Impact of obesity on low density lipoprotein plasmatic levels 6 weeks after an acute coronary syndrome

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Introduction **Cardiovascular disease is the leading cause of death. One of the main factors is obesity, which is on the rise. LDL levels below 0.55 g/L are recommended after ACS. To date, there are no specific recommendations for obese subjects.**

Objective **The primary objective was to assess the impact of obesity on LDL-c target attainment 6 weeks after initiation of statin therapy in subjects admitted for ACS. The secondary objectives were to assess the evolution of cholesterol levels and to characterize lipid-lowering treatments.**

Methods **The single-center observational study took place at Montpellier University Hospital and included patients admitted to the ICU for ACS not treated with statins (T0). Biological tests were performed at 6 weeks. At 3 months, a telephone call was made by two pharmacists to collect the results of their biological work-up and any therapeutic modifications.**

Results **The results were analyzed on 286 patients. A total of 39.5% were overweight and 22.7% obese. After hospitalization, 95.4% were prescribed statins. At 6 weeks, LDL cholesterol averaged 1.58 mmol/L, lower in subjects with a BMI greater than 30 kg/m² (1.32 mmol/L). On average, 49.46% of subjects reached the LDL cholesterol target, with obese subjects achieving a higher rate of 64.9%. There was no significant difference in the prescription of lipid-lowering treatments between the two groups.**

Conclusion **Despite high-intensity statin prescription, the target was achieved by 67.6% of obese subjects and 44.8% of subjects with a BMI of less than 30 kg/m². This shows that post-SCA management needs to be reinforced for the general population.** ***Cardiovasc Endocrinol Metab*** **14: 1–7 Copyright © 2024 The Author(s). Published by Wolters Kluwer Health, Inc.**

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Introduction

According to the WHO, cardiovascular disease is the leading cause of death worldwide, and acute coronary syndrome (ACS) is its main manifestation [1].

Among cardiovascular risk factors, obesity and its complications play a key role. Furthermore, 39% of adults globally are overweight and 13% are obese. This prevalence is expected to rise to 22% by 2045 [2]. In the USA, obesity accounts for 11% of heart disease in men and 14% in women [3]. Additionally, 50% of subjects suffering from ACS are overweight and/or obese [4,5].

Obesity induces volume overload resulting in impaired left ventricular systolic and diastolic function [6]. In addition to the changes in cardiac structure and function induced by obesity, the increased risk of coronary heart disease is also explained by cardiometabolic disorders such as hypertension, dyslipidemia, and type 2 diabetes [7].

According to Mahmood *et al*. [8], women suffering from obesity are five times more likely to die from coronary heart disease than nonobese women, and the risk of coronary heart disease increases by 3.1% for every additional kilogram of body weight. Similarly, a 10% increase in body weight is associated with a 13% increase in coronary heart disease risk in men [8]. In addition, a meta-analysis of around 20 studies involving more than 300 000 people showed a 30% increase in the risk of developing ischemic diseases for each 5-unit increase in BMI [9].

The latest European Society of Cardiology (ESC) guidelines set a low-density lipoprotein (LDL) target of less

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than 0.55 g/L (1.42 mmol/L) for patients following ACS. The initial strategy is based on high-intensity statin treatment prescribed to reach the LDL goal. It is recommended to combine ezetimibe if LDL goal is not achieved [10].

However, specific data on the efficacy of statin treatment and/or reaching LDL target regarding BMI are scarce.

However, this is an important practical question, given the increasing prevalence of obesity. Further obesity may induce a change in the pharmacokinetics of treatments. Indeed, obesity has an impact on both digestive transit, which is accelerated, and on gastric emptying time, which is shortened. These modifications could lead to a decrease in solubilization and absorption of oral medications. Further, drug distribution may be affected during obesity as body fat leads to a significant increase in the volume of distribution of lipophilic drugs. These changes may lead to drug dose adjustments in subjects suffering from obesity [11]

Surprisingly, there are no specific data on the impact of obesity on statin efficacy. To the best of our knowledge, the only data found in the literature are indirect answers to this question. Based on two Canadian prospective observational cohorts, Bhan *et al*. [12] highlight that the percentage of subjects with a history of cardiovascular disease or with diabetes reaching the LDL cholesterol (LDL-c) target was significantly lower in subjects suffering from obesity compared with nonobese subjects (45% in subjects suffering from obesity vs. 48.3% in normal-weight subjects).

Furthermore, the DYSIS study, an international observational, multicenter study carried out in patients aged 45 or over, taking statins, found that subjects suffering from obesity required higher dose of statins and more frequent co-prescription of ezetimibe to reach the LDL target [13].

Therefore, the main objective of this observational study was to assess the impact of obesity on the LDL-c target achievement rate 6 weeks after initiation of statin in subjects admitted for ACS.

The secondary objectives were to (1) assess the change in LDL-c levels between inclusion and after 6 weeks treatment regarding BMI and (2) characterize the lipid-lowering treatments in terms of type of molecule, dosage, and dose at discharge from hospital, at T6 weeks and at M3 according to BMI.

Materials and methods

The detailed method had already been described [14]. We conducted a monocentric prospective study in the ICCU (Intensive Cardiology Care Unit) of the Montpellier University Hospital.

We included adult patients, who were admitted to ICCU for ACS (defined by ST-segment elevation myocardial infarction or non-ST-segment elevation myocardial infarction or unstable angina).

Patients previously treated with a statin at the time of admission to the ICCU were not included, neither those with statin intolerance nor patients under guardianship or curatorship.

Unfolding of the study

The study was carried out in three stages: T0, T6 weeks, and T3 months.

T0 represented the inclusion period, corresponding to the admission of subjects to the university hospital for ACS. At that time, patients were provided with an information letter about the study.

Upon hospital discharge, patients received a prescription for their various treatments, as well as a prescription for a biological checkup including the lipid profile at six weeks in accordance with European guidelines [10]. T6 weeks corresponded to the sixth week after inclusion and, therefore, the time to perform lipid profile. T3 months corresponded to the telephone interview carried out by two pharmacists to collect data relating to the blood test and therapeutic modifications since discharge (Supplementary Figure S1, Supplemental digital content 1, *http://links.lww.com/CAEN/A65*).

Study outcomes

The primary endpoint was the percentage of nonobese and subjects suffering from obesity achieving the LDL-c target at T6 weeks after the introduction of statin.

Achievement of LDL-c targets was assessed following the ESC 2023 guidelines for patients at very high cardiovascular risk (secondary prevention, LDL-c < 0.42 mmol/L).

The secondary endpoints were as follows:

(1) The change in LDL-c (in %) between hospital admission and T6 weeks treatment.
(2) Assessment of statin treatment persistence at M3 after ACS according to obesity.
(3) Assessment of different lipid-lowering treatments at M3 according to obesity.

Data collection

Various data were collected at inclusion: demographic data (age and sex); clinical data (weight, BMI, blood pressure, and medical history); biological data (lipid profile, liver profile, and blood sugar); patient's level of education; and calculation of EPICES score (evaluation of precariousness and health inequalities in health examination center) is an individual indicator of precariousness which takes into account the multidimensional nature of precariousness. The EPICES score consists of 11 questions. The answer to each question is assigned a coefficient,

and the sum of the 11 answers gives the EPICES score. The score is continuous and varies from 0 (no precariousness) to 100 (maximum precariousness).

Six weeks after inclusion, the LDL-c value was collected to evaluate the effectiveness of the statin.

Patients were considered lost to follow-up if there was no response after three consecutive calls (including voicemail) or an e-mail when an e-mail address was available in the patient file.

Ethical procedure

The study was approved by the local ethics committee (IRB ID: 202100815 15/04/2021).

A letter of information was given to the patient, and their nonopposition was collected as recommended by the French authorities. Subject data were anonymized to ensure confidentiality.

Statistical analysis

The data were analyzed by the Montpellier University Hospital data management center (Medical Information Department, Montpellier University Hospital). Qualitative variables were described using the number (percentage) and were compared using the chi-square test or Fisher's exact test. Quantitative variables were described in terms of mean ± SD and were compared between groups using a Student's *t*-test if normality was respected, otherwise using a nonparametric test (Wilcoxon Mann–Whitney test).

Factors likely to influence the achievement of the LDL-c target were studied using a conventional logistic regression model.

A linear regression model was used to study the factors that could influence the variation in LDL-c levels between M3 and admission to hospital.

All variables with a *P*-value <0.20 in univariate analysis were selected for multivariate analysis. A *P*-value ≤0.05 was considered statistically significant in the analysis.

All analyses were performed using SAS software (version 9.04; SAS Institute, Cary, North Carolina, USA).

Results

Our study was conducted between 15 November 2021 and 18 February 2023. Five hundred forty patients were screened; 254 subjects were excluded; 16 died, 193 were lost to follow-up, 45 refused to participate, and three had missing data (weight). The results were therefore analyzed on 286 subjects (Supplementary Figure S2, Supplemental digital content 1, *http://links.lww.com/CAEN/A65*).

Population description

A total of 77.97% of the population were men, with an average age of 64 years. The average BMI was 26.9 kg/m^2.

Of the 286 subjects, 113 were overweight (39.5%) and 65 subjects from suffering obesity (22.7%). The three main comorbidities found were hypertension (38.1%) and diabetes (16%).

The average EPICES score was 20.4, higher in the population suffering from obesity (25.5) ($P = 0.01$). All the characteristics of the subjects according to BMI are described in Table 1.

History and comorbidities

Biological work-up at T0

On hospital admission, there was no significant difference in plasma LDL-c level, although it tends to be slightly higher. Triglyceride levels were significantly higher in subjects suffering from obesity (4.08 mmol/L) ($P < 0.01$) than in those with a BMI of less than 30 kg/m^2 (3.54 mmol/L) (Table 2).

Table 1 **Baseline characteristics of the population according to BMI**

Characteristics			BMI (kg/m^2)		
		Total population	<30	≥30	
Variable		*N* = 286	*N* = 221	*N* = 65	*P* value[a]
Sex	Woman	63 (22.03)	45 (20.36)	18 (27.69)	0.21
	Men	223 (77.97)	176 (79.64)	47 (72.31)	
Age on admission (year)	Mean(±SD)	64.12 (±11.71)	63.92 (±11.94)	64.81 (±10.95)	0.59
BMI (kg/m^2)	Mean (±SD)	26.9 (±4.68)	24.98 (±2.85)	33.54 (±3.46)	<0.01
Retired	Yes	143 (57.20)	54 (55.10)	30 (51.72)	0.34
EPICES score	Mean (±SD)	20.4 (±17.88)	18.8(±17.03)	25.5 (±19.67)	<0.01
Medical history and comorbidities	Hypertension, *n* (%)	109 (38.11)	78 (35.29))	31 (47.69)	0.07
	Diabetes, *n* (%)	46 (16.08)	31 (14.03)	15 (23.08)	0.08
	Prior ischemic cardiomyopathy (ACS, Angina), *n* (%)	24 (8.39)	19 (8.60)	5 (7.69)	0.82
	Smoking, *n* (%)	122 (42.66)	95 (42.99)	27 (41.54)	0.84
	Smoking status, *n* (%)	93 (37.20)	71 (36.79)	22 (38.60)	0.79
	Smoking Weaned assets	52 (20.80)	42 (21.76)	10 (17.54)	
	Alcohol, *n* (%)	21 (7.34)	18 (8.14))	3 (4.62)	0.43

Data are expressed as number (%) or mean ± SD.

EPICES, Evaluation of Deprivation and Inequalities in Health Examination Centers.

[a]*P* value: BMI < 30 kg/m^2 vs. BMI > 30 kg/m^2.

Table 2 Biological assessment at admission (T0) and at 6 weeks postacute coronary syndrome according to BMI

Variable	Total population	BMI (kg/m²)		*P* value[a]
		<30	≥30	
T0, LDL (mmol/L)	3.57 (±1.14)	3.54 (±1.09)	3.70 (±1.14)	0.21
T6 weeks, LDL (mmol/L)	1.58 (±0.78)	1.63 (±0.80)	1.32 (±0.54)	0.03
T0, HDL-c (mmol/L)	1.24 (±0.36)	1.24 (±0.36)	1.19 (±0.39)	0.12
T6 weeks, HDL-c (mmol/L)	1.16 (±0.36)	1.19 (±0.38)	1.14 (±0.31)	0.51
T0, TG (mmol/L)	3.67 (±2.40)	3.54(±3.03)	4.08 (±1.99)	<0.01
T6 weeks, TG (mmol/L)	2.79 (±1.14)	2.76 (±1.12)	2.90 (±1.24)	0.58
T0, ASAT (UI/L)	92.26 (±111.84)	92.95 (±116.05)	90.00 (±97.49)	0.79
T6 weeks, ASAT (UI/L)	25.67 (±12.24)	24.56 (±12.42)	31.00 (±10.30)	0.04
T0, ALAT (UI/L)	38.70 (±74.41)	39.77 (±84.22)	35.13 (±19.11)	0.03
T6 weeks, ALAT (UI/L)	31.05 (±25.10)	29.92 (±26.33)	36.50 (±18.18)	0.13

[a]*P* value: BMI < 30 kg/m² vs. BMI > 30 kg/m².
ALAT, alanin aminotransferase; ASAT, aspartate aminiotransferase; HDL, high-density lipoprotein; LDL, low-density lipoprotein; TG, triglycérides.

Treatment on discharge

A total of 95.4% of the patients received statin treatment at hospital discharge.

There was no difference in statin potency between people with and without obesity. A total of 97% ($P = 0.75$) of subjects received a high-intensity statin, independently of BMI (BMI < 30: 97.06% and BMI > 30: 96.83%).

A total of 43.2% of the population received a prescription for dual therapy with Ezetimibe; this combination was slightly higher in the subjects suffering from obesity (49.2%) than in the population with a BMI < 30 kg/m² (41.4%) ($P = 0.27$).

Biological tests at 6 weeks postacute coronary syndrome

One hundred eighty-two subjects underwent their biological control at 6 weeks. The mean LDL-c level for 6 weeks was 1.58 mmol/L.

This level was lower in the subjects from suffering obesity 1.32 mmol/L than in the population with a BMI < 30 kg/m² ($P = 0.03$). ASAT levels were significantly higher in the population suffering from obesity (31 vs. 24.6 IU/L; $P = 0.04$). There was no difference in high-density lipoprotein (HDL) or triglyceride levels, according to BMI (Table 3).

Achievement of low-density lipoprotein cholesterol target

Less than half of the subjects reached the LDL-c target (49.46%).

Among people suffering from obesity, 64.9% achieved the target LDL-c level, compared with 44.8% for the others ($P = 0.03$) (Table 3).

Between T0 and T6 week, there was an average 76.3% reduction in baseline LDL-c levels, with a tendency to be higher in subjects suffering from obesity, although the difference did not reach significance (–73.6 vs. –86.4%; $P = 0.16$) (Fig. 1).

Treatment with dual therapy lead to a decrease in LDL-c levels superior than the one observed with statin treatment in all groups, independently of BMI.

Predictive factors associated with low-density lipoprotein cholesterol target reaching

Predictive factors associated with reaching the LDL target, in univariate and multivariate analysis (Supplementary Table S1, Supplemental digital content 1, *http://links.lww.com/CAEN/A65*).

In univariate analysis, there was an association between HDL, high blood pressure, and BMI on the risk of not reaching the LDL target. For the multivariate model, only the variables HDL at M3 and hypertension [odds ratio (OR): 0.312; $P = 0.0020$] were significant.

An increase of 0.10 in HDL-c increases the risk of not reaching the LDL-c target.

Treatment at 3 months postacute coronary syndrome

At M3, 249 patients responded to the telephone interview. Of these patients, 97.8% were still being treated with statins. The proportion of patients receiving a statin at 3 months did not differ between those with a BMI > 30 and the others (100% vs. 97.4; $P = 0.34$). Similarly, there was no significant difference between these two groups in terms of statin potency (Supplementary Figure S3, Supplemental digital content 1, *http://links.lww.com/CAEN/A65*). Thus, 93.3% of patients suffering from obesity were on high-intensity statins vs. 96.8% of the others ($P = 0.14$) (Fig. 2). Dual therapy with ezetimibe was slightly more frequent in patients with a BMI > 30, although this difference was NS (41.6 vs. 36.7%; $P = 0.47$) (Fig. 2).

Discussion

This study highlighted the impact of obesity on LDL levels 3 months after the introduction of a statin following ACS in a cohort of 286 subjects admitted to UCI. We found that patients with BMI greater than 30 kg/m² seemed more likely to achieve their LDL-c target (64.9 vs. 44.8%).

In our study, the proportion of subjects suffering from obesity (22.7%) was lower than in other studies, such as that of Hao *et al.* [15] where the prevalence was 37.4%, or in Martín-Castellanos *et al.* [16] study, where it was 31.4%.

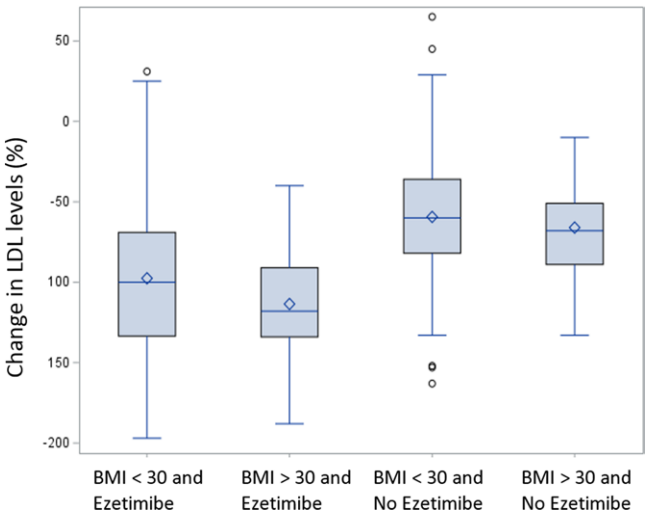
Although our sample is relatively small, it seems representative of the French population, with sociodemographic characteristics similar to those found in the registries. Indeed, the risk factors observed were also

Table 3 Description of low-density lipoprotein cholesterol target attainment at 6 weeks

Variable		Population	Achievement of LDL cholesterol target at 6 Weeks		*P* value
			No	Yes	
Sex, *n* (%)	Women	44 (23.91)	24 (25.81)	20 (21.98)	0.54
	Men	140 (76.09)	69 (74.19)	71 (78.02)	
Age at entry (year)	Mean (±SD)	65.08 (±12.10)	65.17 (±11.65)	64.98 (±12.60)	0.91
Restated, *n* (%)	No	58 (38.67)	32 (43.24)	26 (34.21)	0.26
	Yes	92 (61.3)	42 (56.76)	50 (65.79)	
EPICES Score	Mean (±SD)	21.37 (±19.02)	20.53 (±19.38)	22.20 (±18.75)	0.48
BMI (kg/m²)	Mean (±SD)	26.67 (±4.67)	26.05 (±4.00)	27.32 (±5.22)	0.12
BMI, *n* (%)	<30	145 (79.67)	80 (86.02)	65 (73.03)	0.03
	≥30	37 (20.33)	13 (13.98)	24 (26.97)	
Retail BMI, *n* (%)	<18.5	3 (1.65)	2 (2.15)	1 (1.12)	0.09
	(18.5–24.9)	70 (38.46)	39 (41.94)	31 (34.83)	
	(25–29.9)	72 (39.56)	39 (41.94)	33 (37.08)	
	<30	145 (79.67)	80 (86.02)	65 (73.03)	
	≥30	37 (20.33)	13 (13.98)	24 (26.97)	
	(30–35)	27 (14.84)	11 (11.83)	16 (17.98)	
	(35–40)	8 (4.40)	2 (2.15)	6 (6.74)	
	≥40	2 (1.10)	0 (0.00)	2 (2.25)	

EPICES, Evaluation of Deprivation and Inequalities in Health Examination Centers.

Fig. 1



Change in LDL levels (%) between hospital admission and M3. LDL, low-density lipoprotein.

similar to those collected in the French Registry of Acute ST-Elevation or Non-ST-Elevation Myocardial Infarction (FAST-MI) registry: arterial hypertension (53%), diabetes (22%), and active smoking (35%). In our study, these three risk factors were arterial hypertension (38%), diabetes (16%), and active smoking (42%) [17]. Tobacco consumption is quite high, with 42% of the population actively smoking. Cessation of smoking is considered one of the most effective measures, associated with a 36% reduction in mortality after myocardial infarction [18].

Triglyceride levels were significantly higher in the study population with a BMI over 30 kg/m² (4,08 mmol/L vs 3,54 mmol/L). These results are consistent with the DYSIS study, an international, observational, multicenter study of patients aged 45 or overtaking statins, which showed that subjects with a high BMI had higher plasma concentrations of total cholesterol and triglycerides [13].

The overall aim of the study was to assess the percentage of nonobese and subjects suffering from obesity achieving the LDL-c target. Six weeks after the ACS, 49.5% of patients in our study had reached the LDL-c target defined by European guidelines.

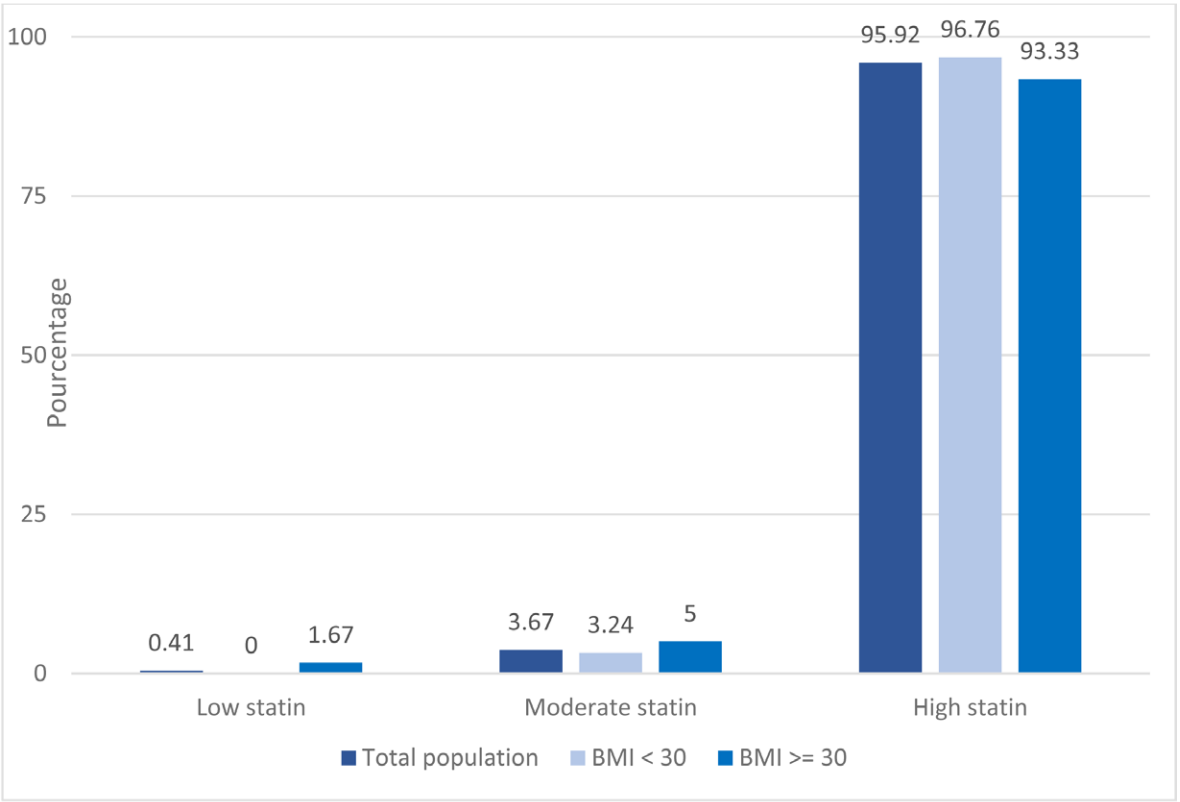
A high BMI does not appear to be a risk factor in not achieving the LDL target. Indeed, 64.9% of patients suffering from obesity were at target, compared with 44.8% of those with a BMI < 30 kg/m². This may be due to better compliance with treatment or better follow-up, as patients suffering from obesity often have other associated complications.

Further, this greater LDL-c target attainment in subjects suffering from obesity than in subjects with a BMI < 30 kg/m² in our study could be explained by a different baseline LDL level between the two groups, but this was higher at T0 (3.70 mmol/L vs. 3.54 mmol/L), so there is indeed a greater reduction in LDL-c –86.40 vs. 73.60% for subjects suffering from obesity.

Another explanation for this difference could be due to a difference in initial statin dose or the use of dual therapy between the two groups, but we found no significant difference. Indeed, at inclusion, 97% of subjects received a prescription for statins and 43.23% of the population for Ezetimibe treatment; the use of this treatment was slightly higher in the population suffering from obesity (49.2%). Moreover, treatment persistence was similar between the two groups at 3 months, with 97.99% of the total population being treated with a statin, most often a high-intensity statin (95.9%).

However, in two other studies, there was a difference in treatment between subjects suffering from obesity and those with a BMI < 30 kg/m²; in the DYSIS study,

Fig. 2



Data are expressed as number (%) or mean±SD
BMI : Body Mass Index.

Potency of statins used according to BMI. Data are expressed as number (%) or mean ± SD.

a higher dose of statins was used (20 vs. 15 mg/day) and ezetimibe was prescribed more frequently (10.5 vs. 3.2%) in subjects suffering from obesity than in those with a BMI below 30 kg/m² [13]. Furthermore, the LEADER 5 study showed that patients with a higher BMI (>25 kg/m²) took a greater number of statins and antihypertensives than normal-weight patients (67.9% vs. 75.9%) [19].

LDL-c target attainment was higher than in other studies [17,20].

This could be explained, at least in part, by a high-intensity treatment regimen involving frequent prescription of ezetimibe (about half the patients).

In contrast, in the FAST-MI 2015 study, only 19% of subjects achieved the LDL-c target [17]. Only 3% were prescribed ezetimibe. Similarly, in the DA VINCI study [20], carried out in 18 European countries on 2128 subjects, the LDL target was only achieved by 18%. The use of statins in combination with ezetimibe concerned only 9% of subjects, with this combination achieving the LDL target in 20% of cases.

This may be explained by the lower potency of the statin: in the FAST-MI study, only 47% of subjects received a high-intensity statin at hospital discharge, while in the DA VINCI study, this figure was only 36.7%.

In our study, 97% of subjects were on high-intensity statins, which explains why the LDL-c target was better achieved.

We also found that the LDL-c target was better achieved in people with a BMI greater than 30 kg/m², which contradicts the study by Bhan *et al.* [12] who showed, from two Canadian prospective observational cohorts, that the percentage of subjects with a history of cardiovascular disease or diabetes achieving the LDL-c target was significantly lower in subjects suffering from obesity (45%) than in nonobese subjects (48.3%).

Using a multivariate model, we showed that BMI had no influence, only the variables HDL at M3 and hypertension (OR: 0.312; $P = 0.0020$) were significant.

A 0.10 increase in HDL-c increases the risk of not reaching the LDL-c target. Arterial hypertension has been

shown to reduce the risk of not reaching the LDL-c target. Indeed, in Milogo's retrospective study, 84.2% of hypertensive patients had dyslipidemia [21]. There is, therefore, a link between hypertension and LDL-c.

Limitations

There are several limitations to our study. First, the study is monocentric; it would have been interesting if it had been carried out in different centers to generalize the results. However, as this is mainly a mechanistic study, we do not think it could impact our conclusions, but local practices such as the prescription of ezetimibe could limit comparisons.

We were confronted with 190 subjects who were lost to follow-up, which resulted in a loss of data and a lower number of subjects included. Having more subjects could have increased the statistical power of the study.

The large number of patients lost to follow-up could be explained by the fact that not all subjects resided in Montpellier or the surrounding area, and therefore carried out their follow-up in other hospitals or private practices, or by the fact that subjects were given a nonopposition form to participate, rather than a consent form. Signing a consent form would perhaps help to retain patients.

It would have been interesting to collect data from patients lost to follow-up, to better understand the mechanisms of this type of subject.

Despite these limitations, the study was carried out under real-life conditions.

Conclusion

The results of the single-center study show that post-ACS management must continue to be strengthened and improved, particularly in patients with a BMI of less than 30 kg/m^2, to achieve the LDL-c target, set by the ESC.

This highlights the fact that the drug strategy does not need to be modified for people suffering from obesity. Rather, it should be reinforced for all types of patients.

Despite the prescription of high-intensity statins, half the population reached this target. A total of 67.6% of subjects suffering from obesity achieved the target, compared with 44.8% of subjects with a BMI of less than 30 kg/m^2.

A multidisciplinary team could improve follow-up after ACS to achieve the LDL-c target and avoid other complications.

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Conflicts of interest

François Roubille: declares honoraria for lectures/consulting byh: Astra Zeneca, Servier, Boehringer, Astra Zeneca, Vifor, Bayer, Pfizer, Novartis, Servier, Novonordisk, Air liquid, Abbott, QuidelOrtho, Newcard, MSD, BMS, Sanofi, Alnylam, Zoll, Implicity, GSK, BMS.

Ariane Sultan: Servier, Viatris, Amgen, Sanofi.

References

1 WHO. *Cardiovascular disease report*, May 17, 2017. https://www.who.int/news-room/fact-sheets/detail/cardiovascular-diseases-(cvds).
2 WHO. *Obesity report*. 2016. https://www.who.int/news-room/fact-sheets/detail/obesity-and-overweight.
3 Klein S, Burke LE, Bray GA, Blair S, Allison DB, Pi-Sunyer X, *et al*; American Heart Association Council on Nutrition, Physical Activity, and Metabolism. Clinical implications of obesity with specific focus on cardiovascular disease: a statement for professionals from the American Heart Association Council on Nutrition endorsed by the American College of Cardiology Association. *Circulation* 2004; **110**:2952–2967.
4 Hamadou B, Thuaire C, Range G, Demicheli T, Kane A, Albert F. Acute coronary syndrome in young Caucasian: about 62 cases. *Pan Afr Med J* 2013. doi: 10.11604/pamj.2013.14.116.2466.
5 Choudhury L, Marsh J. Myocardial infarction in young patients. *Am J Med* 1999; **107**:254–226.
6 Kanitz MG, Giovannucci SJ, Jones JS, Mott M. Myocardial infarction in young adults: risk factors and clinical features. *J Emerg Med* 1996; **14**:139–145.
7 Poirier P, Giles TD, Bray GA, Hong Y, Stern J, Pi-Sunyer X, *et al*. Obesity and Cardiovascular diseases: Pathophysiology, evaluation, effect of weight loss. *Circulation* 2006; **113**:898–918.
8 Mahmood SS, Levy D, Vasan RS, Wang TJ. The Framingham heart study and the epidemiology of cardiovascular disease: a historical perspective [archive]. *Lancet* 2014; **383**:999–1008.
9 Bogers RP, Bemelmans WJ, Hoogenveen RT, Boshuizen HC, Woodward M, Knekt P, *et al*; BMI-CHD Collaboration Investigators. Association of overweight with increased risk of coronary heart disease partly independent of blood pressure and cholesterol levels a meta-analysis of 21 cohort studies including more than 300 000. *Arch Intern Med* 2007; **167**:1720–1728.
10 Marx N, Federici M, Schütt K, Müller-Wieland D, *et al*; ESC Scientific Document Group. 2023 ESC – guidelines for the management of cardiovascular disease in patients with diabetes. *Eur Heart J* 2023; **44**:4043–4140.
11 Gouju J, Legeay J. Pharmacokinetics of obese adults: not only an increase in weight. *Biomed Pharmacother* 2023; **166**:115281.
12 Bhan V, Yan RT, Leiter LA, Fitchett D, Langer A, Lonn E, *et al*. Relation between obesity and the attainment of optimal blood pressure and lipid targets in high vascular risk outpatients. *Am J Cardiol* 2010; **106**:1270–1276.
13 Ferrières J, Lautsch D, Gitt AK, Toplak H, Elisaf M, Drexel H, *et al*. Body mass index impacts the choice of lipid-lowering treatment with no correlation to blood cholesterol – findings from 52 916 patients in the Dyslipidemia International Study (DYSIS). *Diabetes Obes Metab* 2018; **20**:2670–2674.
14 Brost M, *et al*. Impact of obesity on tolerance and persistence of statins in patients within three months following an acute myocardial infarction: a real-world study. *Arch Cardiovasc Dis* 2024.
15 Hao W, Wang X, Fan J, Guo R, Gong W, Yan Y, *et al*. Prognostic implications of OSA in acute coronary syndrome by obesity status. *Chest* 2023; **164**:219–230.
16 Martín-Castellanos A, Barca FJ, Cabañas MD, Martín P, García M, Muñoz MA, *et al*. Obesity and anthropometric indicators in a sample of males with Acute Coronary Syndrome in a Health Area with inclusion of inmates: case-control study. *Rev Esp Sanid Penit* 2015; **17**:20–29.
17 Puymirat E, Simon T, Cayla G, Cottin Y, Elbaz M, Coste P, *et al*; USIK, USIC 2000, and FAST-MI Investigators. Acute myocardial infarction: changes in patient characteristics, management, and 6-month outcomes over a period of 20 years in the FAST-MI Program (French Registry of Acute ST-Elevation or Non-ST-Elevation Myocardial Infarction) 1995 to 2015. *Circulation* 2017; **136**:1908–1919.
18 Critchley JA, Capewell S. Mortality risk reduction associated with smoking cessation in patients with coronary heart disease: a systematic review. *JAMA* 2003; **290**:86–97.
19 Bouzidi A, Rodier M, Masmiquel L, Bain S. LEADER-5 – Prévalence et impact cardiométabolique de l'obésité chez les patients diabétiques de type 2 à haut risque cardiovasculaire: données à l'inclusion de l'étude LEADER. *Diabetes Metab* 2016; **42**:A39.
20 Ray KK, Molemans B, Schoonen WM, Giovas P, Bray S, Kiru G, *et al*; DA VINCI study. EU-wide cross-sectional observational study of lipid-modifying therapy use in secondary and primary care: the DA VINCI study. *Eur J Prev Cardiol* 2021; **28**:1279–1289.
21 Milogo G, Samandoulougou A, Yameogo N, Yaméogo A, Kologo K, Toguyeni J, Zabsonré P. Metabolic syndrome in hypertensive patients in the cardiology service Yalgado Ouedraogo of Ouagadougou, Burkina Faso. *Pan Afr Med J* 2014; **19**:290.