Original paper

Negative impact of reducing lipid-lowering therapy after achieving target LDL-C in patients with a very high cardiovascular risk undergoing cardiac rehabilitation

Martyna Świerkowska[1], Małgorzata Kurpaska[2], Paweł Krzesiński[2], Katarzyna Piotrowicz[2]

[1]Student Scientific Club "Cardiacus" at the Department of Cardiology and Internal Medicine, Military Institute of Medicine – National Research Institute (WIM-PIB), Warsaw, Poland
[2]Department of Cardiology and Internal Diseases, Military Institute of Medicine – National Research Institute (WIM-PIB), Warsaw, Poland

Medical Studies
DOI: https://doi.org/10.5114/ms.2026.161292

Key words: statins, cardiac rehabilitation, low-density lipoprotein cholesterol, lipid-lowering therapy.

Abstract

Introduction: Despite well-documented benefits of intensive lipid-lowering treatment, the use of statins in patients with chronic coronary syndrome remains suboptimal. Statin dose is often reduced after reaching low-density lipoprotein cholesterol (LDL-C) target.
Aim of the research: The purpose of the study was to assess changes in LDL-C levels following lipid-lowering therapy reduction during cardiac rehabilitation (CR).
Material and methods: A total of 162 patients with a very high cardiovascular risk (target LDL-C levels of < 55 mg/dl), in whom LDL-C levels were measured at the beginning and end of phase 2 of CR, were selected out of a single-centre retrospective registry of 518 patients.
Results: Patients had no contraindications to continue high-dose lipid-lowering therapy. Prior to undergoing CR, 63 (38.9%) patients had reached their target LDL-C levels and the statin or ezetimibe dose was reduced in 18 patients, increased in 2 patients, and unchanged in 43 patients. During CR the mean LDL-C levels in the study group decreased significantly from 70.5 ±37.1 mg/dl to 56.2 ±21.5 mg/dl ($p = 0.0002$). Conversely, patients who had their lipid-lowering therapy reduced, showed a significant increase in their LDL-C levels from 32.6 ±13.2 mg/dl to 46.5 ±15.5 mg/dl ($p = 0.0004$), with 6 (33.3%) patients exceeding their target LDL-C levels. At CR completion, a total of 77 (47.5%) patients failed to maintain their target LDL-C levels (< 55 mg/dl).
Conclusions: Reducing of well-tolerated lipid-lowering therapy may lead to the loss of target LDL-C levels. These results emphasise the importance of continuing lipid-lowering therapy at effective doses even after reaching target LDL-C levels.

Introduction

Dyslipidaemia is one of the key cardiovascular risk factors for cardiovascular disease, stroke, and peripheral artery disease [1]. Elevated low-density lipoprotein cholesterol (LDL-C) levels are the main risk factor for atherosclerosis development and progression [2] and coronary event recurrence [3, 4]. Prognosis may be improved with a multidirectional approach, such as a combination of diet and lifestyle modifications and lipid-lowering therapy [3], mainly with the use of statins [2, 5–7]. This approach is introduced during cardiac rehabilitation (CR), including hybrid CR, which combines inpatient care and at-home telerehabilitation, thus extending the period of care anywhere from 6 weeks to 3 months.

Many authors have addressed the issue of lipid-lowering therapy reduction either after target LDL-C levels are reached or irrespective of LDL-C levels [8–10]. Unfortunately, few patients reach target LDL-C levels, which was demonstrated in the 3ST-POL study (with only 12.6% of high-risk Polish outpatients achieving LDL-C levels of < 55 mg/dl [11]). These unsatisfactory outcomes are partly a result of patients' and physicians' concerns about the side effects and their lack of conviction as to the validity of intense pharmacotherapy [12].

The goal of secondary prevention with lipid-lowering therapy in very-high-risk patients (particularly those with evidence of atherosclerotic plaques or a history of coronary revascularization or an aortic aneurysm) is to lower LDL-C levels to < 1.4 mmol/l (< 55 mg/dl) and to reach at least a 50% reduction in LDL-C levels in comparison with baseline [3, 13]. European Society of Cardiology (ESC) guidelines on chronic coronary syndromes (2019) and cardiovascular disease prevention (2021) [5, 14] focused mainly on

reaching the therapeutic target, in the form of LDL-C levels. Since further management after reaching LDL-C levels of < 55 mg/dl has not been specified, a number of clinicians have been lowering statin dosage once target LDL-C levels had been reached.

Aim of the research

The purpose of this study was to assess changes in serum LDL-C levels following reduction in of lipid-reducing treatment in patients with a very high cardiovascular risk who had reached the therapeutic target during CR.

Material and methods

A single-centre retrospective review of the medical records was performed in patients who were eligible for CR in the period from February 2022 to March 2024. From the total of 518 patients included in the study, a group of 162 patients at a very high cardiovascular risk (with the target LDL-C levels of < 55 mg/dl), in whom serum LDL-C levels were measured during eligibility screening for CR (serum LDL-C levels within 56 days after the coronary intervention) and, again, after at least 6 weeks (i.e. at the end of the second phase of CR), with a maximum interval between the two LDL-C measurements of 90 days.

The analysed data included comorbidities, the initially introduced medical treatment and its later modifications, and serum LDL-C levels. LDL-C levels were measured directly, with an enzymatic colorimetric method and the COBAS c 503 analytical unit.

Statistical analysis

Statistical analysis was conducted with Statistica version 12.0 (StatSoft, Tulsa, OK, USA). The distribution of continuous variables was evaluated with the Shapiro–Wilk test. Continuous variables with a normal distribution were expressed as means ± standard deviation (SD), and those with a non-normal distribution were expressed as medians and interquartile ranges (IQR: Q1–Q3). A non-parametric Wilcoxon test was used to assess the statistical significance of changes in LDL-C levels during CR in the entire study population. A sign test was used in the subgroup of patients who had their lipid-lowering therapy reduced after reaching the therapeutic goal, due to the small size of that subgroup (n = 18). The change in the proportion of patients with LDL-C < 55 mg/dl and LDL-C ≥ 55 mg/dl was assessed with McNemar's test. All tests were two-tailed; and the statistical significance was set at $p < 0.05$.

Results

Out of 162 patients, 80% were male. The mean patient age was 63 years, and the median left ventricular ejection fraction was 53%. All patients were characterized by/had a very high cardiovascular risk, with 160 (98.8%) patients having undergone coronary revascularization (Table 1).

The mean pre-CR LDL-C levels were 70.5 ±37.1 mg/dl, and post-CR levels were significantly decreased at 56.2 ±21.5 mg/dl ($p < 0.001$).

Only 63 (38.9%) patients with a history of coronary intervention had reached their target LDL-C levels of < 55 mg/dl prior to CR, whereas most of the patients (61.1%) failed to reach their therapeutic target. Following CR completion, the proportion of patients with a serum LDL-C level of < 55 mg/dl increased by 13.5%. Nonetheless, as many as 77 (47.5%) patients still failed to reach their therapeutic target, despite at least eight weeks of lipid-lowering therapy (Figure 1).

Out of the patients who reached the target LDL-C levels prior to CR (n = 63, 38.9%) and had no contraindications to continue high-dose lipid-lowering therapy, the statin dose was lowered in 16 patients and increased in 2 patients, ezetimibe was discontinued in 2 patients, and the treatment regimen remained unchanged in 43 patients. The decision regarding modification of lipid-lowering therapy was taken individually by the cardiologist.

A follow-up test after CR completion showed a significant increase in LDL-C levels (from 32.6 ±13.2 mg/dl to 46.5 ±15.5 mg/dl; p = 0.0004) in patients with reduced lipid-lowering treatment, with 6 patients (33.3%, p = 0.025) showing loss of target LDL-C levels (Table 2).

Discussion

Modifying medical treatment soon after a coronary intervention, when the patient remains under specialist care during CR, may have important implications for further outpatient management by primary care doctors or cardiologists. Medical treatment optimisation is an important element of comprehensive CR. Relevant guidelines emphasise the significance of appropriate lipid-lowering therapy in patients with a very high cardiovascular risk [5–7]. The goal of treatment is to reach the recommended LDL-C levels, which was achieved in just over a half of patients after CR (52.5%). This was to a large extent due to lipid-lowering treatment reduction/de-escalation during eligibility screening for CR once the therapeutic LDL-C levels were reached.

Improved serum lipid level control during CR was reported by many authors [15–17]. This had been achieved through medical treatment, increased physical activity, and good dietary recommendations [18–20].

The most recent ESC guidelines for the management of acute coronary syndromes (2023) and chronic coronary syndromes (2024) [6, 7] highlighted the need for long-lasting treatment with statins at a maximum tolerated dose. Failure to reach the therapeutic goal indicates the need for medical treatment escalation and introduction of such treatments as proprotein

Table 1. Clinical characteristics of the study population

Parameter	Study population, *n* = 162
Age, mean (SD)	63 (10)
Men, *n* (%)	129 (80)
BMI [kg/m^2], mean (SD)	27.7 (4.1)
HR [min^{-1}], mean (SD)	65 (8)
SBP [mm Hg], mean (SD)	119 (17)
DBP [mm Hg], mean (SD)	70 (10)
Left ventricular ejection fraction [%], median (IQR)	53 (48–60)
Coronary artery revascularization, *n* (%)	160 (98.8)
PCI, *n* (%)	159 (98.2)
CABG, *n* (%)	6 (3.7)
PCI and CABG, *n* (%)	5 (3.1)
Comorbidities, *n* (%)	
Previous myocardial infarction	101 (62.4)
Heart failure	49 (30.3)
Arterial hypertension	114 (70.4)
Diabetes mellitus type 2	44 (27.2)
Peripheral artery disease	7 (4.3)
Chronic kidney disease	2 (1.2)
Laboratory tests during qualification to the cardiac rehabilitation [mg/dl], mean (SD)	
TC	130.6 (43.9)
LDL-C	70.5 (37.1)
HDL-C	43.5 (12.5)
TG	120.4 (66.3)
FG	101.8 (33.6)
Creatinine	1.03 (0.4)
Medications, *n* (%)	
Statin	105 (64.8)
Statin with ezetimibe	57 (35.2)
ACE/ARB	154 (95.1)
BB	140 (86.4)
CB	46 (28.4)
DIU	62 (38.3)
MRA	36 (22.2)
Metformin	39 (24.1)
Flozin	44 (27.2)
Amiodaron	2 (1.2)

ACE – angiotensin-converting enzyme inhibitors, ARB – angiotensin receptor blockers, BB – beta-adrenergic receptor antagonists, CABG – coronary artery bypass grafting, BMI – body mass index, CB – calcium channel blockers, CR – cardiac rehabilitation, DBP – diastolic blood pressure, HR – heart rate, DIU – diuretics, FG – fasting glucose, HDL-C – high-density lipoprotein cholesterol, MRA – mineralocorticoid receptor antagonists, LDL-C – low-density lipoprotein cholesterol, PCI – percutaneous coronary angioplasty, SBP – systolic blood pressure, SD – standard deviation, TC – total cholesterol, TG – triglycerides.

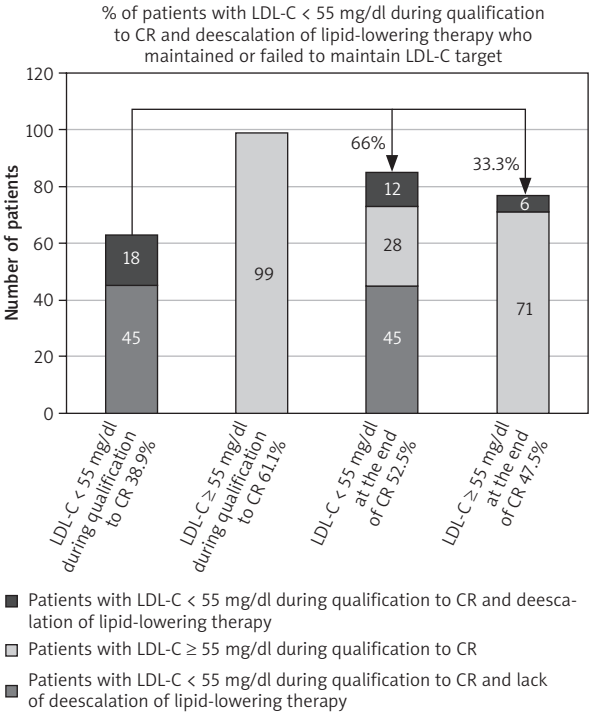

Figure 1. The proportion of patients with LDL-C < 55 mg/dl and ≥ 55 mg/dl prior to and after cardiac rehabilitation

CR – cardiac rehabilitation.

convertase subtilisin/kexin type 9 (PCSK9). Some authors decidedly recommend an initial high-intensity statin therapy (80 mg atorvastatin, 20–40 mg rosuvastatin) in combination with 10 mg ezetimibe and continuation of this treatment after the LDL-C target of < 55 mg/dl (1.7 mmol/l) has been reached in patients with a high and very high cardiovascular risk [13]. Those authors pointed out that the 2023 ESC guidelines for the management of acute coronary syndromes repeatedly emphasised the need for treatment with a statin at the highest tolerated dose; however, the lack of changes in the treatment regimen after the therapeutic goal has been reached was explicitly described only in one figure (no. 18) from that paper. The results of our study unequivocally demonstrated that lipid-lowering treatment reduction once the target LDL-C level had been reached, led to a significant increase in LDL-C levels with a loss of the target levels in a considerable proportion of patients.

Lowering the dose of well-tolerated lipid-lowering treatment may be a result of either patient nonadherence [9, 21] or both doctors' and patients' concerns about potential side effects of statins. It is worth noting that no risks associated with low LDL-C levels have been demonstrated in randomised clinical trials [22] and that serious side effects of statins are rare and include rhabdomyolysis in 1 out of 10,000 patients/year [23], hepatotoxicity (4%) [24], impaired cognitive function (0.08%) [22], and hemorrhagic strokes (0.9%) [25, 26]. Studies showed no association between low LDL-C levels and the suggested development of dementia [27, 28]. Conversely, a lack of statin treatment has been repeatedly demonstrated to increase the risk of cardiovascular events and mortality [29]. There have been a number of studies focusing on reaching target LDL-C levels and evaluating the risk of developing a cardiovascular event following discontinuation of adequate treatment [29–31].

The IDEAL study evaluated de-escalation of lipid-lowering therapy after reaching a therapeutic goal of 39 mg/dl [32]. The study demonstrated ineffectiveness of that type of treatment, which failed to achieve the predetermined minimum LDL-C levels. The decision to de-escalate lipid-lowering therapy during CR may be considered definitive and be upheld during subsequent outpatient management even despite an increase in LDL-C levels and loss of the previously reached therapeutic goal.

Our study sample size was relatively small, and the study had not been designed to assess the effectiveness of treatment reduction instead it was merely a retrospective observational study of clinical practice at a single centre. The practice of refraining from lipid-lowering therapy de-escalation was being introduced at the study centre already after the evaluated period had begun, which may have affected the results of our analysis (underestimation of the problem). Assessing how common this type of management is and evaluating its effects requires further studies in large groups of patients.

Conclusions

Reduction of well-tolerated lipid-lowering therapy after target LDL-C levels have been reached may lead to increased LDL-C levels and loss of target LDL-C levels. Therefore, the therapy should be continued with periodic monitoring of its effectiveness and safety.

Table 2. Changes in LDL-C levels in the group of patients (n = 18) whose lipid-lowering therapy was reduced after their therapeutic target had been reached

LDL-C before CR [mg/dl]	LDL-C after CR [mg/dl]	*P*-value
Mean ± SD: 32.8 ±13.2	Mean ± SD: 46.5 ±15.5	< 0.001
Min.–max.: 12–54	Min.–max.: 18–77	
IQR: 21–43	IQR: 38–59	

LDL-C – low-density lipoprotein cholesterol.

Funding

This study was funded as part of government research grant No. 674 for the Military Institute of Medicine, National Research Institute (WIM PIB grant No. 674).

Ethical approval

The study had been approved by the local ethics committee at the Military Institute of Medicine, National Research Institute (Decision 32/24 of 19 June 2024).

Conflict of interest

The authors declare no conflict of interest.

References

1. Ott C, Schmieder RE. The role of statins in the treatment of the metabolic syndrome. Curr Hypertens Rep. 2009; 11(2): 143-149.
2. Dayar E, Pechanova O. Targeted strategy in lipid-lowering therapy. Biomedicines. 2022; 10(5): 1090.
3. Lewis SJ. Lipid-lowering therapy: who can benefit? Vasc Health Risk Manag. 2011; 7: 525-534.
4. Feher MD. Lipid lowering to delay the progression of coronary artery disease. Heart. 2003; 89(4): 451-458.
5. McDonagh TA, Metra M, Adamo M, Gardner RS, Baumbach A, Böhm M, Burri H, Buler J, Čelutkienė J, Chioncel O, Cleland JGF, Coats AJS, Crespo-Leiro MG, Farmakis D, Gilard M, Heymans S, Hoes AW, Jaarsma T, Jankowska EA, Lainscak M, Lam CSP, Lyon AR, McMurray JJV, Mebazaa A, Mindham R, Muneretto C, Piepoli MF, Price S, Rosano GMC, Ruschitzka F, Skibelund AK; ESC Scientific Document Group. 2021 ESC Guidelines for the diagnosis and treatment of acute and chronic heart failure: Developed by the Task Force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC) With the special contribution of the Heart Failure Association (HFA) of the ESC. European Heart J. 2021; 42(36): 3599-3726.
6. Byrne RA, Rossello X, Coughlan JJ, Barbato E, Berry C, Chieffo A, Claeys MJ, Dan GA, Dweck MR, Galbraith M, Gilard M, Hinterbuchner L, Jankowska EA, Jüni P, Kimura T, Kunadian V, Leosdottir M, Lorusso R, Pedretti RFE, Rigopoulos AG, Rubini Gimenez M, Thiele H, Vranckx P, Wassmann S, Kass Wenger N, Ibanez B, ESC Scientific Document Group. 2023 ESC Guidelines for the management of acute coronary syndromes: Developed by the task force on the management of acute coronary syndromes of the European Society of Cardiology (ESC). European Heart J. 2023; 44(38): 3720-3826.
7. Vrints C, Andreotti F, Koskinas KC, Rossello X, Adamo M, Ainslie J, Banning AP, Budaj A, Buechel RR, Chiariello GA, Chieffo A, Christodorescu RM, Deaton C, Doenst T, Jones HW, Kunadian V, Mehilli J, Milojevic M, Piek JJ, Pugliese F, Rubboli A, Semb AG, Senior R, Ten Berg JM, Van Belle E, Van Craenenbroeck EM, Vidal-Perez R, Winther S, ESC Scientific Document Group. 2024 ESC Guidelines for the management of chronic coronary syndromes: Developed by the task force for the management of chronic coronary syndromes of the European Society of Cardiology (ESC) Endorsed by the European Association for Cardio-Thoracic Surgery (EACTS). European Heart J. 2024; 45(36): 3415-3537.
8. Kardas P, Kwiatek A, Włodarczyk P, Urbański F, Ciabiada-Bryła B. Is the KOS-Zawał coordinated care program effective in reducing long-term cardiovascular risk in coronary artery disease patients in Poland? Insights from analysis of statin persistence in a nationwide cohort. Pol Heart J. 2024; 82(9): 852-860.
9. Kardas P, Kwiatek A, Włodarczyk P, Urbański F, Ciabiada-Bryła B. Statins use amidst the pandemic: prescribing, dispensing, adherence, persistence, and correlation with COVID-19 statistics in nationwide real-world data from Poland. Front Pharmacol. 2024; 15: 1350717.
10. Cecha P, Chromik A, Piotrowska I, Zabojszcz M, Dolecka-Ślusarczyk M, Siudak Z. Assessment of application of the new 2019 European Society of Cardiology/European Atherosclerosis Society Guidelines for the Management of Dyslipidaemias in daily clinical practice – one center study. Folia Med Cracov. 2021; 61(3): 43-54.
11. Wełnicki M, Śliż D, Filipiak K, Siebert J, Naruszewicz M, Mamcarz A. Różnice w skuteczności leczenia dyslipidemii u mężczyzn z rozpoznaną otyłością w porównaniu z mężczyznami nieotyłymi. Badanie 3ST-POL. Forum Med Rodz. 2016; 10(4): 179-188.
12. Benn M, Nordestgaard BG, Frikke-Schmidt R, Tybjærg-Hansen A. Low LDL cholesterol, PCSK9 and HMGCR genetic variation, and risk of Alzheimer's disease and Parkinson's disease: Mendelian randomisation study. BMJ. 2017; 357: j1648.
13. Tada H, Usui S, Sakata K, Takamura M, Kawashiri MA. Low-density lipoprotein cholesterol level cannot be too low: considerations from clinical trials, human genetics, and biology. J Atheroscler Thromb. 2020; 27(6): 489-498.
14. Knuuti J, Wijns W, Saraste A, Capodanno D, Barbato E, Funck-Brentano C. Wytyczne ESC dotyczące rozpoznawania i leczenia przewlekłych zespołów wieńcowych (2019). Polish Heart J. 2020; 78(I): 10-87.
15. Gao Y, Ding J, Kim C, Spaulding E, Isakadze N, Stewart K, Broderick A, Bush A, MacFarlane Z, Nimbalkar M, Desai A, Mathews L, Marvel F, Garcvia-Garcia H, Mardtin SS. Abstract 4142395: Lipid trends of adults who participate in hybrid cardiac rehabilitation. Circulation. 2024; 150 (suppl 1).
16. Meslet JB, Dugué B, Brisset U, Pianeta A, Kubas S. Evaluation of a hybrid cardiovascular rehabilitation program in acute coronary syndrome low-risk patients organised in both cardiac rehabilitation and sport centres: a model feasibility study. Int J Environ Res Public Health. 2022; 19(15): 9455.
17. Harzand A, Alrohaibani A, Idris MY, Spence H, Parrish CG, Rout PK, Nazar R, Davis-Watts ML, Wright PP, Vakili AA, Abdelhamid S, Vathsangam H, Adesanya A, Park LG, Whooley MA, Wenger NK, Zafari AM, Shah AJ. Effects of a patient-centered digital health intervention in patients referred to cardiac rehabilitation: the Smart HEART clinical trial. BMC Cardiovasc Disord. 2023; 23(1): 453.
18. Scolaro B, Nogueira MS, Paiva A, Bertolami A, Barroso LP, Vaisar T, Heffron SP, Fisher EA, Castro IA. Statin dose reduction with complementary diet therapy: a pilot study of personalized medicine. Mol Metab. 2018; 11: 137-144.
19. Schoeneck M, Iggman D. The effects of foods on LDL cholesterol levels: a systematic review of the accumulated evidence from systematic reviews and meta-analyses

of randomized controlled trials. Nutr Metab Cardiovasc Dis. 2021; 31(5): 1325-1338.
20. Lira FS, Zanchi NE, Lima-Silva AE, Pires FO, Bertuzzi RC, Santos RV, Caperuto É, Kiss M, Seelaender M. Acute high--intensity exercise with low energy expenditure reduced LDL-c and total cholesterol in men. Eur J Appl Physiol. 2009; 107(2): 203-210.
21. Fang HSA, Gao Q, Lee ML, Hsu W, Tan NC. LDL-cholesterol change and goal attainment following statin intensity titration among Asians in primary care: a retrospective cohort study. Lipids Health Dis. 2021; 20(1): 2.
22. Goldstein LB, Toth PP, Dearborn-Tomazos JL, Giugliano RP, Hirsh BJ, Peña JM, Selim MH, Woo D; American Heart Association Council on Arteriosclerosis, Thrombosis and Vascular Biology; Council on Cardiovascular and Stroke Nursing; Council on Peripheral Vascular Disease; and Stroke Council. Aggressive LDL-C lowering and the brain: impact on risk for dementia and hemorrhagic stroke: a scientific statement from the American Heart Association. Arterioscler Thromb Vasc Biol. 2023; 43(10): e404-e442.
23. Starzyk K, Wożakowska-Kapłon B. Objawy mięśniowe w przebiegu stosowania statyn – fakty, mity, rzeczywistość i stanowiska ekspertów. Folia Cardiol. 2015; 10(5): 354-360.
24. Jose J, Al-Tamimi FA, Helal MM, Jimmy B, Al Riyami Q. Statin associated hepatic adverse effects: a retrospective review from a regional hospital in sultanate of Oman. Oman Med J. 2014; 29(5): 351-357.
25. Urząd Rejestracji Produktów Leczniczych, Wyrobów Medycznych i Produktów Biobójczych. Charakterystyka Produktu Leczniczego: Atorvox 40 mg, tabletki powlekane. Retrieved April 28, 2025, from https://leki.urpl.gov.pl/files/77_Atorvox_tabletki_powlekane_40mg.pdf
26. Dobrowolski P, Prejbisz A, Kuryłowicz A, Baska A, Burchardt P, Chlebus K, Dzida G, Jankowski P, Jaroszewicz J, Jaworski P, Kamiński K, Kapłon-Cieślicka A, Klocek M, Kukla M, Mamcarz A, Mastalerz-Migas A, Narkiewicz K, Ostrowska L, Śliż D, Tarnowski W, Wolf J, Wyleżoł M, Zdrojewski T, Banach M, Januszewicz A, Bogdański P. Zespół metaboliczny – nowa definicja i postępowanie w praktyce. Stanowisko PTNT, PTLO, PTL, PTH, PTMR, PTMSŻ, sekcji Prewencji i Epidemiologii PTK, „Klubu 30" PTK oraz sekcji Chirurgii Metabolicznej i Bariatrycznej TChP. Nadciśnienie Tętnicze w Praktyce. 2022; 8(2): 47-72.
27. Shepherd J, Blauw GJ, Murphy MB, Bollen ELEM, Buckley BM, Cobbe SM, Ford I Gaw A, Hyland M, Jukema JW, Kamper AM, Macfarlane PW, Meinders AE, Norrie J, Packard CJ, Perry IJ, Stott DJ, Sweeney BJ, Twomey C, Westendorp RGJ; PROSPER study group. PROspective Study of Pravastatin in the Elderly at Risk. Pravastatin in elderly individuals at risk of vascular disease (PROSPER): a randomised controlled trial. Lancet. 2002; 360(9346): 1623-1630.
28. MRC/BHF Heart Protection Study of cholesterol lowering with simvastatin in 20,536 high-risk individuals: a randomised placebo-controlled trial. Lancet. 2002; 360(9326): 7-22.
29. Peixoto C, Choudhri Y, Francoeur S, McCarthy LM, Fung C, Dowlatshahi D, Lemay G, Barry A, Goyal P, Pan J, Bjerre LM, Thompson W. Discontinuation versus continuation of statins: a systematic review. J Am Geriatr Soc. 2024; 72(11): 3567-3587.
30. Lee SH, Kwon HS, Park YM, Ko SH, Choi YH, Yoon KH, Ahn YB. Statin discontinuation after achieving a target low density lipoprotein cholesterol level in type 2 diabetic patients without cardiovascular disease: a randomized controlled study. Diabetes Metab J. 2014; 38(1): 64-73.
31. Ageeb Shahd A, Abdelmoghith A, ElGeed H, Awaisu A, ElMansor A, Owusu Yaw B. Prevalence, associated risk factors, and adverse cardiovascular outcomes of statins discontinuation: a systematic review. Pharmacoepidemiol Drug Safety. 2024; 33(8): e5879.
32. Pedersen TR, Faergeman O, Kastelein JJP, Olsson AG, Tikkanen MJ, Holme I, Larsen ML, Bendiksen FS, Lindahl C, Szarek M, Tsai J; Incremental Decrease in End Points Through Aggressive Lipid Lowering (IDEAL) Study Group. High-dose atorvastatin vs usual-dose simvastatin for secondary prevention after myocardial infarction the IDEAL Study: a randomized controlled trial. JAMA. 2005; 294(19): 2437-2445.

Address for correspondence

Martyna Świerkowska
Student Scientific
Club "Cardiacus''
Department of Cardiology
and Internal Medicine
Military Institute of Medicine
– National Research Institute
(WIM-PIB)
Warsaw, Poland
E-mail: m.swierku2@gmail.com

Received: 25.11.2025
Accepted: 4.01.2026
Online publication: 23.04.2026