CASE SERIES ASIAN JOURNAL OF MEDICAL SCIENCES

Do symptoms arise from hyperlipidemia due to hyperviscosity or through its atherosclerotic consequences? – A case series exploration

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Submission: 19-05-2025 Revision: 30-06-2025 Publication: 01-08-2025

ABSTRACT

Hyperlipidemia does not cause any symptoms itself. Hypercholesterolemia contributes to atherosclerotic plaques inside arteries and obstruction of arteries, producing clinical manifestations. Very severe hypertriglyceridemia can cause acute pancreatitis. The three patients with hyperlipidemia in the present study had complaints of chest pain, breathlessness, limb aches, tingling sensations, and sleepiness without any evidence of atherosclerosis. The possible explanation of the visible symptoms in the patients can be due to a mild increase in blood viscosity due to hyperlipidemia. However, further intensive studies may be required to comprehend the direct symptoms of hyperlipidemia towards early detection and intervention to reduce the burden on healthcare facilities.

Key words: Hyperlipidemia; Hyperviscosity; Symptoms

Access this article online

Website: https://ajmsjournal.info/index.php/AJMS/index

DOI: 10.71152/ajms.v16i8.4631

E-ISSN: 2091-0576

P-ISSN: 2467-9100

INTRODUCTION

Hyperlipidemia is defined as an increase in the serum level of lipids such as cholesterol and triglycerides. Hyperlipidemia does not cause any symptoms itself.[1] It is often detected incidentally on routine blood testing. Hypercholesterolemia is a known cause of atherosclerosis as it forms plaques inside the arterial walls and obstruction of major arteries supplying the heart, brain, kidneys, and peripheries.[2] Moreover, subsequent haemodynamic stress induced endothelial damage, rupture of plaque, and superimposed thrombosis with obstruction of arteries, also producing clinical manifestations.[2] There is no evidence of a direct link between hypertriglyceridemia and atherosclerosis. Very severe hypertriglyceridemia of more than 1000 mg/dL can cause acute pancreatitis. The other less common manifestation from extremely high lipid levels is lipid deposits in skin, xanthelasma; in tendons, xanthoma, and creamy appearance of retinal vessels, lipemia retinalis in very severe hypertriglyceridemia. Here in this present article, we present three case studies having symptomatic hyperlipidemia due to hyperviscosity without any evidence of atherosclerosis, including known less common manifestations. Those manifestations in our article are not much discussed in the literature.

CASES

Patient 1

A 30-year-old female attended the outpatient department of our hospital in August 2023 with complaints of

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heaviness in the central chest and limb aches for 7 days. She was also complaining of tingling sensations in both hands with increased sleepiness for the past 1 month. On examination, her pulse and blood pressure were found to be 70/min and 130/80 mm of Hg, respectively, with normal systemic examination findings. On investigation, the complete blood count, fasting sugar, renal function test, liver function test, and troponin I level were seen as normal. The total cholesterol, low-density lipoprotein (LDL) cholesterol, high-density lipoprotein (HDL) cholesterol, and triglycerides were 294 mg/dL, 205 mg/dL, 62 mg/dL, and 158 mg/dL, respectively. The electrocardiogram and chest skiagram were also normal. Because of chest heaviness and hypercholesterolemia, a computed tomographic coronary angiogram was done. The report was normal. She was treated with lifestyle modification, atorvastatin 20 mg at night daily, and metoprolol 25 mg/day for chest pain. After 3 months, she was asymptomatic, and LDL serum cholesterol was 92 mg/dL

Patient 2

A 39-year-old non-smoking male with a past history of hypertension presented to the emergency department of our hospital in February 2024 with a complaint of compressive chest pain for the last 18 h. The examination findings were pulse 92/min and blood pressure 138/82 mmHg. The chest X-ray, electrocardiogram, and echocardiogram reports were normal. The laboratory reports were hemoglobin 14.3 g/dL, white blood cell 6200/cumm, platelet 3.1 lakh/cumm, urea 23 mg/dL, creatinine 0.9 mg/dL, fasting sugar 95 mg/dL, alanine aminotransferase (ALT) 23 U/L, aspartate aminotransferase (AST) 29 U/L, total cholesterol 382 mg/dL, triglyceride 845 mg/dL, LDL cholesterol 152 mg/dL and HDL cholesterol 53 mg/dL. The computerized tomographic coronary angiogram showed normal coronary arteries. He was discharged with the advice of lifestyle modification, atorvastatin 10 mg/day, fenofibrate 160 mg/day, and metoprolol 25 mg/day for chest pain. After 3 months of treatment, he was asymptomatic with total cholesterol 187 mg/dL, triglyceride 212 mg/dL, and LDL cholesterol 93 mg/dL.

Patient 3

A 35-year-old male smoker came to the outpatient department of our hospital in December 2024 with complaints of central chest pain and breathlessness on exertion for the previous 3 weeks. On examination, his pulse and blood pressure were 88/min and 134/88 mmHg, respectively. The respiration rate and oxygen saturation were 20/min and 98%, respectively. The systemic examination findings were normal. The investigatory report, such as complete blood count, fasting sugar, urea, and creatinine, was also normal. The liver function test and lipid profile report showed total bilirubin 0.86 mg/dL, ALT 45 U/L, AST 48 U/L, alkaline phosphatase 86 U/L, serum protein 6.5 g/dL, total cholesterol 321 mg/dL, LDL cholesterol 230 mg/dL, HDL cholesterol 42 mg/dL, triglyceride 234 mg/dL. The chest skiagram, electrocardiogram, and computerized tomographic coronary angiogram reports were normal. The ultrasound of the whole abdomen showed grade I fatty liver. Hence, the diagnosis was hyperlipidemia with nonalcoholic fatty liver disease. He was advised to undergo lifestyle modification and a dosage of atorvastatin 20 mg/day. The repeat blood reports after 12 weeks were total cholesterol 202 mg/dL, LDL cholesterol 98 mg/dL, triglyceride 192 mg/dL, ALT 32 mg/dL, and AST 36 mg/dL, and he was free from his symptoms.

DISCUSSION

Hyperlipidemia can be divided into two types based on etiology. The primary hyperlipidemia is caused by a genetic mutation that affects the metabolism of lipids. Some of the examples of primary hyperlipidemia are familial hypercholesterolemia, familial hypertriglyceridemia, and familial combined hyperlipidemia. The estimated prevalence of familial hypercholesterolemia ranges from 1 in 500 to 1 in 250 in most population.[1] The prevalence of familial hypertriglyceridemia and familial combined hyperlipidemia in the general population is 1 in 500 and 1 in 100, respectively.[1] Some examples of secondary hyperlipidemia risk factors are physical inactivity, unhealthy nutrition, obesity, diabetes, hypothyroidism, chronic kidney disease, liver disease, alcohol abuse, smoking, and the use of certain drugs.[3] The comprehensive data on the epidemiology of hyperlipidemia is currently lacking in different populations and settings, as hyperlipidemia is usually asymptomatic, and it is difficult to estimate the prevalence.[1] The patient with underlying hyperlipidemia usually remains asymptomatic.[4] The major clinical consequences are atherosclerotic cardiovascular diseases of the coronary artery (coronary artery disease), manifesting as angina and myocardial infarction, cerebral artery (cerebrovascular disease), manifesting as cerebral infarction and transient ischemic attacks, and peripheral artery (peripheral vascular disease), manifesting as intermittent claudication and critical limb ischemia.

The three patients in the present study had complaints of chest pain, breathlessness, limb aches, tingling, and sleepiness. There is a report that hypercholesterolemia is associated with increased blood viscosity.[5] In some cases of severe hypertriglyceridemia, symptoms or complications due to hyperviscosity were observed.[6-8] The viscosity of a fluid is measured by a fluid's internal resistance to flow, or it can be thought of as the thickness or stickiness of a fluid. When the viscosity of blood increases, it travels

slowly, and there is decreased microvascular circulation and hypoperfusion of tissues.[9]

Severe increase in viscosity of blood causes hyperviscosity syndrome that classically presents with triad of neurological deficits, visual changes, and mucosal bleeding.[9] However, a mild increase in viscosity leads to many symptoms, such as chest pain, breathlessness, headache, dizziness, ataxia, tingling sensation, hearing impairment, and blurred vision, due to decreased blood supply to tissues.[9] Rosenson et al. mentioned that elevated triglyceride levels raise the plasma viscosity, which may decrease blood flow to the target tissue.[10] Zhou et al. described one patient with metastatic rectal carcinoma who presented with chest pain and hypertriglyceridemia in the emergency department, with normal coronary arteries in angiogram.[11] Therefore, a mild increase in blood viscosity due to hyperlipidemia can be the possible reason for the symptoms as observed in the patients in the present study.

CONCLUSION

At present, a routine lipid screening, especially in the high-risk population, is the usual norm for early detection and effective management of hyperlipidemia. As hyperlipidemia often progresses silently, understanding its symptoms is crucial for timely intervention and preventing further complications. The symptoms of mild hyperviscosity due to hyperlipidemia, such as chest pain, breathlessness, limb aches, tingling, and sleepiness, observed in the present study, will redefine the criterion for lipid screening. More intensive studies are however suggested for a detailed and better understanding of symptoms of hyperlipidemia which may help in early detection and positive medical intervention.

ACKNOWLEDGMENT

The authors take this opportunity to thank patients and Jalpaiguri Government Medical College, West Bengal, for their wholehearted support in publishing this article.

REFERENCES

1. Pappan N, Awosika AO and Rehman A. Dyslipidemia. In: Statpearls. Treasure Island, FL: Statpearls Publishing; 2025. Available from: https://www.ncbi.nlm.nih.gov/books/nbk560891 [Last accessed on 2024 Mar 04].
2. Sloop GD. A critical analysis of the role of cholesterol in atherogenesis. Atherosclerosis. 1999;142(2):265-268. https://doi.org/10.1016/s0021-9150(98)00270-6
3. Saavedra A, Rodrigues E and Carvalho D. Dyslipidemia secondary to hypothyroidism and cholestasis. Acta Med Port. 2020;33(3):204-207. https://doi.org/10.20344/amp.9944
4. Hill MF and Bordoni B. Hyperlipidemia. In: Statpearls. Treasure Island, FL: Statpearls Publishing; 2025. Available from: https://www.ncbi.nlm.nih.gov/books/nbk559182 [Last accessed on 2023 Aug 08].
5. Ambrosi P and Juhan-Vague I. Dyslipidemia, lipid lowering drugs and thrombosis. Arch Mal Coeur Vaiss. 1995;88(11):1641-1645.
6. Stone NJ, Robinson JG, Lichtenstein AH, Bairey Merz CN, Blum CB, Eckel RH, et al. 2013 ACC/AHA guideline on the treatment of blood cholesterol to reduce atherosclerotic cardiovascular risk in adults: A report of the American college of cardiology/American heart association task force on practice guidelines. J Am Coll Cardiol. 2014;63(25 Pt B):2889-2934. https://doi.org/10.1016/j.jacc.2013.11.002
7. Jaross W, Assmann G, Bergmann S and Schulte H. Comparison of risk factors for coronary heart disease in Dresden and Münster. Results of the DRECAN (Dresden cardiovascular risk and nutrition) study and the PROCAM (prospective cardiovascular Münster) study. Eur J Epidemiol. 1994;10(3):307-315. https://doi.org/10.1007/BF01719355
8. Yang AL and McNabb-Baltar J. Hypertriglyceridemia and acute pancreatitis. Pancreatology. 2020;20(5):795-800. https://doi.org/10.1016/j.pan.2020.06.005
9. Perez RA and Estes M. Hyperviscosity syndrome. In: Statpearls. Treasure Island, FL: Statpearls Publishing; 2025. Available from: https://www.ncbi.nlm.nih.gov/books/nbk518963 [Last accessed on 2023 Mar 13].
10. Rosenson RS, Shott S, Lu L and Tangney CC. Hypertriglyceridemia and other factors associated with plasma viscosity. Am J Med. 2001;110(6):488-492. https://doi.org/10.1016/s0002-9343(01)00643-x
11. Zhou W, Ni L, Luo M, Shu X, Cao P and Qiu M. Rare capecitabine-induced acute hypertriglyceridemia with angina: A case report and review of the literature. Ann Palliat Med. 2022;11(6):2152-2156. https://doi.org/10.21037/apm-21-1109

Authors' Contribution:
AC- Literature survey, data collection, and manuscript preparation; **SuB**- Data collection and coordination, manuscript revision; **SM**- Literature survey and review manuscript; **AS**- Concept, design, literature survey, prepared first draft of manuscript, and manuscript revision; **SK**- Literature survey and data collection, **SmB**- Review manuscript.

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Source of Support: Nil, **Conflicts of Interest:** None declared.