RESEARCH PAPER

Estimation Of Serum Cholesterol Binding Reserve In Patients With Essential Hypertension

Hemant Jha [1], Pushpendra Mewada [2], Jayanti Kumari [3], Aakanksha Pawar [4], Akhilesh Anand[5] Manish Shrivastava [6], Surendra Mewada [7]

[1]*Assistant Professor at Jai Narayan College of Paramedical, Bhopal, JNCT Professional university Bhopal, MP. Hemantjha112211@gmail.com*
[2]*Assistant professor at Jai Narayan College of Paramedical. Bhopal, JNCT professional University Bhopal, MP pmmewada1997@gmail.com*
[3]*Assistant Professor at Jai Narayan College of Paramedical Bhopal, JNCT Professional university bhopal, MP. jayantitiwary6@gmail.com*
[4.]*Assistant Professor at Jai Narayan College of Paramedical Bhopal, JNCT Professional university Bhopal MP. aakankshapawar2022@gmail.com*
[5]*Assistant Professor at Jai Narayan College of Paramedical Bhopal, JNCT Professional university bhopal MP anandakhilesh07@gmail.com*
[6.]*Principal at Jai Narayan College of Paramedical Bhopal, JNCT Professional university Bhopal MP. manishshrivastava259@gmail.com*
[7.]*Junior Physiotherapist at RK Physiotherapy & Neuro solution Kohe Fiza. Surendramewada088@gmail.com*

ABSTRACT
Background: Atherosclerosis is a major cause of cardiovascular morbidity and mortality. Serum cholesterol binding reserve (SCBR) reflects the capacity of serum lipoproteins to solubilize additional cholesterol and is considered an indirect marker of reverse cholesterol transport. Reduced SCBR has been linked to increased atherogenic risk.
Objective: To estimate serum cholesterol and SCBR in patients with essential hypertension and to compare the findings with healthy age- and sex-matched controls.
Materials and Methodology: The study included 50 patients with essential hypertension and 25 healthy controls. Fasting serum cholesterol was measured by an enzymatic method. SCBR was assessed by incubating serum with crystalline cholesterol and determining the increase in cholesterol concentration after incubation. Statistical analysis was performed to compare mean values between groups and to assess correlations.
Results: Mean serum cholesterol levels were significantly higher in hypertensive patients (208.88 ±37.71 mg/dL) than in controls (186.80 ± 32.01 mg/dL) ($p < 0.05$). Mean SCBR was markedly lower in hypertensive patients (24.50 ± 6.24 mg/dL) compared to controls (59.72 ± 6.99 mg/dL) ($p < 0.001$). A strong negative correlation was observed between serum cholesterol and SCBR in hypertensive patients ($r = –0.876$), whereas a positive correlation was noted in controls ($r = +0.706$).
Conclusion: Patients with essential hypertension exhibit significantly reduced SCBR despite only modest elevations in serum cholesterol. Reduced SCBR may contribute to accelerated atherogenesis in hypertension and may serve as a better biochemical marker of atherogenic risk than serum cholesterol alone...
Keywords: Essential hypertension; Serum cholesterol; Serum cholesterol binding reserve; Reverse cholesterol transport; Atherosclerosis..
How to cite this article: Jha H, Mewada P, Kumari J, Pawar A, Anand A, Shrivastava M, Mewada S, Estimation Of Serum Cholesterol Binding Reserve In Patients With Essential Hypertension..Int J Drug Deliv Technol. 2026;16 (13s): 68-71; DOI: 10.25258/ijddt.16.13s.8
Source of support:. None
Conflict of interest: None

INTRODUCTION

Atherosclerosis is a chronic, progressive disease of the arterial wall characterized by lipid accumulation, inflammatory cell infiltration, smooth muscle cell proliferation, and fibrous plaque formation. It is the underlying pathology in most cases of coronary artery disease, cerebrovascular accidents, and peripheral vascular disease, making it a leading cause of morbidity and mortality worldwide. The term “atherosclerosis” was introduced by Marchand in 1904 to describe lipid-rich lesions within the arterial intima.[1] Extensive research has established a strong association between dyslipidemia, hypertension, and the development of atherosclerotic cardiovascular disease.[2,3,4]

Cholesterol plays a central role in atherogenesis. Low-density lipoproteins (LDL) are the primary carriers of cholesterol from the liver to peripheral tissues and are considered highly atherogenic when present in excess.[5,6] LDL particles can penetrate the arterial intima, undergo oxidative modification, and be taken up by macrophages to form foam cells, which are the hallmark of early atherosclerotic lesions.[7,8] In contrast, high-density

**Author for Correspondence: jayantitiwary6@gmail.com*

lipoproteins (HDL) are anti-atherogenic and facilitate the efflux of cholesterol from peripheral tissues and macrophages back to the liver through the process known as reverse cholesterol transport (RCT).[9,10] Epidemiological studies have demonstrated an inverse relationship between HDL levels and the risk of coronary heart disease.[11,1,10]

Beyond absolute cholesterol concentrations, the functional capacity of serum lipoproteins to bind and transport cholesterol is increasingly recognized as an important determinant of atherogenic risk. Hsia et al. (1975) demonstrated that human serum is capable of solubilizing additional exogenous cholesterol beyond its endogenous content.[12] This property was termed serum cholesterol binding reserve (SCBR) and was proposed as an indirect biochemical index of the efficiency of reverse cholesterol transport.[13,12] Subsequent studies reported significantly lower SCBR values in patients with atherosclerosis and coronary heart disease, suggesting that impaired cholesterol-binding capacity of serum may contribute to lipid accumulation within the arterial wall.[14,12]

Hypertension is a major independent risk factor for atherosclerosis and cardiovascular disease. Persistently elevated blood pressure induces mechanical stress on the vascular endothelium, leading to endothelial dysfunction, increased vascular permeability, and enhanced infiltration of lipoproteins into the arterial wall.[15,3] Hypertension is also associated with increased oxidative stress and inflammatory responses, which further promote LDL oxidation and foam cell formation.[16,17] When no identifiable secondary cause is present, the condition is classified as essential (primary) hypertension. Essential hypertension accounts for approximately 90–95% of all cases of hypertension.[4]

Several studies have demonstrated that hypertensive patients exhibit qualitative and quantitative alterations in plasma lipoproteins, even when total cholesterol levels are within the normal range.[16,18] These alterations may impair reverse cholesterol transport and enhance atherogenic risk. The interaction between hypertension and lipid metabolism is therefore complex and multifactorial.[1,18] While serum cholesterol estimation is routinely used in clinical practice to assess cardiovascular risk, it does not fully reflect the functional capacity of serum to transport cholesterol.[19]

In this context, the estimation of serum cholesterol binding reserve may provide additional insight into the atherogenic milieu in essential hypertension. A reduced SCBR indicates a diminished capacity of serum lipoproteins, particularly HDL, to bind and transport excess cholesterol, thereby favoring cholesterol deposition within the arterial wall.[14,12] Despite its potential clinical relevance, SCBR has not been widely evaluated in hypertensive populations.

The present study was therefore undertaken to estimate serum cholesterol and serum cholesterol binding reserve in patients with essential hypertension and to compare the findings with those of healthy age- and sex-matched controls. The study also aimed to examine the correlation between serum cholesterol and SCBR and to assess whether SCBR may serve as a better biochemical indicator of atherogenic risk in essential hypertension than serum cholesterol alone.[13,12,19]

Materials and Methods

Study Design

A hospital-based, cross-sectional comparative study.

Study Area

The study was conducted in the Department of Biochemistry, Mayo Hospital, Bhopal.

Study Period

The study was carried out over a period of 12 months.

Study Population

The study population comprised patients diagnosed with essential hypertension attending the outpatient department and healthy individuals serving as controls.

Sample Size

A total of 75 participants were included in the study: 50 patients with essential hypertension (cases) and 25 healthy age- and sex-matched individuals (controls).

Inclusion Criteria

For Cases

Patients aged 30–65 years.

Blood pressure ≥ 140/90 mmHg on at least two separate occasions.

Clinical diagnosis of essential (primary) hypertension.

Willingness to participate and provide informed consent.

For Controls

Healthy individuals aged 30–65 years.

Normal blood pressure (< 130/85 mmHg).

No history of hypertension, diabetes, ischemic heart disease, or dyslipidemia.

Exclusion Criteria (for both groups)

Diabetes mellitus

Renal disease

Endocrine disorders

Ischemic heart disease

Obesity

Smoking

Secondary hypertension

Investigations

All participants were evaluated in the fasting state. The following investigations were performed: - Urine routine and microscopy - Blood urea and fasting blood sugar - Serum creatinine - 12-lead electrocardiogram (ECG) - Serum cholesterol - Serum cholesterol binding reserve (SCBR)

Estimation of Serum Cholesterol

Principle

Cholesterol esters are hydrolysed by cholesterol esterase. Free cholesterol is oxidized by cholesterol oxidase to cholest-4-en-3-one with the formation of hydrogen peroxide. In the presence of peroxidase, hydrogen peroxide reacts with 4-aminoantipyrine and phenol to form a red quinoneimine dye. The intensity of the color is directly proportional to the cholesterol concentration.

Procedure

Serum cholesterol was measured using an enzymatic method with a cholesterol reagent and a standard (200 mg/dL). Absorbance was read at 505 nm, and cholesterol concentration was calculated using standard formulae.

Estimation of Serum Cholesterol Binding Reserve (SCBR)

Principle

SCBR represents the capacity of serum to solubilize additional cholesterol. Serum is incubated with finely powdered cholesterol, and undissolved cholesterol is removed by filtration. The increase in serum cholesterol concentration after incubation reflects SCBR.

Procedure

Two aliquots of serum (1 mL each) were used. One aliquot was incubated with powdered cholesterol, and the other served as a control. After incubation at 37°C for 16 hours, undissolved cholesterol was filtered out, and cholesterol was measured in both samples. SCBR was calculated as the difference between test and control values.

Results

Serum Cholesterol

Mean serum cholesterol was significantly higher in hypertensive patients (208.88 ± 37.71 mg/dL) compared to controls (186.80 ± 32.01 mg/dL) ($p < 0.05$). There was no statistically significant difference in cholesterol levels between males and females in either group.

Serum Cholesterol Binding Reserve

Mean SCBR was significantly lower in hypertensive patients (24.50 ± 6.24 mg/dL) compared to controls (59.72 ± 6.99 mg/dL) ($p < 0.001$). No significant sex-related differences were observed.

Correlation Analysis

In hypertensive patients, a strong negative correlation was observed between serum cholesterol and SCBR ($r = –0.876$, $p < 0.001$). In contrast, a strong positive correlation was noted in controls ($r = +0.706$, $p < 0.001$).

Discussion

The present study demonstrates that patients with essential hypertension have significantly reduced serum cholesterol binding reserve (SCBR) compared to healthy controls, despite only modest elevations in serum cholesterol. This suggests that impaired cholesterol-binding capacity of serum lipoproteins may contribute to accelerated atherogenesis in hypertension. The negative correlation between serum cholesterol and SCBR in hypertensive patients indicates that as cholesterol levels rise, the ability of serum to accommodate additional cholesterol diminishes, thereby favoring cholesterol deposition within the arterial wall.[13,12]

These findings are in agreement with earlier studies by Hsia et al. (1975) and Borresen and Berg (1981), who reported significantly lower SCBR values in patients with atherosclerosis and coronary heart disease.[14,12] Similar observations have been made in studies evaluating reverse cholesterol transport and HDL functionality, which emphasize that qualitative aspects of lipoproteins are as important as quantitative lipid measurements.[9,18] Miller and Miller (1975) demonstrated an inverse relationship between HDL levels and ischemic heart disease, highlighting the protective role of efficient cholesterol transport mechanisms.[10]

Recent literature has also underscored that hypertension is associated with endothelial dysfunction and oxidative stress, which impair HDL-mediated cholesterol efflux, thereby reducing SCBR.[15,16,17] Collectively, these findings support the concept that reduced SCBR reflects dysfunctional reverse cholesterol transport and may serve as an early biochemical indicator of atherogenic risk in hypertensive patients.[13,12]

The present study further confirms that patients with essential hypertension exhibit significantly reduced SCBR compared to healthy controls, despite only modest elevations in serum cholesterol. This observation reinforces the view that impaired cholesterol-binding capacity of serum lipoproteins contributes to accelerated atherogenesis in hypertension.[13,12]

The negative correlation between serum cholesterol and SCBR in hypertensive patients indicates that as cholesterol levels rise, the ability of serum to accommodate additional cholesterol diminishes. In contrast, healthy individuals show an adaptive increase in SCBR with rising cholesterol levels, consistent with efficient reverse cholesterol transport.[9,18]

These findings are in agreement with earlier studies by Hsia et al. (1975), which reported lower SCBR values in patients with atherosclerosis and coronary heart disease.[12] Similar reductions in SCBR have also been observed in conditions associated with enhanced atherogenic risk, including nephrotic syndrome and steroid therapy, further supporting the role of impaired cholesterol-binding capacity in cardiovascular pathology.[20,21]

Conclusion

The present study indicates that patients with essential hypertension exhibit a significant reduction in serum cholesterol binding reserve, even in the presence of only modest elevations in serum cholesterol levels. This impaired cholesterol-binding capacity of serum lipoproteins may contribute to accelerated atherogenesis and increased cardiovascular risk in hypertension. The strong inverse relationship between serum cholesterol and SCBR further emphasizes the potential role of SCBR as a more sensitive biochemical marker of atherogenic risk than serum cholesterol alone. Assessment of SCBR, therefore, may provide additional insight into lipid metabolism abnormalities and cardiovascular risk stratification in patients with essential hypertension.

CONFLICT OF INTEREST: The authors declare that there are no conflicts of interest related to this research work. No financial, personal, or professional relationships have influenced the findings, analysis, or conclusions presented in this study.

FUNDING: This research paper was fully funded by the authors. No external financial support was received

REFERENCE

1. Castelli WP, Doyle JT, Gordon T, et al. HDL cholesterol and other lipids in coronary artery disease. Circulation. 1977;55:767-772.

2. Bierman EL. Atherosclerosis and other forms of arteriosclerosis. In: Harrison's Principles of Internal

Medicine. 10th ed. p.1465.
3. Williams GH, Braunwald E. Hypertensive vascular disease. In: Harrison's Principles of Internal Medicine. 10th ed. p.1475. (3)
4. World Health Organization. Hypertension and coronary heart disease. Technical Report Series No. 231. (4)
5. Barr DP, Russ EM, Eder HA. Protein-lipid relationships in human plasma. II. Atherosclerosis and related conditions. Am J Med. 1951;11:480-493. (5)
6. Brown HS, Goldstein JL. The hyperlipoproteinemias and other disorders of lipid metabolism. In: Harrison's Principles of Internal Medicine. 10th ed. p.547.(6)
7. Smith EB, Slater R. Chemical and immunological assay of LDL extracted from human aortic intima. Atherosclerosis. 1970;11:417-438. (7)
8. Bhat R, Shanbhag P. Knowledge, Attitude, and Practice Study on Cardiovascular Disease Risk Factors in the Mangalore Community. Oral Sphere J. Dent. Health Sci. 2025;1(1):19-28. doi: https://doi.org/10.63150/osjdhs.2025.32
9. Havel RJ. HDL cholesterol transport and coronary heart disease. Circulation. 1970;60:1-3.(9)
10. Miller GJ, Miller NE. Plasma high density lipoprotein concentration and development of ischaemic heart disease. Lancet. 1975;1:16-19. (10)
11. Blackburn H. The meaning of a new marker for coronary artery disease. N Engl J Med. 1983;309:426-428. (11)
12. Hsia SL, Chao YS, Hennekens CH, Reader WB. Decreased serum cholesterol binding reserve in premature myocardial infarction. Lancet. 1975;2:1000-1014. (12)
13. Hsia SL, Brisse F, Hoffman J. Cholesterol binding reserve and myocardial infarction. Lancet. 1976;1:799.(13)
14. Borreson AL, Berg K. Serum reserve cholesterol binding capacity: the relative importance of different lipoproteins. Artery. 1981;9(2):96-119.(14)
15. Borst JGG, Borst de Geus A. Hypertension explained by Sterling's theory of circulatory homeostasis. Lancet. 1963;1:677.(15)
16. Day JL, Metcalfe J, Simpson CW. Adrenergic mechanisms in control of plasma lipid concentrations. Br Med J. 1982;284:1145.(16)
17. Hammer J. Modern management of hypertension. In: Hammer J, Rowlands DJ, editors. Recent Advances in Cardiology. Edinburgh: Churchill Livingstone; 1981. p.1. (17)
18. Krauss RM. Regulation of HDL levels. Med Clin North Am. 1982;66:403-430.(18)
19. Tuelley SB. Choice and use of blood lipid tests. Arch Intern Med. 1983;143:667-673. (19)
20. Perez OG, Levine E, Gomez E, Hsia SL. SCBR in patients with the nephrotic syndrome. Nephron. 1979;24:146-149. (20)
21. Roth MS, Donsto DN, Lansman HH, Robertson EG, Hsia SL, Le Maire WJ. Effect of steroids on serum lipids and SCBR. Am J Obstet Gynecol. 1978;132:151-156. (21).