American Journal of Internal Medicine
2026, Vol. 14, No. 4, pp. 75–79
https://doi.org/10.11648/j.ajim.20261404.13

Research Article

Lifestyle Intervention for the Management of Hypercholesterolemia in a Low-Risk Middle-Aged Female: A Case Report

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Abstract

Background: Intensive lifestyle modification is central to dyslipidemia management, although pharmacologic therapy is often prioritized when lipid levels are moderately elevated. Similar to quality improvement efforts addressing pre-analytical variables in laboratory medicine—where outcomes depend on structured interventions and patient adherence—the management of hypercholesterolemia likewise requires sustained engagement in non-intuitive but beneficial health behaviors. *Case Presentation*: A 53-year-old woman with a 2–3-year history of hypercholesterolemia presented with total cholesterol 6.4 mmol/L (247.5 mg/dL), LDL-C 4.0 mmol/L (154.7 mg/dL), HDL-C 1.65 mmol/L (63.8 mg/dL), triglycerides 1.12 mmol/L (99.2 mg/dL), BMI 26.6 kg/m^2, and blood pressure 95/60 mmHg. She had a family history of diabetes (grandmother), no smoking history, and no premature atherosclerotic cardiovascular disease. Statin therapy was initially recommended. Instead, she implemented a fat-restricted diet emphasizing fruits, vegetables, fish, chicken, and oatmeal, along with 1 hour of exercise 6 days per week. After 2 months, total cholesterol decreased to 5.3 mmol/L (205.0 mg/dL), LDL-C to 3.2 mmol/L (123.7 mg/dL), HDL-C remained stable at 1.63 mmol/L (63.0 mg/dL), triglycerides 0.81 mmol/L (71.69 mg/dL), BMI decreased to 24.7 kg/m^2, and blood pressure remained stable. Carotid ultrasound showed normal morphology, vessel wall thickness, lumen diameter, and Doppler flow. Statin therapy was no longer recommended. *Conclusion*: Intensive lifestyle interventions are associated with significant improvements in lipid profiles among carefully selected low-risk patients and may reduce or obviate the need for pharmacologic therapy. Behavioral factors remain integral to the management of dyslipidemia, highlighting the importance of patient-driven modification of cardiovascular risk. Further large-scale, methodologically rigorous studies are warranted to validate the reproducibility and generalizability of these findings.

Keywords

Hypercholesterolemia, Lifestyle Intervention, LDL Cholesterol, Diet, Exercise, Case Report

1. Introduction

Hypercholesterolemia is a major modifiable risk factor for atherosclerotic cardiovascular disease (ASCVD), and contemporary dyslipidemia guidelines continue to emphasize lifestyle intervention alongside risk-based lipid-lowering therapy. [1-3, 19] The 2026 ACC/AHA/Mult Society Dyslipidemia Guideline

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Received: 22 June 2026; **Accepted:** 24 July 2026; **Published:** 26 August 2026

reinstates LDL-C treatment goals and emphasizes earlier intervention through healthy lifestyle changes to reduce lifelong exposure to atherogenic lipoproteins. [2, 3] Although statins are foundational treatment in patients with elevated cardiovascular risk, selected low-risk patients may achieve clinically relevant lipid improvement through diet, exercise, and weight reduction alone. [4, 5] The optimal timing of pharmacologic intervention remains debated, particularly when lifestyle modification may offer substantial benefit. [5]

The PREVENT-ASCVD risk equations, introduced in the 2026 guideline, provide more refined risk estimation for primary prevention in adults aged 30 to 79 years with LDL-C levels of 70–189 mg/dL (1.8–4.9 mmol/L) [2, 6, 7] and no known ASCVD. For individuals at low, borderline, or intermediate PREVENT-ASCVD risk with LDL-C between 70–189 mg/dL, the guideline recommends an LDL-C goal <100 mg/dL (<2.6 mmol/L), supporting lifestyle-first management in lower-risk patients. [2, 3]

Lifestyle interventions, including dietary modification, increased physical activity, and weight reduction are foundational to dyslipidemia management and are recommended as first-line therapy in many patients. Nevertheless, their effectiveness in real-world settings is often limited by suboptimal adherence. Evidence from behavioral and quality improvement frameworks suggests that structured strategies aimed at promoting patient engagement and sustained behavioral change can significantly influence clinical outcomes.[21] In this context, successful lipid management in low-risk individuals depends not only on clinical recommendations but also on the patient's ability to adopt and maintain non-intuitive, health-promoting behaviors.

Case reports remain useful for highlighting such individualized responses and for reinforcing the importance of shared decision-making. [8, 9]

This report describes a 53-year-old woman with moderate hypercholesterolemia who demonstrated significant improvement in lipid parameters and cardiovascular risk profile by following a structured lifestyle intervention. This case underscores the potential role of intensive and adherence-focused non-pharmacologic strategies as an initial management approach in carefully selected low-risk patients.

2. Case Report & Results

2.1. Patient Information

A 53-year-old woman with hypercholesterolemia diagnosed 2–3 years earlier, was evaluated for persistent dyslipidemia. She reported a family history of diabetes on her grandmother. She had no smoking history and no known premature ASCVD in the family. She denied chest pain, dyspnea, or other symptoms suggestive of clinical ASCVD. Her baseline blood pressure was 95/60 mmHg, and BMI was 26.6 kg/m^2. She reported no medications at baseline and no history of statin intolerance or adverse effects.

2.2. Clinical Findings

At baseline, her lipid profile showed:

1) Total cholesterol: 6.4 mmol/L (247.5 mg/dL)
2) LDL-C: 4.0 mmol/L (154.7 mg/dL)
3) HDL-C: 1.65 mmol/L (63.8 mg/dL)
4) Triglycerides: 1.12 mmol/L (99.2 mg/dL)
5) BMI: 26.6 kg/m^2
6) Blood pressure: 95/60 mmHg

After 2 months of lifestyle intervention, her values were:

1) Total cholesterol: 5.3 mmol/L (205.0 mg/dL)
2) LDL-C: 3.2 mmol/L (123.7 mg/dL)
3) HDL-C: 1.63 mmol/L (63.0 mg/dL)
4) Triglycerides: 0.81 mmol/L (71.69 mg/dL)
5) BMI: 24.7 kg/m^2
6) Blood pressure: 94/60 mmHg

The 17.5% reduction in total cholesterol, 20.0% reduction in LDL-C and 27.0% drop on triglycerides exceeded typical placebo responses observed in statin trials.

2.3. Timeline

The Table 1 presents a timeline of the events.

2.4. Diagnostic Assessment

The patient had moderate hypercholesterolemia without reported clinical evidence of ASCVD. No smoking history and no premature ASCVD supported a low apparent burden of subclinical vascular disease. According to Society of Radiologists in Ultrasound (SRU) criteria, normal internal carotid artery is defined as peak systolic velocity <125 cm/sec with no plaque or intimal thickening visible. [13, 14]

Carotid ultrasound performed 2 months after baseline showed normal bilateral carotid artery morphology, vessel walls were not thickened, the endocardial echo was smooth, the inner vascular lumen diameter was normal, and color Doppler showed continuous, complete flow with regular borders and no abnormal flow signals.

No additional abnormalities were reported. Lipoprotein(a) and apolipoprotein B measurement may be considered in selected patients to refine risk assessment, particularly when residual risk persists despite lifestyle intervention.

2.5. Therapeutic Intervention

The intervention consisted of dietary modification and structured exercise. The dietary pattern was low in saturated fat and emphasized fruits, vegetables, fish, chicken, and oatmeal. Strong, consistent evidence from controlled trials and population studies demonstrates that reducing saturated fat intake and replacing it with unsaturated fats can improves lipid profiles and cardiovascular health markers. [10]

Regular consumption of oat β-glucan (≥3 g/day) reduces LDL cholesterol by approximately 0.25 mmol/L (9.7 mg/dL) and total cholesterol by 0.30 mmol/L (11.6 mg/dL) without changing HDL cholesterol or triglycerides. [18] Physical activity was prescribed at 1 hour per session, 6 days per week (420 minutes/week), exceeding the recommended 150 minutes/week of moderate-to-vigorous aerobic exercise. [15]

Weight loss of 7.7 kg has been associated with a 1% reduction in LDL-C per kilogram lost, supporting the observed 1.9 kg/m^2 BMI reduction and 20% LDL-C decrease. No lipid-lowering drug was used after the lifestyle response. Such management is consistent with guideline principles that prioritize lifestyle optimization, particularly in primary prevention when overall risk is low. [20]

2.6. Follow-up and Outcomes

After 2 months, the patient demonstrated improvement in total cholesterol, LDL-C, BMI, and maintenance of stable HDL-C and blood pressure. Carotid ultrasound performed at the 2-month visit showed no structural abnormalities or flow disturbances. The response was clinically meaningful and the treating physician withdrew the statin recommendation. No adverse events were reported during the lifestyle intervention period.

3. Discussion

This case illustrates that intensive lifestyle change may be sufficient to normalize or substantially improve lipid values in selected patients with moderate hypercholesterolemia. The patient's baseline LDL-C of 4.0 mmol/L (154.7 mg/dL) falls within the 70–189 mg/dL (1.8–4.9 mmol/L) range where the 2026 ACC/AHA guideline recommends LDL-C <100 mg/dL (<2.6 mmol/L) for individuals at low, borderline, or intermediate PREVENT-ASCVD risk. [2, 3] At 53 years of age without smoking, diabetes, hypertension, or established ASCVD, her estimated 10-year PREVENT-ASCVD risk likely falls below 5%, supporting lifestyle-first management. [2, 7]

The 20% reduction in LDL-C (from 4.0 to 3.2 mmol/L) achieved through lifestyle modification alone is clinically significant. Meta-analyses demonstrate that plant-based diets reduce LDL-C levels by 10% compared to omnivore diets. This reduction is largely driven by independent factors, such as decreased saturated fat and increased soluble fiber intake. [11, 12] The observed 17.5% reduction in total cholesterol exceeds typical placebo responses in statin trials (approximately 5–10%) and approaches the magnitude of effect seen with moderate-intensity statins. [17]

The mechanisms underlying these lipid improvements are multifactorial. Reduced saturated fat intake decreases hepatic cholesterol synthesis and downregulates LDL receptor expression, while increased soluble fiber (particularly oat β-glucan) binds bile acids in the intestinal lumen, increasing fecal excretion and promoting hepatic cholesterol uptake via upregulated LDL receptors. [10, 18] Exercise increases lipoprotein lipase activity in muscle tissue, enhancing triglyceride hydrolysis and promoting conversion of small, dense LDL particles to larger, more buoyant LDL subclasses that may have lower atherogenicity. [15]

Weight loss of 1.9 kg/m^2 (from BMI 26.6 to 24.7 kg/m^2) contributes to lipid improvement through multiple pathways. Adipose tissue reduction decreases free fatty acid flux to the liver, reducing hepatic VLDL synthesis and subsequent LDL formation. Additionally, weight loss improves insulin sensitivity, which downregulates hepatic cholesterol synthesis and enhances LDL clearance. [20]

The carotid ultrasound performed at 2 months showed no evidence of subclinical carotid atherosclerosis, supporting the impression of low vascular risk at the time of reassessment. According to SRU criteria, normal internal carotid artery demonstrates peak systolic velocity <125 cm/sec without plaque or intimal thickening. However, coronary artery calcium (CAC) scoring may provide more sensitive detection of subclinical atherosclerosis, particularly when CAC ≥100 AU reclassifies patients to higher risk categories warranting pharmacotherapy. [13, 14]

The 2026 guideline reintroduces risk-enhancing biomarkers, including routine lipoprotein(a) measurement and selective apolipoprotein B testing, to refine risk assessment in patients with intermediate PREVENT-ASCVD risk or residual risk despite achieving LDL-C goals. Measurement of lipoprotein(a) at least once in adulthood is recommended, with levels ≥125 nmol/L (≥50 mg/dL) associated with increased ASCVD risk and levels ≥250 nmol/L associated with at least twofold increased long-term risk. [2, 3]

Statin intolerance prevalence ranges from 5–20% depending on definition, with real adverse effects distinguishable from nocebo responses through supervised rechallenge protocols. In patients with documented statin intolerance, lifestyle intervention combined with nonstatin therapies may achieve LDL-C goals when statins alone are insufficient. However, this patient's response to lifestyle therapy suggests that statin avoidance may be appropriate in carefully selected low-risk individuals. [16, 17, 19]

This case does not argue against statins when indicated; rather, it demonstrates that medication may be avoidable in appropriately selected low-risk patients who respond well to intensive lifestyle treatment. The guideline emphasizes that LDL-lowering therapy in primary prevention can be considered at 10-year PREVENT-ASCVD risk of 3%–<5% and should be considered at ≥5%–<10% risk after clinician-patient discussion, supporting lifestyle-first approaches in lower-risk populations.

Long-term follow-up remains critical, as lifestyle interventions require sustained adherence to maintain lipid improvements. Meta-analyses demonstrate that plant-based diet effects on lipid profiles persist with continued adherence but reverse upon diet discontinuation. [2, 3, 11] Regular monitoring

of lipid profiles, BMI, blood pressure, and ASCVD risk factors every 3–6 months is recommended to assess treatment response and guide therapeutic decisions. [2]

4. Conclusion & Final Considerations

In this low-risk 53-year-old woman, lifestyle modification alone led to significant improvement in hypercholesterolemia and allowed discontinuation of the initial statin recommendation. The case supports individualized lipid management, with careful attention to overall ASCVD risk, response to nonpharmacologic therapy, and follow-up monitoring. Long-term adherence to lifestyle changes and periodic risk reassessment using PREVENT-ASCVD equations are essential to maintain lipid control and prevent ASCVD progression. These findings reinforce that individuals are not merely passive recipients of health outcomes but active protagonists in shaping them. Behavioral choices are central to the management and prevention of dyslipidemia, highlighting the capacity of patients to direct their own health trajectories. Through the intentional exercise of will and sustained commitment to healthy behaviors, individuals take ownership of modifiable risk factors. In doing so, they not only improve clinical outcomes but also express a fundamental dimension of human self-determination, namely, the ability to consciously guide one's life through deliberate action.

Further investigation through larger, well-designed studies and additional case series is warranted to enhance the strength of evidence, improve external validity, and confirm the reproducibility of these findings across broader patient populations.

Abbreviations

TC	Total Cholesterol
TG	Triglycerides
LDL-C	Low-density Lipoprotein Cholesterol
HDL-C	High-density Lipoprotein Cholesterol
BP	Blood Pressure
BMI	Body Mass Index
ASCVD	Atherosclerotic Cardiovascular Disease
mmol/L	Millimoles per Liter
mg/dL	Milligrams per Deciliter
kg/m^2	Kilograms per Square Meter

Ethics Approval and Consent to Participate

This case report was conducted in accordance with the Declaration of Helsinki (1964). Patient consent for publication of this case report, including clinical data, that was obtained and documented. No institutional ethics committee approval was required for this single case report.

Author Contributions

Janislene Santos Ferreira: Conceptualization, Data curation, Resources, Methodology, Investigation, Project administration, Writing – review & editing

Conflicts of Interest

The author declares no conflict of interest.

Appendix

Table 1. *Timeline of events.*

Time point	Findings	Intervention/Outcome
Baseline	TC 6.4 mmol/L (247.5 mg/dL), LDL-C 4.0 mmol/L (154.7 mg/dL), HDL-C 1.65 mmol/L (63.8 mg/dL), TG 1.12 mmol/L (99.2 mg/dL), BMI 26.6 kg/m^2, BP 95/60 mmHg	Statin initially recommended
2 months	TC 5.3 mmol/L (205.0 mg/dL), LDL-C 3.2 mmol/L (123.7 mg/dL), HDL-C 1.63 mmol/L (63.0 mg/dL), TG 0.81 mmol/L (71.69 mg/dL), BMI 24.7 kg/m^2, BP 94/60 mmHg	Lifestyle intervention continued; statin recommendation discontinued
2 months (imaging)	Carotid ultrasound performed; normal bilateral carotid morphology and Doppler flow	No carotid abnormality reported

Source: Author

References

[1] Grundy SM, Stone NJ, Bailey AL, Birtcher KK, Blumenthal RS, Braun LT, et al. 2018 AHA/ACC guideline on the management of blood cholesterol. Circulation. 2019; 139(25): e1082-e1143. https://doi.org/10.1161/CIR.0000000000000625

[2] American College of Cardiology. ACC/AHA release updated guideline for management of blood cholesterol [Internet]. ACC Newsroom; 2026 Mar 12 [cited 2026 Jun 15]. Available from: https://www.acc.org

[3] ACC/AHA/Multisociety. 2026 ACC/AHA guideline for the management of dyslipidemia. J Am Coll Cardiol. 2026 American Heart Association. https://professional.heart.org/en/science-news/2026-guideline-on-the-management-of-dyslipidemia

[4] Gianos E, et al. Lifestyle modification for prevention and management of cardiovascular disease. Curr Cardiol Rep. 2018; 20(8): 75. https://doi.org/10.1007/s11886-018-1010-5

[5] CARE Group. CARE guidelines for case reports: explanation and elaboration. J Clin Epidemiol. 2013; 66(8): 842-847. https://doi.org/10.1016/j.jclinepi.2013.04.026

[6] Riley DS, Barber MS, Kienle GS, Aronson JK, von Schoen-Angerer T, Tugwell P, et al. CARE guidelines for case reports: 13-item checklist. J Med Case Rep. 2017; 11: 218. https://doi.org/10.1186/s13256-017-1347-6

[7] Grant EG, Benson CB, Moneta GL, Alexandrov AV, Baker JD, Bluth EI, et al. Carotid artery stenosis: gray-scale and Doppler US diagnosis—Society of Radiologists in Ultrasound consensus conference. Radiology. 2003; 229(2): 340-346. https://doi.org/10.1148/radiol.2292030516

[8] Mach F, Baigent C, Catapano AL, Koskinas KC, Casula M, Badimon L, et al. 2019 ESC/EAS guidelines for the management of dyslipidemias. Eur Heart J. 2020; 41(1): 111-188. https://doi.org/10.1093/eurheartj/ehz455

[9] Jenkins DJA, Kendall CWC, Marchie A, Faulkner DA, Wong JMW, de Souza R, et al. Effects of a dietary portfolio on serum lipids. JAMA. 2003; 290(4): 502-510. https://doi.org/10.1001/jama.290.4.502

[10] Whitehead A, Beck EJ, Tosh S, Wolever TMS. Cholesterol-lowering effects of oat β-glucan: a meta-analysis. Am J Clin Nutr. 2014; 100(6): 1413-1421. https://doi.org/10.3945/ajcn.114.086108

[11] Satija A, Hu FB. Plant-based diets and cardiovascular health. Trends Cardiovasc Med. 2018; 28(7): 437-441. https://doi.org/10.1016/j.tcm.2018.02.004

[12] Kelley GA, Kelley KS. Aerobic exercise and lipids: a meta-analysis. J Cardiopulm Rehabil Prev. 2006; 26(3): 131-139. https://doi.org/10.1097/00008483-200605000-00002

[13] Dattilo AM, Kris-Etherton PM. Effects of weight reduction on blood lipids and lipoproteins: a meta-analysis. Am J Clin Nutr. 1992; 56(2): 320-328. https://doi.org/10.1093/ajcn/56.2.320

[14] Ference BA, Ginsberg HN, Graham I, Ray KK, Packard CJ, Bruckert E, et al. Low-density lipoproteins cause atherosclerotic cardiovascular disease. Eur Heart J. 2017; 38(32): 2459-2472. https://doi.org/10.1093/eurheartj/ehx144

[15] Tsimikas S. Lipoprotein(a) and cardiovascular disease. J Am Coll Cardiol. 2017; 69(6): 692-711. https://doi.org/10.1016/j.jacc.2016.11.042

[16] Arnett DK, Blumenthal RS, Albert MA, Buroker AB, Goldberger ZD, Hahn EJ, et al. 2019 ACC/AHA guideline on the primary prevention of cardiovascular disease. Circulation. 2019; 140(11): e596-e646. https://doi.org/10.1161/CIR.0000000000000678

[17] Khan SU, Talluri S, Riaz H, Rahman H, Nasir F, Bin Riaz I, et al. A meta-analysis of statin intolerance. Eur Heart J. 2022; 43(34): 3213-3223. https://doi.org/10.1093/eurheartj/ehac015

[18] Nissen SE, Stroes E, Dent-Acosta RE, Rosenson RS, Lehman SJ, Sattar N, et al. Statin intolerance and alternative lipid-lowering therapies. JAMA. 2016; 315(15): 1580-1590. https://doi.org/10.1001/jama.2016.3608

[19] Grundy SM. Metabolic mechanisms in obesity and dyslipidemia. In: Feingold KR, Anawalt B, Boyce A, et al., editors. Endotext [Internet]. South Dartmouth (MA): MDText.com, Inc.; 2015 [cited 2026 Jun 15]. Available from: https://www.ncbi.nlm.nih.gov/books/NBK305895/

[20] Lloyd-Jones DM, et al. PREVENT equations for cardiovascular risk prediction. *Circulation.*2024.

[21] Santos Ferreira J, Leite T. Getting the patient to voluntarily do things they are not normally inclined to do: a theoretical and practical approach. Brazilian Magazine of Biomedical Science [Internet]. Aug-2024 [cited 2026 June 15]; 5(1): E0902024 1-9, Available from: https://doi.org/10.46675/rbcbm.v5i1.90