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Disparities in patterns and outcomes of dyslipidemic patients with acute coronary syndrome: A tertiary cardiac center registry

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1. Introduction

Cardiovascular diseases (CVD) are the leading cause of death worldwide [1]. Hypercholesterolemia is a well-known risk factor for coronary artery disease (CAD), particularly due to prolonged exposure to elevated levels of low-density lipoprotein cholesterol (LDL-C). [2] Familial hypercholesterolemia (FH) results in lifelong elevated plasma LDL-C levels, leading to premature CVD, especially CAD. Untreated FH patients face a 20-fold increased risk of premature CAD compared to individuals without this condition, significantly raising the associated morbidity and mortality related to CVD.

Therefore, early diagnosis and treatment of FH in the general population are crucial [3]. Furthermore, identifying FH in patients hospitalized for an ACS would allow specific counseling for diet and cardiovascular (CV) risk factors, ensure proper lipid-lowering therapy (LLT) prescription at discharge, and provide appropriate referral to lipid clinics for further close follow-up [8]. Different diagnostic algorithms have been developed for FH diagnosis; the most commonly used are the Dutch Lipid Clinic Network (DLCN) criteria, the UK Simon Broome Register (SBR) criteria, and the US Make Early Diagnosis Prevent Early Death (MEDPED) criteria [4–6].

Unfortunately, recent data indicate that fewer than half of FH patients, particularly young adults with LDL-C levels exceeding 190 mg/dL, are receiving statins. This is concerning given their elevated risk of atherosclerotic events from an early age [7].

Increasing awareness of the incidence and management of dyslipidemia among high-risk groups is contributing to better control of this serious risk factor in certain regions [10]. However, to enhance these efforts, it is crucial that the identification and screening for dyslipidemia become integral components of a national strategy aimed at preventing the occurrence and progression of CVD, supported by accurate national statistics on the prevalence and determinants of dyslipidemia [9].

This study aimed to assess the patterns of dyslipidemia and the incidence of FH in patients with ACS, along with the variations among the algorithms used to diagnose FH.

2. Methods

This was a prospective observational study, conducted between December 2018 and November 2021 and included two thousand ACS patients who were admitted to the CCU at Prince Khaled Ben Sultan's Cardiac Center, Abha, Saudi Arabia.

Inclusion criteria: These criteria consisted of adult patients (≥18 years old) admitted to the cardiac care unit (CCU) with ACS. ACS encompasses a variety of acute CV conditions, including myocardial infarction (MI), which is further classified into ST-segment elevation myocardial infarction (STEMI) and non-ST-segment elevation myocardial infarction (NSTEMI). Both types were diagnosed in this study according to the fourth universal definition of MI [11]. Additionally, ACS includes unstable angina (UA), characterized by new, worsening or resting chest discomfort that usually lasts more than 20 min in patients whose cardiac biomarkers do not fulfill the criteria of the fourth universal definition of MI, even if their admission ECG shows ST segment deviation or T wave inversion.

Exclusion criteria: Patients were excluded if values for any of the items of the lipid profile were missing, lipid profile blood sampling was conducted more than 24 h after CCU admission, baseline triglyceride (TG) levels were greater than 400 mg/dL, or if they presented with hemodynamic instability or developed cardiogenic shock. Patients with chronic renal failure (i.e., eGFR <60 mL/min/1.73 m^2), or presenting with acute renal failure at the time of admission, and those consuming more than 14 units of alcohol per week were also excluded.

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2.1. Study procedures

Consecutive patients presenting with ACS and admitted to the CCU were enrolled in the study. A thorough history-taking was performed. It included demographic data (e.g., age, gender), risk factors for CAD including personal/family history of premature CVD, other chronic medical conditions such as diabetes mellitus (DM) and hypertension (HTN) and drug history especially LLT. The diagnosis of ACS was based on criteria stated in the guidelines [11,12]. Moreover, clinical signs of dyslipidemia and FH like corneal arcus and tendinous xanthomas were also looked for and assessed [11].

For LDL-C assessment; fasting or non-fasting total cholesterol, HDL-C and triglycerides (TG), within 24 h of hospital admission, were measured with Direct Detect Spectrometer using Abbot Alinity C device [13].

The multiplication factors for converting units of mmol/L to mg/dl for cholesterol (total cholesterol, LDL-C, and HDL-C) and triglycerides were 38.67 and 88.57 respectively.

If pretreatment LDL-C levels were unavailable for patients on LLT prior to hospitalization, untreated LDL-C levels were estimated based on the type and dosage of medication administered before hospitalization, by applying a correcting factor according to the reported efficacy of each drug [14–18].

Patients were then categorized according to the DLCN score and the SBR criteria to identify those at risk of having FH. The DLCN criteria represent a scoring system based on personal or family history of premature coronary or vascular disease, the presence of corneal arcus or tendon xanthomas upon clinical examination, pre-treatment LDL-C levels, and the presence of functional mutations in the LDLR, ApoB, or PCSK9 genes. The total score is interpreted as follows: (0–2) → Unlikely FH, (3–5) → Possible FH, (6–8) → Probable FH, and (>8) → Definite FH [4]. On the other hand, the UK SBR criteria diagnose possible FH as elevated LDL-C > 190 mg/dL (or TC > 290 mg/dL) along with family or personal history of premature atherosclerosis. A definite FH diagnosis, however, requires the aforementioned LDL-C and TC levels, in addition to the presence of tendon xanthomas (in the patient or relatives) or positive DNA mutation associated with FH [5].

Management during the hospital stay was documented, including medications, reperfusion therapy or procedures, and discharge medications.

2.2. Statistical analysis

Data were analysed using IBM© SPSS© Statistics version 26 (IBM© Corp., Armonk, New York, USA). Categorical variables are presented as numbers or proportions and percentages and intergroup differences are compared using the Pearson chi-squared test or Fisher's exact test. Ordinal data are compared using the chi-squared test for trend. Skewed numerical data are presented as medians with interquartile ranges, and between-group differences are compared using the Jonckheere-Terpstra trend test, with the Conover test applied for post-hoc comparisons if needed.

3. Results

Out of 2367 patients presenting with acute coronary syndrome, 2000 ACS patients were included in the study. The main causes of exclusion were the presence of acute/chronic kidney disease (234 patient), triglyceride above 400 mg/dL (43 patients), missing values for lipid profile (24 patients), and more than 24 h between symptom onset and lipid testing (66 patients). Baseline demographic, clinical, and biochemical characteristics of the study population are presented in (Table 1). Regarding serum lipid profile values (mean ± SD), mean serum TC was 159.2 ± 46.9 mg/dL, mean serum LDL was 99.9 (±40.9) mg/dL, the mean corrected LDL was 148.2 ± 51.7 mg/dL, mean serum HDL was 34.9 ± 10.3 mg/dL, and the mean serum TG was 131.2 ± 78.5 mg/dL.

Table 1
Baseline demographic, clinical, & biochemical characteristics of the study population.

Demographics	
Age (years) ± SD	64.4 ± 13.8 %
Male (%)	1528 (76.4 %)
Smoking status n (%)	337 (16.9 %)
Comorbidities n (%)	
Hypertension	1378 (68.9 %)
Diabetes mellitus	1330 (66.5 %)
Dyslipidemia	1078 (53.9 %)
Prior statin intake	1156 (57.9 %)
PVD	46 (2.3 %)
CVS	135 (6.8 %)
IHD:	
Prior MI	298 (14.9 %)
Prior angina	519 (26 %)
Prior PCI	437 (21.9 %)
Prior CABG	115 (5.8 %)
Admission Diagnosis n (%)	
UA	521 (26.05 %)
NSTEMI	922 (46.1 %)
STEMI	557 (27.85 %)
Lipid Profile: Mean ± SD	
Total Cholesterol (mg/dL)	159 ± 46.9
LDL-C (mg/dL)	99.9 ± 40.9
Corrected LDL-C (mg/dL)	148.2 ± 51.7
HDL-C (mg/dL)	34.9 ± 10.3
TG (mg/dL)	131.2 ± 78.5
Other Biochemical: Mean ± SD	
Peak troponin (ng/ml)	7.9 ± 16
Peak CK-MB (ng/ml)	32.2 ± 61.2
Hemoglobin (g/dl)	13.9 ± 4.8
Creatinine (mg/dl)	0.9 ± 0.2

The dyslipidemia patterns and lipid profile characterization among the study population are shown in (Figs. 1–4).

Using the DLCN criteria, 97 patients (4.9 %) had probable/definite FH (PDFH) (score ≥6) and 434 (21.7 %) had possible FH (PFH) (score 3–5) (Fig. 5), while the SBR criteria identified 229 patients (11.5 %) with PFH and only 3 patients (0.2 %) with definite FH (Fig. 6).

Baseline demographics and clinical data of all patients with respect to FH diagnosis using DLCN criteria are shown in (Table 2).

Upon comparison between groups of FH as per the DLCN criteria, it could be noticed that patients in the PDFH group presented at a younger age (median; 50 years) compared to PFH and the unlikely group ($p < 0.001$). Similarly, in the PFH group, the age was younger (median; 60 years) compared to the unlikely group ($p < 0.001$). This reflects the premature presentation of ACS patients in the PDFH and the PFH group.

Comparing lipid profile values between the groups of the DLCN, in the PDFH group, the median serum cholesterol was 232.8 mg/dL (IQR 207.9–255.44 mg/dL), the median LDL-C level was 174 mg/dL (IQR 143–194.5 mg/dL) and the median corrected LDL-C level was 228.2 mg/dL (IQR 205.1–256.4 mg/dL). In the PFH group, the median serum cholesterol was 199.3 mg/dL (IQR 174.0–224.7 mg/dL), the median LDL-C level was 135.3 mg/dL (IQR 109.8–162.0 mg/dL) and the median

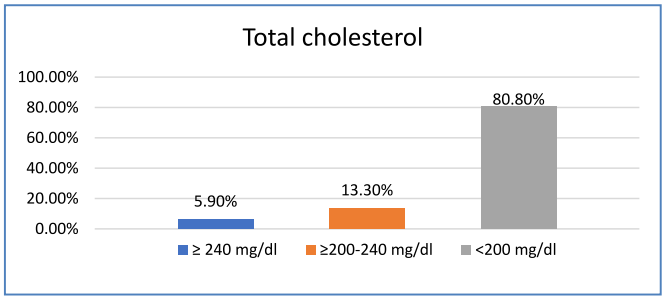

Fig. 1. Percentage of the patients according to different TC categories. TC, total cholesterol.

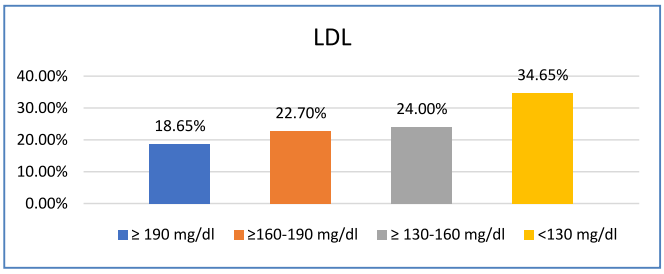

Fig. 2. Percentage of patients according to different LDL-C categories. LDL-C, low-density lipoprotein -cholesterol.

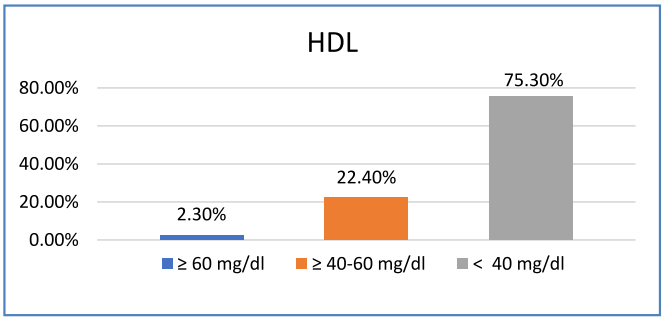

Fig. 3. Percentage of patients according to different HDL-C categories. DDL-C, high-density lipoprotein-cholesterol.

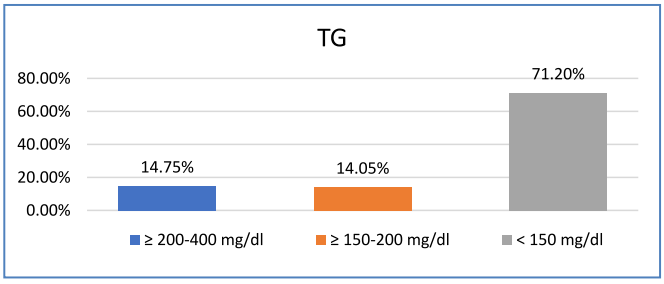

Fig. 4. Percentage of patients according to different TG categories. TG, triglycerides.

corrected LDL-C level was 195.9 mg/dL (IQR 175.2–219.3 mg/dL. These differences in the lipid profile between the PDFH and PFH groups were statistically significant ($p < 0.001$ for all).

Regarding the unlikely FH group, the median cholesterol level was 138.1 mg/dL (IQR 114.5–166.3 mg/dL), the median LDL-C level was 80.8 mg/dL (IQR 63.0–104.4 mg/dL), the median corrected LDL-C level was 134.2 mg/dL (IQR 98.6–158.2 mg/dL) and the median TG was 104.5 mg/dL (IQR 74.4–146.1 mg/dL). These results were significantly lower than those in the PDFH & the PFH groups ($p < 0.001$).

However, there was no statistically significant difference between the PDFH and PFH groups regarding the median serum TG level, which was 139.9 mg/dL (IQR 93.9–211.5) in the PDFH group and 133.3 mg/dL (88.6–196.6 mg/dL) in the PFH group. Both groups had significantly higher TG levels than the unlikely FH group ($p < 0.01$).

The HDL-C levels were 34.8 mg/dL (IQR 30.8–41.4 mg/dL) in the PDFH, 35.4 mg/dL (IQR 30.2–41.0 mg/dL) in the PFH group and 33.6 mg/dL (IQR 27.8–39.4 mg/dL) in the unlikely FH group. The differences between the PFH and the unlikely FH groups, as well as between the PDFH and the unlikely group were statically significant ($p < 0.01$).

Compared with patients without FH, patients with FH had higher proportions of prior angina ($p < 0.001$), MI ($p = 0.002$), percutaneous coronary intervention (PCI) ($p < 0.001$), family history of dyslipidemia ($p < 0.001$), premature CAD ($p < 0.001$) and prior statin intake ($p < 0.001$).

Diagnostic coronary angiography was not done in 282 patients mainly due to multiple comorbidities and/or frailty (213 patient), high bleeding risk (56 patients) or patient refusal (13 patients). An interventional strategy was more commonly used in the PDFH group than in the PFH and FH unlikely groups. Target vessel revascularization to the left anterior descending artery (LAD), left circumflex artery (LCX), and obtuse marginal arteries (OM) was also more common. Furthermore, these patients were more likely to be discharged on ezetimibe, evolocumab, or a combination of ezetimibe and evolocumab, along with high-intensity statin therapy (Table 3). On analysis of the data patients on prior statin therapy at presentation were less likely to present with STEMI and more likely to present with NSTEMI (Table 4).

4. Discussion

Knowing the dyslipidemia patterns in ACS patients is a critical issue in the management strategy. Our study examined the dyslipidemia patterns among 2000 patients hospitalized with ACS. The primary finding in this study was that FH is common in our ACS population. Based on the DLCN criteria, the prevalence of PDFH group reached 4.9 %, which equals about 1 in every 20 ACS patients, and this is 12 folds higher than the prevalence in the general population (0.4 % based on 1/250 prevalence) using a similar diagnostic algorithm. The prevalence of PFH was 21.7 %. In other words, FH could be suspected in 26.6 % (4.9 % of PDFH and 21.7 % of PFH) of patients with ACS. Numerous studies investigated FH in the context of ACS, with substantial variations in the reported prevalence.

Our results agree to a great extent with the results of recently published registries. One meta-analysis showed that the pooled prevalence of FH was 4.7 % (95 % CI 3.0–7.3 %) using random-effects methodology, and PDFH was 5.5 % (95 % CI 3.0–10.0 %) based on DLCN criteria [19]. Similar figures for PDFH prevalence were also observed in South European countries (Spain 4.1 %; Greece 3.8 %; France 4.4 %) [20]. Furthermore, the Gulf COAST registry, which was conducted in four Arabian Gulf countries and included 3224 ACS patients, found that the prevalence of PDFH was 3.7 % [21]. The investigators attributed their high prevalence of FH to the high rate of positive consanguinity among Gulf citizens, which inflated the prevalence of genetically determined conditions such as FH.

On the other hand, some registries showed a lower prevalence of FH compared to our study. For example, in the large French RICO survey that involved 11,624 patients hospitalized with acute MI, the prevalence of PDFH was 2.1 % [22]. Meanwhile, the prevalence of PDFH in the large Switzerland study, which included 4778 patients with ACS, was 1.6 % according to the DLCN algorithm [23]. Additionally, in a recent study from Denmark that included 13,174 patients with MI who were referred for coronary angiography, the prevalence of PDFH was 0.4 %, and PFH was 9.7 % based on the DLCN algorithm [24]. However, in the last two studies mentioned, the reported low prevalence of PDFH (1.6 % and 0.4 %, respectively) was probably because some of the aspects of the DLCN criteria (the presence of corneal arcus and tendon xanthomas or family history of premature CVD) were not recorded for the included patients, which likely resulted in an underestimate of the true prevalence of FH.

Despite the high prevalence of FH in our study population, other studies reported a higher prevalence. In an Australian study [25], the prevalence of probable FH in patients with early-onset CAD reached 14.3 %, and definite FH was identified in 2.3 % of the study participants. This could be explained by the more extensive use of statins (68 % vs. 26 %) and the more prominent correction factor (2.0) used to adjust LDL-C in individuals on statins. In the EUROASPIRE IV study, which included 7000 patients hospitalized for ACS or revascularization procedures, the prevalence of FH was estimated to be 8.3 %, with wide variations across different countries, which could be explained by the difference in

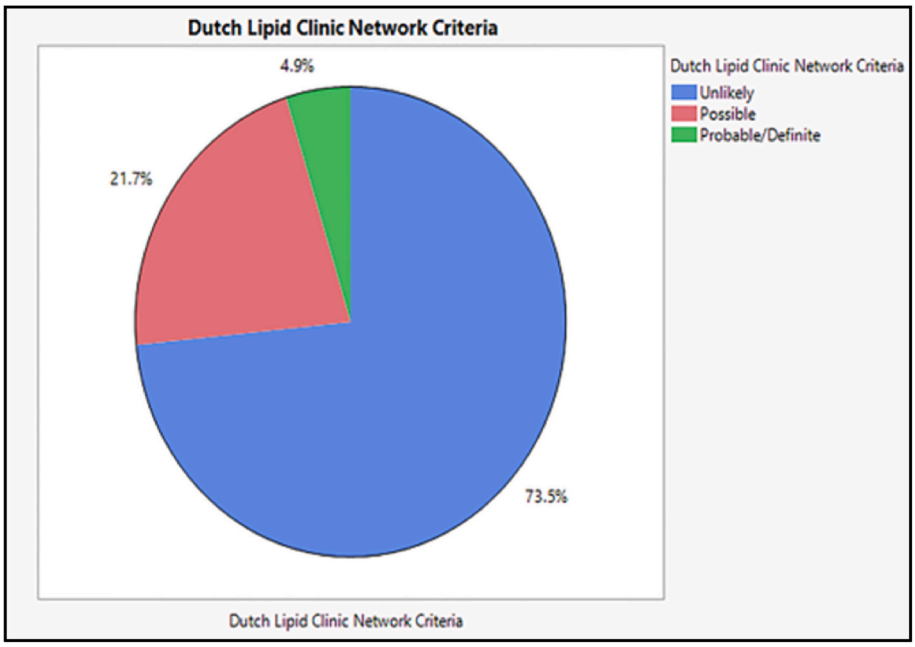

Fig. 5. Patient classification according to the Dutch Lipid Clinic Network (DLCN) Criteria.

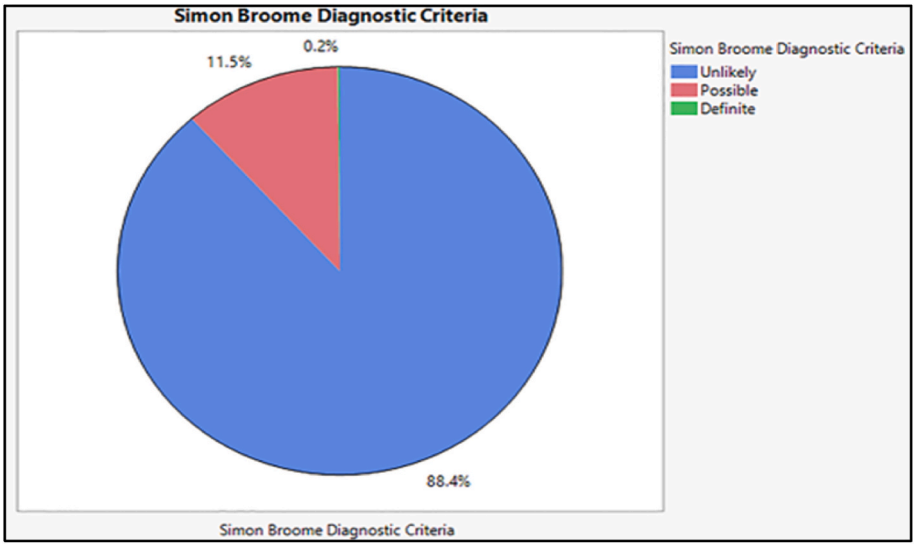

Fig. 6. Patient classification according to the Simon Broome Diagnostic Criteria.

recruitment methodology [20]. Despite this difference between studies, the prevalence of FH in ACS patients is several-fold higher than in the general population.

The excess CV risk conferred by FH is thought to be primarily related to premature CV events. Our cohort found a higher prevalence of premature ACS in the PDFH and PFH groups than in the FH unlikely group. Previous data supported this finding. In Switzerland's (SPMM - ACS) study, the prevalence of PDFH was 4.8 % among 1451 premature ACS patients using the same diagnostic criteria [23].

Furthermore, there was a significant increase in the number of premature ACS patients with both a family history of premature CVD and high LDL-C (≥160 mg/dL) in the PDFH group. Similarly, a recently published study from the USA involving 1996 young patients (under the age of 50 years) with MI reported that the prevalence of FH among patients with a family history of premature CVD was approximately 2 in 10 patients, while in those with LDL-C ≥ 160 mg/dL, the prevalence was around 3 in 10 patients. The prevalence reached 6 in 10 patients if they had both risk factors [26]. From these cumulative results, we can conclude that the risk of FH occurrence should always be considered among premature ACS patients with a family history of premature ACS or LDL-C≥ 160 mg/dL. This was also seen in our study were the prevalence of FH was significantly higher in premature ACS patients with a family history of premature CAD and elevated LDL-C levels in both the PFH and PDFH groups.

It was also observed that FH is more common in males than in females. This gender difference could be attributed to the higher representation of males in the overall cohort, as male gender is generally a risk factor for CAD. Furthermore, in a meta-analysis of 12 studies examining the prevalence of FH in ACS patients, the number of males was approximately 3-fold higher than that of females. In our study, male patients were also about 3 times more prevalent than female patients [19].

Regarding our cohort characteristics, we observed several differences from previous cohorts, primarily a younger age at presentation,

Table 2
Baseline demographic, biochemical & clinical characteristics of all patients with respect to FH diagnosis using DLCN criteria.

	Unlikely FH	PFH	PDFH	P value
Number	1469	434	97	
Percentage	73.5 %	21.7 %	4.9 %	
Demographic				
Age, median (years) (IQR)	67.0 (58.0–77.0)	61.0 (50.0–72.0)	50.0 (46.0–54.0)	**<0.001**
Gender				0.291
Male n (%)	1122 (76.4 %)	321 (74.0 %)	85 (87.6 %)	
Female n (%)	347 (23.6 %)	113 (26 %)	12 (12.4 %)	
Comorbidities n (%)				
Smoking	209 (14.2 %)	89 (20.5 %)	39 (40.2 %)	**<0.001**
Family history of dyslipidemia & premature CAD	101 (6.9 %)	50 (11.5 %)	29 (29.9 %)	**<0.001**
DM	976 (66.4 %)	303 (69.8 %)	51 (52.6 %)	0.285
Hypertension	1016 (69.2 %)	310 (71.4 %)	52 (53.6)	0.106
Dyslipidemia	748 (50.9 %)	268 (61.8 %)	62 (63.9 %)	**<0.001**
PVD	32 (2.2 %)	13 (3.0 %)	1 (1 %)	0.882
CVS	99 (6.7 %)	29 (6.7 %)	7 (7.2 %)	0.923
Statin intake	769 (52.3 %)	318 (73.3 %)	69 ((71.1 %)	**<0.001**
IHD:				
MI	208 (14.2 %)	66 (15.2 %)	24 (24.7 %)	**0.022**
Angina	357 (24.3 %)	124 (28.6 %)	38 (39.2 %)	**0.001**
PCI	288 (19.6 %)	112 (25.8 %)	37 (38.1 %)	**<0.001**
CABG	92 (6.3 %)	18 (4.1 %)	5 (5.2 %)	0.164
Biochemical ; median (mg/dl) (IQR)				
Cholesterol	138.1 (114.5–166.3)	199.3 ‡ (174.0 to 224.7)	232.8 ‡ § (207.9 to 255.44)	**<0.001**
LDL-C	80.8 (63.0–104.4)	135.3 ‡ (109.8–162.0)	174.0 ‡ § (143.0to 194.5)	**<0.001**
Corrected LDL-C	134.2 (98.6–158.2)	195.9 ‡ (175.2–219.3)	228.2 ‡ § (205.1 to 256.4)	**<0.001**
HDL	33.6 (27.8–39.4)	35.4 ‡ (30.2–41.0)	34.8 ‡ (30.8–41.4)	**<0.001**
TG	104.5 (74.4–146.1)	133.3 ‡ (88.6–196.6)	139.9 § (93.9–211.5)	**<0.001**
Other Biochemical ; median (IQR)				
Peak troponin (ng/ml)	0.97 (0.04–6.80)	1.20 (0.04–8.82)	2.00 (0.04–12.45)	**0.290**
Peak CK-MB (ng/ml)	7.0 (2.1–26.0)	8.3 (2.2–39.0)	12.2 (2.1–77.4)	**0.214**
Hemoglobin (g/dl)	13.7 (12.2–15.0)	13.9 (12.4–15.0)	**14.9** ‡§ (13.2–15.8)	**<0.001**
Creatinine (mg/dl)	0.95 (0.79–1.06)	0.89 (0.76–1.04)	0.88 (0.79–1.01)	**0.418**

† Jonckheere-Terpstra trend test. ‡ Statistically significant difference versus Unlikely Class at P < 0.01 b y Conover post hoc test.
§Statistically significant difference versus Possible Class at P < 0.01 b y Conover post hoc test.
IQR; interquartile range; PAD, peripheral arterial disease; CVS, cardiovascular disease; MI, myocardial infarction; PCI, percutaneous coronary intervention; CABG, coronary artery bypass graft; LDL-C, low density lipoprotein; HDL-C, high density lipoprotein; TG, triglyceride; UA, unstable angina; STEMI, ST elevation myocardial infarction; NSTEMI, non-ST segment elevation myocardial infarction; n, number.

with a mean age of 64.4 years with a high prevalence of younger age in the PFH and PDFH groups compared to the FH unlikely group. Similarly, Indian ACS patients had a mean age of 57 [20,23].

Furthermore, our population exhibited a higher prevalence of atherosclerotic CVD risk factors such as HTN (68.9 %), DM (66.5 %), and smoking (16.9 %). The elevated prevalence of these risk factors underscores the endemic nature of the problem. The Arabian Gulf ACS registry (Gulf RACE) also reported a high prevalence of various comorbidities in the Gulf region [27].

Our study showed that patients with FH had a more severe clinical presentation of CAD compared to non-FH patients, as they were more likely to have a prior MI, PCI, angina, dyslipidemia, a family history of premature CAD, despite the use of statin therapy before admission. This reflects the high cumulative LDL-C burden associated with FH.

Also, Lipid-lowering therapy with statins significantly alters plaque morphology within the coronary arteries, potentially influencing the ratio of STEMI to non-STEMI events. In our study, we observed that patients who were on statin therapy prior to the index event exhibited a higher tendency to present with non-STEMI compared to STEMI in cases of myocardial infarction. These results align with previously published research [28].

Our findings highlight the importance of raising awareness among physicians regarding the necessity of early screening for FH in patients at high CV risk and particularly those presenting with ACS. This screening can be conducted using well-validated clinical criteria, such as the DLCN and the SBR, which can be implemented at a low cost and without the need for genetic testing. Moreover, it is crucial to maintain a high level of suspicion for FH when managing younger individuals with ACS. Patients diagnosed with FH should receive optimal management, which may include coronary revascularization, if necessary, in addition to medical therapy including high-intensity statins, ezetimibe and/or PCSK9 inhibitors, as indicated. Furthermore, family members of those identified with FH should also undergo screening.

4.1. *Limitations*

Genetic testing was not performed to identify monogenic mutations associated with FH. The detection rate for monogenic disorders is approximately 25 % among patients diagnosed with PFH and about 75 % in patients with PDFH. Therefore, our estimates should not be directly compared to studies where FH was confirmed through genetic testing. Additionally, calculating LDL levels in patients on regular statins might have led to a slight overestimation of LDL-C levels, and this approach also does not account for individual variability in treatment response. Furthermore, lipid parameters may significantly vary within the first 24 h after ACS potentially impacting the accuracy of the results. In the current study, we did not require a specific time for blood sample withdrawal for the lipid profile following admission apart from being withdrawn within the first 24 h in accordance with the hospital protocol and in line with the ESC guidelines [11,29,30].

5. Conclusions

A clinical diagnosis of PDFH, PFH, and dyslipidemia is common among patients hospitalized with ACS, particularly those with premature ACS. A high degree of suspicion for FH should be maintained in any young individual hospitalized with ACS.

Ethical considerations

The study protocol was approved by both the cardiovascular departments and the local Research Ethical Committees at Kasr Al-Ainy University Hospital and Prince Khaled Ben Sultan Cardiac Center, Abha, Saudi Arabia. Patients were only enrolled after obtaining written informed consent in a private space respecting patients' privacy. Moreover, patients' data was confidential and there was no mention of their names or identity. The authors are solely responsible for the design and conduct of this study, all study analyses, the drafting and editing of the paper, and its final contents.

Funding

None.

Table 3
Baseline medications and management of patients with respect to FH diagnosis using DLCN criteria.

Variable	Unlikely (N = 1469)		Possible (N = 434)		Probable/Definite (N = 97)		P value[a]
	N	%	N	%	N	%	
In-hospital medications							
Aspirin	1469	100.0 %	434	100.0 %	97	100.0 %	NC
P2Y12 inhibitor	1469	100.0 %	434	100.0 %	97	100.0 %	NC
Statin	1467	99.9 %	433	99.8 %	97	100.0 %	0.952
Antihypertensive	1072	73.0 %	329	75.8 %	65	67.0 %	0.905
Management							
Conservative	416	28.3 %	112	25.8 %	14	14.4 %	**0.007**
Interventional	1053	71.7 %	322	74.2 %	83	85.6 %	
Reason for medical treatment							
No coronary angiography	215	51.7 %	60	53.6 %	7	50.0 %	0.736
Normal coronary angiography	89	21.4 %	25	22.3 %	2	14.3 %	
Insignificant atherosclerotic CAD	92	22.1 %	16	14.3 %	4	28.6 %	
Diffuse MVD	2	0.5 %	6	5.4 %	0	0.0 %	
Slow flow	18	4.3 %	5	4.5 %	1	7.1 %	
Interventions							
Stent implantation	893	60.8 %	283	65.2 %	63	64.9 %	0.100
Balloon dilatation	16	1.1 %	2	0.5 %	1	1.0 %	0.418
CABG	150	10.2 %	39	9.0 %	20	20.6 %	0.080
Target vessel							
LM	67	4.6 %	16	3.7 %	2	2.1 %	0.185
LAD	703	47.9 %	224	51.6 %	55	56.7 %	**0.040**
Diagonal branch	91	6.2 %	24	5.5 %	12	12.4 %	0.183
Ramus	23	1.6 %	6	1.4 %	0	0.0 %	0.299
LCX	155	10.6 %	55	12.7 %	16	16.5 %	**0.043**
OM	168	11.4 %	47	10.8 %	24	24.7 %	**0.014**
RCA	352	24.0 %	95	21.9 %	31	32.0 %	0.517
LIMA graft	8	0.5 %	3	0.7 %	0	0.0 %	0.806
Pathological Q on discharge	396	27.0 %	112	25.8 %	31	32.0 %	0.668
Medications on discharge							
Aspirin	1412	96.1 %	419	96.5 %	97	100.0 %	0.102
P2Y12 inhibitor	1369	93.2 %	407	93.8 %	92	94.8 %	0.474
Statin	1457	99.2 %	434	100.0 %	97	100.0 %	0.051
High-dose statin	1370	93.3 %	406	93.5 %	92	94.8 %	0.579
Ezetimibe	95	6.5 %	44	10.1 %	11	11.3 %	**0.004**
Evolocumab	4	0.3 %	3	0.7 %	15	15.5 %	**<0.001**
Ezetimibe + Evolocumab	1	0.1 %	5	1.2 %	3	3.1 %	**<0.001**
OAC	34	2.3 %	14	3.2 %	3	3.1 %	0.312
NOAC	130	8.8 %	31	7.1 %	6	6.2 %	0.172
Antihypertensive	1213	82.6 %	339	78.1 %	76	78.4 %	**0.038**

N, number; CABG, coronary artery bypass graft; CAD, coronary artery disease; MVD, muti-vessel disease; LM, left main vessel; LAD, left anterior descending artery; LCX, left circumflex artery; OM, obtuse marginal branch; RCA, right coronary artery; LIMA, left internal mammary artery; OAC, oral anticoagulant; NOAC, new oral anticoagulant.

[a] Chi-squared test for trend.

Table 4
Age, pattern of presentation, and LDL & corrected LDL levels in patients with or without prior statin intake.

	No prior statin intake (N = 844)	Prior statin intake (N = 1156)	P value[a]
Age (years)	62 (51–73)	67 (57–77)	**<0.0001**
Sex			0.510[b]
Female	193 (22.9 %)	279 (24.1 %)	
Male	651 (77.1 %)	877 (75.9 %)	
Presentation			
Unstable angina	199 (23.6 %)	322 (27.9 %)	0.031[b]
STEMI	296 (35.1 %)	261 (22.6 %)	<0.001[b]
NSTEMI	349 (41.4 %)	573 (49.6 %)	<0.001[b]
LDL (mg/dl)	167.1 (77.3–138.8)	85.1 (65.7–113.7)	<0.0001
Corrected LDL (mg/dl)	107.3 (77.3–138.8)	168.6 (147.7–196.8)	<0.0001

Data are median (interquartile range) or number (%).
IQR, interquartile range; LDL-C, low density lipoprotein; HDL-C, high density lipoprotein; TG, triglyceride; UA, unstable angina; STEMI, ST elevation myocardial infarction; NSTEMI, non-ST segment elevation myocardial infarction; n, number.

[a] Mann-Whitney test unless otherwise indicated.

[b] Pearson chi-squared test.

Declaration of interests

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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