.3±34.5 | 0.308 |
| HDL-C | 41.1±1.0 | 40.6±12.1 | 41.8±13.2 | 0.610 |
| Triglycerides | 58.1±34.4 | 58.9±36.6 | 56.9±30.9 | 0.319 |
| Non-HDL-C | 111.2±43.7 | 114.3±46.7 | 106.8±38.9 | \textless 0.001 |

\( P \) values reflect \( \chi^2 \) or Mann–Whitney-Wilcoxon test between values for treated and untreated patients. Statistically significant values are bolded. Data are presented as numbers and percentages and mean with standard deviations. ACS: Acute coronary syndrome, LLT: Lipid-lowering therapy, HbA1c: Glycated hemoglobin, SBP: Systolic blood pressure, DBP: Diastolic blood pressure, COPD: Chronic obstructive pulmonary disease, NSTEMI: Non-ST-elevated myocardial infarction, SD: Standard deviation, LDL-C: Low-density lipoprotein cholesterol, HDL-C: High-density lipoprotein cholesterol.
The LLT-treated patients had higher mean LDL-C level (87.4 ± 41.7 vs. 81.3 ± 34.5) and total cholesterol level (154.9 ± 47.7 vs. 148.7 ± 41.4) than the non-treated patients. In total, 58% of the patients were on LLT; 28%, 21%, and 9% received high-, moderate-, and low-intensity statins (9%), respectively [Figure 1a]. A large proportion of the patients (42%) did not have current evidence of any LLT; 32.8% had no written prescription of an LLT during the 2 years prior to the index hospitalization date, and 9.2% had a previous (but no current) LLT prescription. Among the patients using statin therapy, 41% were on atorvastatin; 15% were on simvastatin; and 2% were on rosuvastatin and pravastatin [Figure 1b].

Low-density lipoprotein cholesterol changes after 3 months and 6 months

In the overall cohort, a higher proportion of patients with LDL-C <70 mg/dl (40.5%) or 71–100 mg/dl (31.8%) had prescribed high-intensity statins (19%) compared with the patients with LDL-C > 100 mg/dl [Table 2]. At 6 months (n = 2046; 66.7%), 27.7% and 16.7% of the patients with any LLT therapy achieved an LDL-C of <70 mg/dl and 70–100 mg/dl, respectively. In the ACS population, the highest level of LDL-C reduction by 50% within 6 months was observed among patients using moderate-intensity statin (37.2%) compared with high- and low-intensity statins (35.6% and 32.1%) [Figure 2]. Correspondingly, mean changes in lipid parameters between men and women at 3 months and 6 months are presented in Table 3. The mean reduction in LDL-C level in men was −2.4 mg/dl at 3 months and −7.1 mg/dl at 6 months. The mean reduction in LDL-C level in women was −3.3 mg/dl at 3 months and −5.2 mg/dl at 6 months.

Predictors of low-density lipoprotein cholesterol target attainment for patients treated with LLT

Several variables increased the likelihood of patients reaching an LDL-C level of < 70 mg/dl. Increasing age significantly increased the odds of achieving this level [Table 4]. In addition, hypertension (OR: 0.89; 95% CI: 0.754–1.050), diabetes (OR: 0.94; 95% CI: 0.810–1.090), and high-intensity statin (OR: 0.923; 95% CI: 0.749–1.136) were also associated with lower odds of LDL-C target value attainment but not statistically significant. On the other hand, being female (OR: 1.05; 95% CI: 0.90–1.22), obesity (OR: 1.00; 95% CI: 0.83–1.20), heart failure (OR: 1.04; 95% CI: 0.84–1.28), and moderate-intensity statin (OR: 1.28; 95% CI: 0.96–1.70) decreased the odds of achieving the LDL-C level of < 70 mg/dl.

DISCUSSION

The current study is the first to assess LLT utilization and achievement of LDL-C targets after ACS in real-world practice in Abu Dhabi using a large EMR database. We identified 3066 Emirati patients with ACS during the study period. The patients who had at least two lipid profiles within 180 days and those on/not on LLT were evaluated. The results of the study suggest two important findings. First, only 58.1% of the patients were treated with LLT. Second, only 27.7% of the patients on LLT therapy achieved a guideline-recommended LDL-C threshold.

Statin therapy has a significant role in secondary prevention by decreasing the burden of atherosclerotic plaque, the risk of adverse events, and revascularization in symptomatic patients with CAD. Compared with other studies, we observed a low percentage of Emirati...
Shehab and Bhagavathula: Statin utilization and LDL-C goal attainment in the UAE

Table 2: Low-density lipoprotein cholesterol changes after 3 months and 6 months of lipid-lowering therapy

|                   | <70 mg/dl | 71-100 mg/dl | 101-159 mg/dl | 151-190 mg/dl | >190 mg/dl |
|-------------------|-----------|--------------|---------------|---------------|------------|
| **At discharge**  |           |              |               |               |            |
| High intensity    | 342 (11.2)| 240 (7.8)    | 208 (6.8)     | 67 (2.2)      | 19 (0.6)   |
| Moderate          | 261 (8.5 )| 195 (6.4)    | 134 (4.4)     | 35 (1.1)      | 12 (0.4)   |
| Low               | 89 (2.9)  | 111 (3.6)    | 57 (1.9)      | 6 (0.2)       | 4 (0.1)    |
| Not on LLT        | 551 (18)  | 429 (14)     | 240 (7.8)     | 57 (1.9)      | 7 (0.2)    |
| **After 3 months**|           |              |               |               |            |
| High intensity    | 383 (12.5)| 246 (8)      | 189 (6.2)     | 47 (1.5)      | 11 (0.4)   |
| Moderate          | 272 (8.9 )| 205 (6.7)    | 126 (4.1)     | 29 (0.9)      | 5 (0.2)    |
| Low               | 80 (2.6)  | 80 (2.6)     | 53 (1.7)      | 12 (0.4)      | 3 (0.1)    |
| Not on LLT        | 394 (12.9)| 394 (12.9)   | 253 (8.3)     | 52 (1.7)      | 13 (0.4)   |
| **At 6 months (n=2046)** |     |              |               |               |            |
| High intensity    | 276 (13.5)| 165 (8.1)    | 110 (5.4)     | 19 (0.9)      | 3 (0.1)    |
| Moderate          | 210 (10.3)| 125 (6.1)    | 63 (3.1)      | 20 (1)        | 1 (0.0)    |
| Low               | 82 (4)    | 52 (2.5)     | 46 (2.2)      | 6 (0.3)       | 4 (0.2)    |
| Not on LLT        | 415 (20.3)| 243 (11.9)   | 172 (8.4)     | 25 (1.2)      | 7 (0.3)    |

LDL-C: Low-density lipoprotein cholesterol, LLT: Lipid-lowering therapy

Table 3: Mean ± standard deviation changes in lipid parameters

| Mean changes       | Baseline         | Week 12         | Week 24         |
|--------------------|------------------|-----------------|-----------------|
|                    | Male             | Female          | Male            | Female          |
| LDL-C (mg/dl)      | 85.2±40.1        | 84.3±36.8       | 82.8±38.2       | 81±37           | 78.1±34.8 | 79.1±36.4     |
| HDL-C (mg/dl)      | 38.4±11.0        | 46±14           | 45.1±14.2       | 45.1±13.8       | 43.9±12.6 | 44.3±13.4     |
| TC (mg/dl)         | 150.1±43.0       | 156±43.0        | 153.6±43.8      | 154.4±44.8      | 151±43    | 151.2±41      |
| TG (mg/dl)         | 57±33.8          | 60±36.8         | 59.2±45.3       | 58.4±35.5       | 55.5±33   | 56.4±31       |
| Non-HDL-C          | 111.6±45.3       | 110.3±40.9      | 110.5±46        | 109.4±47        | 107±44.8 | 106.9±43.1    |

LDL-C: Low-density lipoprotein cholesterol, HDL-C: High-density lipoprotein cholesterol, TC: Total cholesterol, TG: Triglyceride

Table 4: Predictors of achieving low-density lipoprotein cholesterol <70 mg/dl after 6 months of follow-up

| Reached LDL-C levels <70 mg/dl | OR (95% CI) | P     |
|--------------------------------|-------------|-------|
| Achieved (n=1243; 40.5%)       |             |       |
| Not achieved (n=1823; 59.5%)   |             |       |
| Gender                         |             |       |
| Male                           | 809 (65.1)  | 1167 (64) | -    | -    |
| Female                         | 434 (34.9)  | 656 (36)  | 1.048 (0.901-1.218) | 0.544 |
| BMI >30 (kg/m²)                | 310 (25)    | 489 (26.8) | 1.001 (0.834-1.201) | 0.994 |
| Age                            |             |       |
| 18-45                          | 47 (3.8)    | 210 (11.5) | 0.613 (0.393-0.956) | 0.031 |
| 46-50                          | 54 (4.3)    | 148 (8.1)  | 0.480 (0.321-0.719) | <0.001 |
| 51-55                          | 89 (7.2)    | 191 (191)  | 0.327 (0.223-0.481) | <0.001 |
| 56-60                          | 136 (10.9)  | 199 (10.9) | 0.262 (0.189-0.364) | <0.001 |
| >60                            | 917 (73.8)  | 1075 (59)  | -    | -    |
| Comorbidities                  |             |       |
| Hypertension                   | 688 (55.3)  | 965 (52.9)  | 0.890 (0.754-1.050) | 0.168 |
| Diabetes                       | 682 (54.9)  | 967 (53)    | 0.940 (0.810-1.090) | 0.412 |
| Heart failure                  | 200 (16.1)  | 296 (16.2)  | 1.038 (0.840-1.282) | 0.733 |
| Chronic kidney disease         | 313 (25.2)  | 459 (25.2)  | 0.951 (0.776-1.165) | 0.625 |
| Prior angina                   | 195 (15.7)  | 281 (15.4)  | 1.000 (0.818-1.222) | 0.996 |
| Statin intensity*              |             |       |
| Not using statins              | 551 (44.3)  | 733 (40.2)  | -    | -    |
| High-intensity statin          | 342 (27.5)  | 534 (29.3)  | 0.923 (0.749-1.136) | 0.449 |
| Moderate-intensity statin      | 261 (21)    | 376 (20.6)  | 1.281 (0.960-1.709) | 0.092 |
| Low-intensity statin           | 89 (7.2)    | 178 (9.8)   | 0.852 (0.715-1.015) | 0.073 |

*Some patients are on statin + ezetimibe. Statistically significant values are bolded. OR: Odds ratio, CI: Confidence interval, LDL-C: Low-density lipoprotein cholesterol, BMI: Body mass index

patients who received LLT (58.1%) while being at high risk of CV events.[22-24] Thus, it confirms that the use of statins in the UAE remains comparatively low despite the guideline recommendation worldwide. Further analysis
showed that suboptimal dosing of statin prescription in secondary prevention and poor intensification of statin therapy were only moderate after ACS. In the present study, 28% of the patients received high-intensity statins and 30% received low-to-moderate-intensity statins. This result suggests a considerable discordance between the real-world practice and the guideline-recommended lipid targets in the UAE. Our results are similar to those observed in other studies\textsuperscript{[25-28]} and confirm that statin treatment is an essential component for the secondary prevention of CVD.

Despite the recommendations of international guidelines for the use of high-intensity statins and the greater probability of goal achievement with increasing statin intensity, only 27.5% of the patients taking high-intensity statins achieved the LDL-C targets and 21% on moderate-intensity statins reached the very high-risk targets (70 mg/dl) in our study.

A study\textsuperscript{[29]} that measured non-HDL-C levels in a Gulf population found that 73.3% had non-HDL-C levels <130 mg/dl (ESC/EAS high CV risk target: 3.3 mmol/l) and 48.6% had non-HDL-C levels <100 mg/dl (very high CV risk target: <2.6 mmol/l). Our study results are consistent with the evidence of the guideline-recommended goal achievement found in other studies.\textsuperscript{[14,16,19,22]}

In the Dyslipidemia International Study II,\textsuperscript{[116]} which evaluated LDL-C goal achievement in 18 countries, found that 19% and 37% of the patients with CHD and ACS treated with statins identified by the ESC/EAS guidelines achieved the <70 mg/dl LDL-C target at admission and follow-up, respectively. In the EUROASPIRE IV study, only 19.3% had an LDL-C level of <70 mg/dl despite the widespread use of LLT in this population.\textsuperscript{[30]}

A study reported that a reduction of 39 mg/dl (1 mmol/dl) of LDL-C was associated with an approximate 23% reduction in CV events.\textsuperscript{[31]} The results of our study suggest that many Emirati patients requiring LDL-C lowering are not receiving statin LLT. Therefore, closely implementing the guidelines in the UAE represents the best practice and would ultimately reduce the risk of CV-related events.

A multivariate regression analysis was performed to evaluate characteristics that may increase or decrease the likelihood of LDL-C target attainment in patients treated with LLT. Predictors for better LDL-C target attainment were younger age, comorbidities, and statin treatment intensity. A significant strong inverse correlation was found between age and attaining LDL-C target. With the increase in age, ACS patients were less likely to reach LDL-C level < 70 mg/dl in our study, in contrast to previous reports.\textsuperscript{[32-34]} Although the reason for this is not apparent, we did not reach any conclusion and suggested that additional studies are necessary to clarify these points specific to the Emirati population.

The use of combination therapy with statins and ezetimibe or proprotein convertase subtilisin/kexin type 9 inhibitors is an emerging therapeutic option that lowers LDL-C by 50%–70% and reduces CV events in patients with recent ACS to a greater degree than a statin alone.

Limitations

This study had several limitations that should be considered. This is a retrospective observational study; identifying the causes of low utilization of LLT and poor achievement of LDL-C target attainment in the UAE population is not possible. The cohort represents only the Emirati population treated under SEHA facilities in a general practice setting. Thus, caution should be taken when extrapolating the results to the general population.

Due to the retrospective nature of the study, some data were not available. Patients were selected based on the availability of lipid measurements, which might not be representative of the total UAE patients. The characteristics of medical conditions and risk factors were limited to EMR. Moreover, the population included only patients on some form of LLT, and the data were captured using prescription order in the CERNER database. Finally, the study did not include any evaluation of statin monotherapy versus statin with other LLTs. Despite these limitations, our results provided a useful overview of the current lipid management and LDL-C goal attainment in the UAE population.

CONCLUSION

A large proportion of Emirati patients were not on LLT after ACS, and the rate of LDL-C target value attainment was extremely poor. Optimal statin utilization by closely implementing the guidelines in the UAE is recommended so that patients could receive adequate LLT. Furthermore, not attaining the LDL-C goal despite statin therapy suggests that higher statin doses with proven non-statin treatments could improve lipid control and CVD outcomes. These findings suggest that there is a substantial opportunity to improve through optimization of intensity of statin therapy in the secondary prevention of ASCVD in the UAE.

Acknowledgments

We would like to thank Ms. Eiman Nasser Alshamisi, senior analytical officer, SEHA for providing extensive support in data collection and data filtration. The preliminary findings of the research are presented in the 10th Emirates Cardiac Society–American College of Cardiology Middle East conference on October 3–5, 2019, Dubai, UAE.
Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

REFERENCES

1. Barquera S, Pedroza-Tobías A, Medina C, Hernández-Barrera L, Bibbins-Domingo K, Lozano R, et al. Global Overview of the Epidemiology of Atherosclerotic Cardiovascular Disease. Arch Med Res 2013;46:328-38.

2. Mundal I, Igland J, Ose L, Holven KB, Veierød MB, Leren TP, et al. Cardiovascular disease mortality in patients with genetically verified familial hypercholesterolemia in Norway during 1992-2013. Eur J Prev Cardiol 2017;24:137-44.

3. Teo KK, Dokainish H. The Emerging Epidemic of Cardiovascular Risk Factors and Atherosclerotic Disease in Developing Countries. Can J Cardiol 2017;33:65-65.2.

4. Benjamin EJ, Blaha MJ, Chiuve SE, Cushman M, Das SR, Deo R, et al. Heart disease and stroke statistics-2017 update: A report from the American heart association. Circulation 2017;135:e1-e603.

5. US Preventive Services Task Force, Curry SJ, Krist AH, Owens DK, Barry MJ, Caughley AB, et al. Risk Assessment for Cardiovascular Disease With Nontraditional Risk Factors: US Preventive Services Task Force Recommendation Statement. JAMA 2018;320:272-80.

6. Health Statistics 2016, Health Authority of Abu Dhabi, Department of Health, Abu Dhabi, UAE. Available from: https://haad.ae/HAAD/tabid/58/ctl/Details/Mid/417/Id/688/Default.aspx (19). [Last accessed on 31 May 2020].

7. Catapano AL, Graham I, De Backer G, Wiklund O, Chapman MJ, Drexel H, et al. 2016 ESC/EAS guidelines for the management of dyslipidaemias. Eur Heart J 2016;37:2999-3058.

8. Stone NJ, Robinson JG, Lichtenstein AH, Bairey Merz CN, Blum CB, Eckel RH, et al. 2013 ACC/AHA guideline on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults: A report of the American college of cardiology/American heart association Task force on Practice guidelines. J Am Coll Cardiol 2014;63:2889-934.

9. European Association for Cardiovascular Prevention and amp; Rehabilitation, Reiner Z, Catapano AL, De Backer G, Graham I, Taskinen MR, et al. ESC/EAS Guidelines for the management of dyslipidaemias: The task force for the management of dyslipidaemias of the European Society of Cardiology (ESC) and the European Atherosclerosis Society (EAS). Eur Heart J 2011;32:1769-818.

10. Collins R, Reith C, Emberson J, Armitage J, Baigent C, Blackwell L, et al. Interpretation of the evidence for the efficacy and safety of statin therapy. Lancet 2016;388:2532-61.

11. Cholesterol Treatment Trialists’ (CTT) Collaboration, Fulcher J, O’Connell R, Voysey M, Emberson J, Blackwell L, et al. Efficacy and safety of LDL-lowering therapy among men and women: Meta-analysis of individual data from 174,000 participants in 27 randomised trials. Lancet 2015;385:1397-405.

12. Navarese EP, Robinson JG, Kowalewski M, Kolodziejczak M, Andreotti F, Blieden K, et al. Association Between Baseline LDL-C Level and Total and Cardiovascular Mortality After LDL-C Lowering: A Systematic Review and Meta-analysis. JAMA 2018;319:1566-79.5.

13. Silverman MG, Ference BA, Im K, Wiviott SD, Giugliano RP, Grundy SM, et al. Association Between Lowering LDL-C and Cardiovascular Risk Reduction Among Different Therapeutic Interventions: A Systematic Review and Meta-analysis. JAMA 2016;316:1289-97.

14. Kuiper JG, Sanchez RJ, Houben E, Heintjes EM, Penning-van Beest FJA, Khan I, et al. Use of lipid-modifying therapy and LDL-C Goal attainment in a high-cardiovascular-risk population in the Netherlands. Clin Ther 2017;39:819-270.

15. Chiang CE, Ferrière J, Gotcheva NN, Raal FJ, Shehab A, Sung J, et al. Suboptimal control of lipid levels: Results from 29 countries participating in the centralized pan-regional surveys on the undertreatment of hypercholesterolaemia (CEPHEUS). J Atheroscler Thromb 2016;23:567-87.

16. Gitt AK, Lautsch D, Ferrière J, De Ferrari GM, Vyas A, Baxter CA, et al. Cholesterol target value attainment and lipid-lowering therapy in patients with stable or acute coronary heart disease: Results from the Dyslipidemia International Study II. Atherosclerosis 2017;266:158-66.

17. Fox KM, Tai MH, Kostev K, Hatz M, Qian Y, Laufs U. Treatment patterns and low-density lipoprotein cholesterol (LDL-C) goal attainment among patients receiving high- or moderate-intensity statins. Clin Res Cardiol 2018;107:380-8.

18. Poh KK, Ambegaonkar B, Baxter CA, Brudi P, Buddhari W, Chiang FT, et al. Low-density lipoprotein cholesterol target attainment in patients with stable or acute coronary heart disease in the Asia-Pacific region: Results from the dyslipidemia international study II. Eur J Prev Cardiol 2018;25:1950-63.

19. Al-Zakwani I, Shehab A, Al-Hinai AT, Al Mahmeed W, Arafah M, Al Tamimi O, et al. Gender disparity in lipid target achievements in high and very high atherosclerotic cardiovascular disease risk patients in the Arabian gulf. Curr Vasc Pharmacol 2017;15:51-8.

20. Al Sifri S, Al Shammeri O, Al Jaser S, Alkhazen A, Bin Shafi Shafru’rehman A, Morcos B, et al. Prevalence of lipid abnormalities and cholesterol target value attainment in patients with stable coronary heart disease or an acute coronary syndrome in Saudi Arabia. Saudi Med J 2018;39:697-704.

21. Al-Rasadi K, Al-Zakwani I, Alsheikh-Ali AA, Almahmeed W, Arafah M, Al Tamimi O, et al. Prevalence, management, and outcomes of familial hypercholesterolemia in patients with acute coronary syndromes in the Arabian Gulf. J Clin Lipidol 2018;12:685-920.

22. Arca M, Ansell D, Avema M, Fanelli F, Gorgyca K, Iorga ŞR, et al. Statin utilization and lipid goal attainment in high or very-high cardiovascular risk patients: Insights from Italian general practice. Atherosclerosis 2018;271:120-7.

23. Boklage SH, Malangone-Monaco E, Lopez-Gonzalez L, Ding Y, Henrique S, Ellass J, Statin utilization patterns and outcomes for patients with acute coronary syndrome during and following inpatient admissions. Cardiovasc Drugs Ther 2018;32:273-80.

24. Maddox TM, Borden WB, Fang T, Virani SS, Oetgen WJ, Mullen JB, et al. Implications of the 2013 ACC/AHA coronary artery disease prevention guidelines for adults in contemporary cardiovascular practice: Insights from the NCDR PINNACLE registry. J Am Coll Cardiol 2014;64:2183-92.

25. Salama AM, Warraich H, Valero-Elizondo J, Spatz ES, Desai NR, Rana JS, et al. National trends in statin use and expenditures in the US adult population from 2002 to 2013: Insights from the medical expenditure panel survey. JAMA Cardiol 2017;2:56-65.

26. Rosenson RS, Kent ST, Brown TM, Farkouh ME, Levitan EB, Yun H, et al. Underutilization of high-intensity statin therapy after hospitalization for coronary heart disease. J Am Coll Cardiol 2015;65:270-7.

27. Danchin N, Almahmeed W, Al-Rasadi K, Azizi J, Berrah A, Cuneo CA, et al. Achievement of low-density lipoprotein cholesterol goals in 18 countries outside Western Europe: The International Cholesterol management Practice Study (ICLPS). Eur J Prev Cardiol 2018;25:1087-94.

28. Yeh YT, Yin WH, Tseng WK, Lin FJ, Yeh HI, Chen JW, et al. Lipid
lowering therapy in patients with atherosclerotic cardiovascular diseases: Which matters in the real world? Statin intensity or low-density lipoprotein cholesterol level? Data from a multicenter registry cohort study in Taiwan. PLoS One 2017;12:e0186861.

29. Al-Hashmi K, Al-Zakwani J, Al Mahmeed W, Arafah M, Al-Hinai AT, Shehab A, et al. Non-high-density lipoprotein cholesterol target achievement in patients on lipid-lowering drugs and stratified by triglyceride levels in the Arabian Gulf. J Clin Lipidol 2016;10:368-77.

30. Reiner Ž, De Backer G, Fras Z, Kotseva K, Tokgözoglu L, Wood D, et al. Lipid lowering drug therapy in patients with coronary heart disease from 24 European countries – Findings from the EUROASPIRE IV survey. Atherosclerosis 2016;246:243-50.

31. Cannon CP, Blazing MA, Giugliano RP, McCagg A, White JA, Theroux P, et al. Ezetimibe added to statin therapy after acute coronary syndromes. N Engl J Med 2015;372:2387-97.

32. Mohd-Zulkifli SZ, Omar MS, Md-Redzuan A. Factors associated with lipid goal attainment among acute coronary syndrome patients. Patient Prefer Adherence 2016;10:1631-7.

33. Chen Y, Li D, Jing J, Yan H, Liu J, Shen Z, et al. Treatment trends, effectiveness, and safety of statins on lipid goal attainment in Chinese percutaneous coronary intervention patients: A multicenter, retrospective cohort study. Clin Ther 2017;39:1827-390.

34. Ferrieres J, De Ferrari GM, Hermans MP, Elisaf M, Toth PP, Horack M, et al. Predictors of LDL-cholesterol target value attainment differ in acute and chronic coronary heart disease patients: Results from DYSIS II Europe. Eur J Prev Cardiol 2018;25:1966-76.