RESEARCH ARTICLE

Correlation of Serum Cholesterol and S.C.B.R. in Control study of Essential Hypertension Patient

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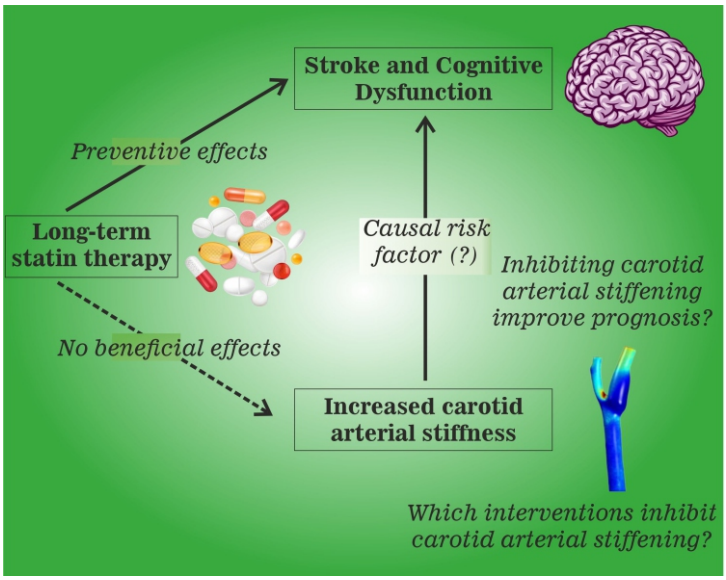


KEYWORDS: SCBR, EDTA, Atherosclerosis, Cholesterol, Serum Cholesterol.

Abstract

Introduction:
Arteriosclerosis, a Genetic term for Thickening and Hardening of the Arterial Wall is now responsible for the Majority of Deaths. Arthrosclerosis" was First Introduced by Marchand In 1904. Atherosclerosis may be Accelerated by a Decreased Clearance of Cholesterol from Arterial Wall Secondary to a Reduction of Plasma Concentration of H.D.L. In Atherosclerotic Lesion, the Lipid Content of Elastin increases Progressively with increasing Severity of Atherosclerosis. The Increases in the Lipid Content of Plaque Elastin are Mainly due to large increases in Cholesterol.

Aim: Correlation of Serum Cholesterol and S.C.B.R. in Control study of Essential Hypertension.
Inclusion Criteria:25 patients of essential hypertension.
Exclusion Criteria: Nonsmokers and Secondary Hypertension.

Methodology: Obtain Serum specimen in usual way, alternatively EDTA plasma may also be used as specimen. The sample should preferable be 12- 14 hours fasting and using the principle of Serum Cholesterol and SCBR, readings has come out, the readings are further calculated.

Result: Mean S.C.B.R. was 59.719±6.99 mg% and mean serum cholesterol was 186.8±32.009 mg% in 25 controls. Correlation coefficient between serum cholesterol and S.C.B.R. was +0.706 ($p < 0.001$), denoting that S.C.B.R. rises with increasing level of cholesterol in control.

Conclusion: The study shows significance coo-relation between Serum cholesterol and S.B.C.R

INTRODUCTION

Abnormalities in serum lipid and lipoprotein levels (dyslipidemia) are recognized as major modifiable cardiovascular disease (CVD) risk factors[1]. Dyslipidemia is more common in untreated hypertensives than normotensives, and lipid levels increase as BP increases[2,3]. Hypertension is known to be associated with alterations in lipid metabolism which gives rise to abnormalities in serum lipid and lipoprotein levels. It has also been documented that presence of hyperlipidemia substantially worsens the prognosis in hypertensive patients[4]. The frequent clustering of hypertension, lipid abnormalities, and other metabolic abnormalities, in an individual has been clearly demonstrated to be synergistic in accelerating atherosclerosis and development of CVD[5].Many studies have shown, Atherosclerosis patient blood report have cholesterol in high quantity. The ability of serum to bind cholesterol in soluble lipoprote in complexes referred the deposition of cholesterol in atherosclerotic plaques. This ability by serum cholesterol binding reserve (SCBR), defined as the amount of cholesterol which a serum specimen is capable of solubilizing in addition cholesterol content. The cholesterol content of human serum is normally below the full capacity of the serum lipoproteins for binding cholesterol. Normal human serum can solubilize considerable amount of exogenous cholesterol in addition to its cholesterol content. This capability was known as serum cholesterol binding reserve (SCBR)[8]. Accordingly, the serum cholesterol binding reserve, (herein designated SCBR) which is a measure of this ability should be a useful factor for predicting the risk of coronary heart disease.

Aim:

Correlation between levels of serum cholesterol and S.C.B.R. in control study of

Hypertension.

Objective:

To find the association between Serum Cholesterol and S.C.B.R in Hypertensive Patient.

Methodology:

25 Patient selected for controls study from 50 patients of essential hypertension.

These patients were selected from outpatient, Department of Biochemistry, R.G.P.V., Bhopal (M.P.).

Inclusion Criteria:

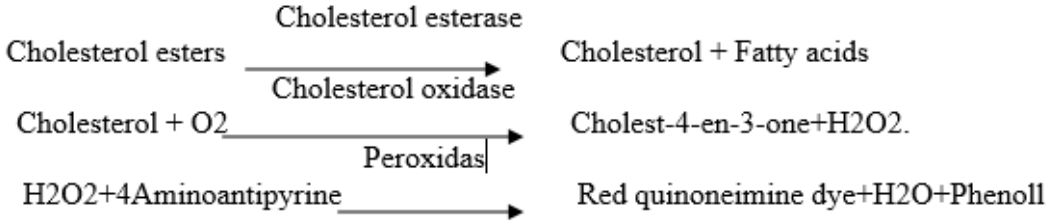
25 patients of essential hypertension. 19 males (76%) and 6 female (24%) in control group on the basis of raised B.P. greater than or equal to 140/80 mm of Hg and evidence of increased vascular reactivity.

Exclusion Criteria:

Nonsmokers and Secondary Hypertension.

Procedure:

Cholesterol esters are hydrolyzed by choles--terol esterase. The free cholesterol produced is oxidized by cholesterol oxidase to form cholest-4-en- 3-one with simultaneous prod--uction of hydrogen peroxide which oxidati--vely couples with 4 aminoantipyrine and phenol in presence of peroxidase to yield a red colored quinoneimine dye complex. Intensity of the colour formed is directly pro--portional to the amount of cholesterol present in the sample.



Reagents Provided

Cholesterol Reagent : Enzyme Reagent

Cholesterol Standard : 200 mg/dl

Pipette into clean and dry test tubes labeled as Blank (B), Standard (S), and Test (T). :

Additional Sequence	Blank	Standar	Test
Cholesterol Reagent	1000 l	1000 l	1000
Distilled Water	10 l	-	-
Cholesterol Standard	-	10 l	-
Specimen	-	-	10 l

- Mix well and incubate at 37^0 C for 5 min or at RT (25^{0}C) for 15 min.
- Measure the absorbance of Standard (A--bs. S) and Test Sample (Abs. T) against the blank at 505 nm.
- Color is stable for 60 minutes when pr--otected from light so absorbance should be measured within that period.

$$\text{Mg. Cholesterol / 100 ml Serum} = \frac{\text{(Abs) OD. Of Test}}{\text{(Abs) O.D. of Standard}} \times 200$$

(O.D. = Optic Density)

Reference Values:

Serum/Plasma : 150-260 mg/dl

Estimation of S.C.B.R.

S.C.B.R. implies the capacity of serum to solubilize exogenously added cholesterol. The S.C.B.R. can be measured by incubating the serum with finely powdered cholesterol for suitable length of the time and the removing the undissolved cholesterol by filtration. The increase in value of "Serum cholesterol", observed after incubation shall represent S.C.B.R.

About 2 ml, serum was collected in each case 1 ml. of the serum was taken in a test tube labeled (T) and a Pinch of cholesterol (15-20 mg) sonicated to a particle size of 10-60 milli micron was added to it and mixed. Another 1 ml of serum of same individual was taken in a separate test tube labeled (c) to which no cholesterol was added. Both the test tubes were then rotated continuously at a speed of 40 RPM for about 16 hours in side an incubator maintained at 37^0 C. After 16 hours of incubation, the tubes were taken out and the serum was filtered carefully using small piece of 42 mm. Whatman Grade Filter Paper, so as to remove all the undissolved particles of cholesterol.

Total cholesterol was estimated in both the tubes simultaneously and

S.C.B.R. was calculated as follows :-

S.C.B.R. (in mg%) = Cholesterol level in Test -Tube marked T) (Cholesterol level in test tube marked C).

Result:

TABLE NO. 1: Showing distribution of cases and control according to sex.

Sex	Cases	%	Control	%
Male	34	68	19	76
Female	16	32	6	24

Total number of cases were 50, while control were 25 in number. Male Compared 68% in case group and 76% in control group. Female were 32% in case group and 24% in control group.

GRAPH 1: Showing distribution of cases and control according to sex

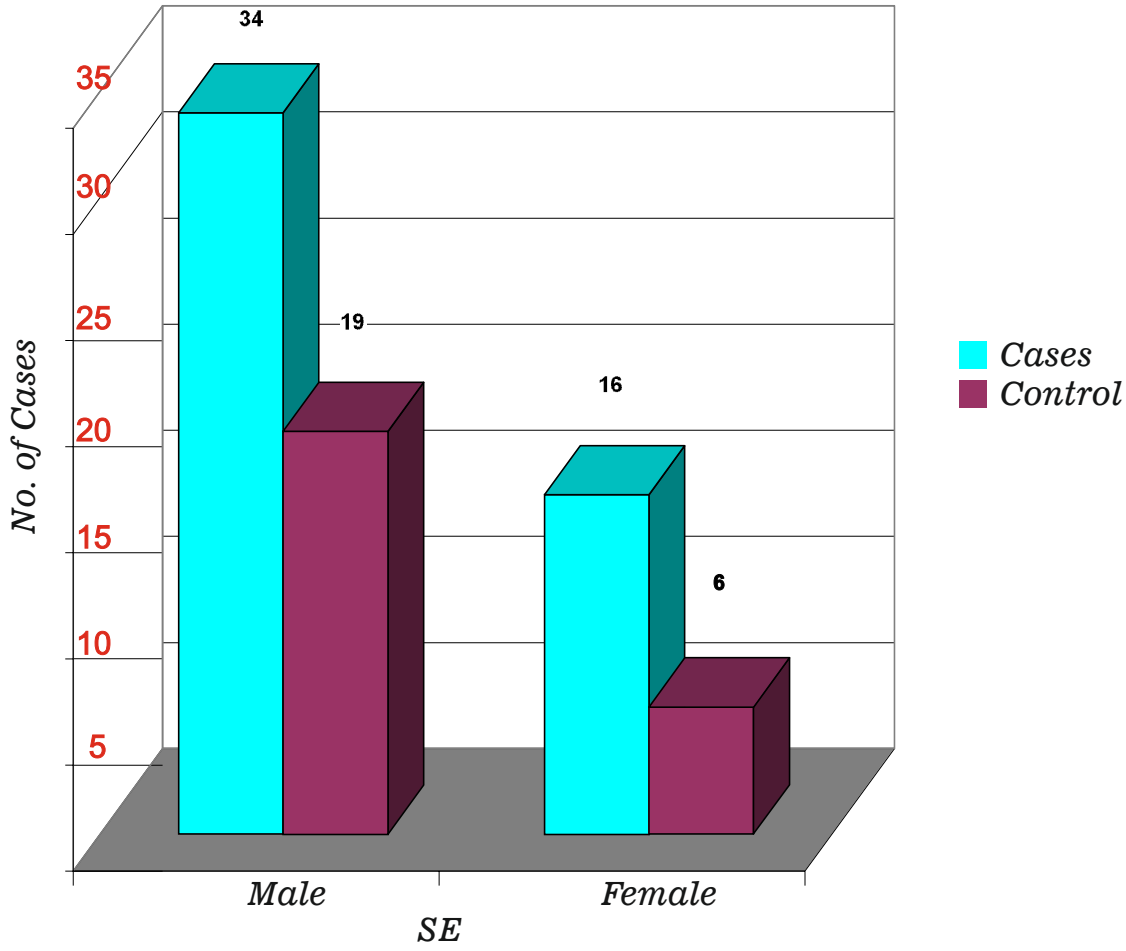


TABLE NO. 2: Showing distribution of cases and control according to age in two groups
Group I – 31-50 (in yrs)
Group II – 51-70 (in yrs)

Age (in yrs)	Case			Control		
	Male	Female	Total	Male	Female	Total
31-50 (group I)	26	12	38	13	6	19
51-70 (group II)	8	4	12	6	0	6
Total	34	16	50	19	6	25

This table show that maximum number of cases were in group I (31-50 yrs) while mini-mum in group II (51-70 yrs) in cases and co-ntrol group both .

GRAPH 2: Showing distribution of cases and control: according to age in two groups

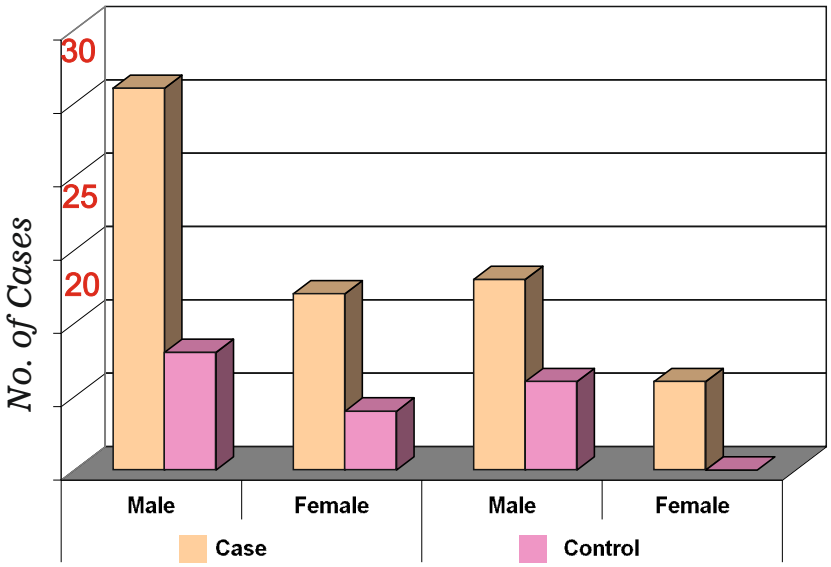


TABLE NO. 3 : Showing statistical analysis of b.p. systolic and diastolic between cases and control

	Systolic Pressure (in mmHg)						Diastolic Pressure (in mmHg)					
	Range (mmHg)	Mean (mmHg)	S.D.	't'	'p'	Result	Range (mmHg)	Mean (mmHg)	S.D.	't'	'p'	Result
Cases	150-248	171.04	19.79	24.81	<0.001	Highly Significant	94-106	99.56	2.2729	47.31	<0.001	Highly Significant
Control	116-118	117.2	0.9797				74-78	76.4	1.265			

This table shows that degree of Blood pressure both systolic and diastolic were hig-hly significantly higher in case group as co-mpared with control group.

TABLE NO. 4:Showing statistical analysis of serum cholesterol in mg% in cases and control

	Case	Control
Number	50	25
Range	144-296	152-262
Mean	208.88	186.8
S D	37.7171	32.009
't'	2.50	-
'p'	<0.05	
Result	Just Significant	

This table shows that level of serum choleste- -rol were just significant in cases as compar- -ed to controls.

Table no. 5 Showing statistical analysis of serum cholesterol binding reserve (s.c.b.r.) in mg% in cases and control

	Case	Control
Number	50	25
Range	10-35	50-80
Mean	24.5	59.719
S D	6.249	6.99
't'	22.11	-
'p'	<0.001	
Result	Highly Significant	

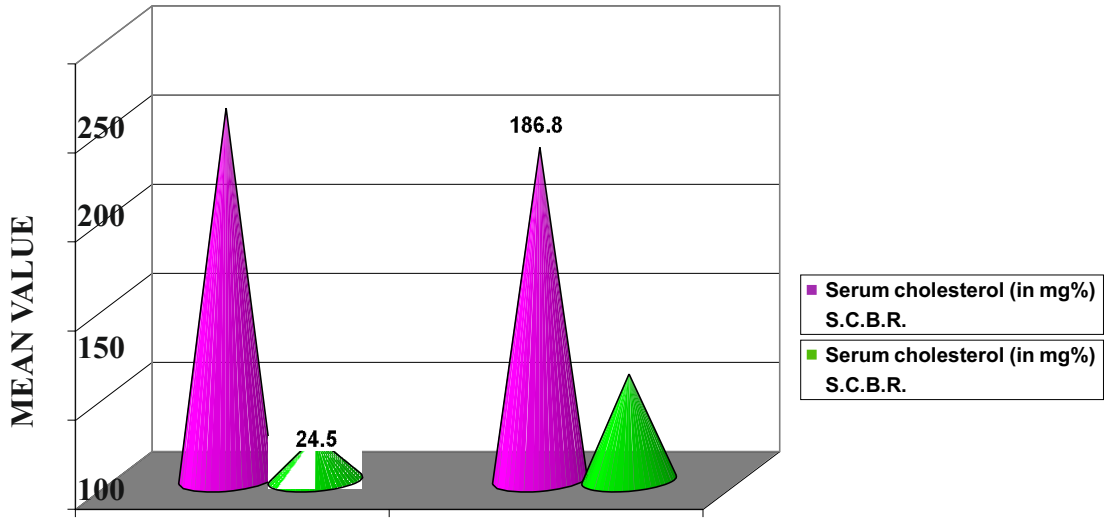
This table shows that level of serum choleste- -rol binding reserve were highly significan- -tly lower in cases than control.

Table 6: Showing Statistical Analysis Between Cholesterol and S.C.B.R. In Cases And Controls

	Serum cholesterol (in mg%)		S.C.B.R. (in mg%)		'r'	'p'	Result
	Mean	SD	Mean	SD			
Case (50 cases)	208.88	37.7171	24.5	6.249	-0.876	<0.001	Highly Significant
Control (25 cases)	186.8	32.009	59.719	6.99	+0.706	<0.001	Highly Significant

This table denote that S.C.B.R. fall with increasing level of serum choles- -terol in cases. While S.C.B.R. rises with increasing level of cholesterol in controls.

Graph 3: Showing statistical analysis between cholesterol and S.C.B.R. in cases and controls



Discussion:

Correlation coefficient between serum cholesterol and S.C.B.R. was +0.706 ($p < 0.001$), denoting that S.C.B.R. rises with increasing level of cholesterol in control.

Hsia et al[6,7]. (1975) revealed a trend of increasing S.C.B.R. value with increasing serum concentration of cholesterol and triglycerides among the controls but this trend was virtually lost among the atherosclerotic patient. This suggest that controls could expand their S.C.B.R. in response to increased serum lipid concentration; while patient could not. This difference in the relationship of S.C.B.R. to lipid may have significance in the etiology of atherosclerosis and may provide a new means to assess the risk of premature myocardial infarction among individual with raised serum lipid level. It seems that a balance of opposing force is operating in atherogensis, the process is accentuated by increased serum lipid level but retarded by expanded S.C.B.R. Petterson et al. (1972) and Lewis (1974) have also shown a significant elevation of serum cholesterol in cases of atherosclerosis as compared to control.

Conclusion:

Increasing level of serum cholesterol in controls, S.C.B.R. showed a corresponding rise with a 'r' value +0.706 ($p > 0.001$). In contrast, hypertensive group showed correlation coefficient (r) with – 0.876 ($p < 0.001$), denoting that S.C.B.R. falls with increasing level of serum cholesterol

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