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# hsCRP in Pre-Hypertension And Hypertension: A Cross Sectional Study

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**Abstract:** Hypertension is turned into a leading cause of non-communicable disease associated mortality and morbidity in both developing as well as developed world. Hypertension is reported to be the fourth contributor to premature death in developed countries and the seventh in developing countries. The purpose of the study was to analyze the correlation between plasma hsCRP and lipid profile in pre-hypertensive as well as hypertensive in Indian patients, attending Diabetic clinic at Index Hospital, Indore and normal control subjects from within campus and surrounding areas. A total of 150 in which 50 newly diagnosed hypertensives (age and sex matched), 50 pre-hypertensives (age and sex matched) and 50 normo-tensive healthy subjects (age and sex matched) were selected for the study. The mean hsCRP value of pre-hypertensive group was higher than the control group and hypertensive group. The mean hsCRP value of pre-hypertensive group was also significantly higher than the hypertensive group. On comparing the parameters of lipid profile among the three study groups, mean cholesterol levels was highest in hypertensive group followed by pre-hypertensive and lowest in control group. These differences however were not significant statistically. Similarly the differences in mean values of LDL were also statistically non significant. Mean HDL level was highest in hypertensive group followed by normal group and lowest in pre-hypertensive group. The difference in mean HDL levels on comparing control group with pre-hypertensive groups and pre-hypertensive group with hypertensive group was highly significant ($p<0.001$). But there was no significant difference in mean HDL values of control and hypertensive groups. Thus HDL was lowest in prehypertensive group. In conclusion, our results suggest that increased serum CRP levels are associated with hypertension, more significantly with prehypertension and in new onset patients with hypertension. Thus serum CRP estimation can be a potential tool for early identification of individuals at the risk for development of hypertension and eventually CVDs.

**Keywords:** Hypertension, Pre-hypertension, hsCRP, Lipid Profile, Cardiovascular morbidity.

## INTRODUCTION

Hypertension is a common, asymptomatic, readily detectable and usually easily treatable disease that leads to lethal complications if left untreated [1]. According to JNC 7 systolic blood pressure is more than 140mmhg and diastolic more than 90 mm hg is considered as stage I hypertension. In this regard the concept of pre-hypertension, defined as a systolic blood pressure of 120–139 mmHg and/or a diastolic blood pressure of 80– 89 mmHg was introduced as the new guideline for the management of blood pressure [2]. The concept of pre-hypertension, defined as a systolic blood pressure of 120–139 mmHg and/or a diastolic blood pressure of 80– 89 mmHg was introduced as the new guideline for the management of blood pressure [2]. There are many established risk factors for development of hypertension which reiterates the importance of an early diagnosis preferably at the stage of pre-hypertension [2].

High-sensitive C-reactive protein (hs-CRP), an acute-phase reactant protein, is a proinflammatory atherogenic circulating marker which may to be an early cardiac risk predictor [3]. According to epidemiology data, hs-CRP can predict coronary artery diseases. The Adult Treatment Panel III Guidelines of the National Cholesterol Education Program suggest that the level of hs-CRP and fibrinogen together with a general biochemical substance check can be used as a risk indicator [4].

Stroke risk has been shown to increase with the severity of prehypertensive status [5] and is intermediate among the associations observed for optimal BP and hypertensive status [5]. Prehypertension and hypertension status cluster with stroke risk factors, such as high-sensitivity C-reactive protein (hsCRP), elevated body mass index, and glucose [6].

C-reactive proteins (CRP) is a plasma protein, present in trace amounts (1mg/L) in healthy subjects whose concentration increases 100 fold in response to injury, infection or inflammation. CRP is named so for its ability to precipitate the somatic C-polysaccharides of Streptococcus pneumoniae and is the first acute phase protein to be described [7, 8]. CRP is primarily synthesized by liver in response to interleukin-6 (IL-6) and interleukin-1 (IL-1). As a risk assessment tool, it has good points like it is stable, has a long half life of 19 hours and shows small variation in values between fresh and frozen forms that makes it an excellent diagnostic marker [9,10].

Hence, we hypothesized that the pre-hypertensive and hypertensive condition is associated with a proinflammatory condition that can be linked to a significant increase in the levels of hsCRP in plasma. This is a simple, cross-sectional observational study of hypertensive patients. In this study patients will be evaluated for serum high sensitive CRP levels which will be correlated with degree of hypertension (HTN), lipid profile and will be compared with normal healthy subjects. The purpose of the study was to analyze the correlation between plasma hsCRP and lipid profile in pre-hypertensive as well as hypertensive in Indian patients, attending Diabetic clinic at Index Hospital, Indore and normal control subjects from within campus and surrounding areas.

## MATERIALS AND METHODS

This cross sectional hospital based study was carried out at the Department of Biochemistry, Index Medical College, Indore, M.P. The duration of study was one year (01.12.2014 to 09.12.2015). A total of 150 in which 50 newly diagnosed hypertensives (age and sex matched), 50 pre-hypertensives (age and sex matched) and 50 normo-tensive healthy subjects (age and sex matched) were selected for the study. Written informed consent was taken from them. Cases were selected from clinically newly diagnosed hypertensive and pre-hypertensive patients attending the medicine outdoor patient department of Index Hospital, Nemawar Road, Indore, M.P and age and sex matched healthy control subjects were also selected from the attendants of hypertensive patients and healthy subjects from the Hospital campus for the study.

### Aims

To find out the correlation of inflammation with the development, degree and cardiovascular complications of hypertension

### Objectives

- Estimate the serum hsCRP, lipid profile in hypertensive, pre-hypertensive & control group
- Find out any significant difference of these parameters between these groups
- Find out the existence of any statistical correlation between hs-CRP and parameters of lipid profile in hypertensive and pre-hypertensive group

### Inclusion criteria

Subjects between the age group of 40 -60 years were selected. Samples from cases were collected before institution of anti-hypertensive treatment. The criterion for diagnosis of hypertension was systolic pressure of ≥140 mm of Hg and diastolic pressure of ≥90 mm of Hg; pre-hypertension was systolic pressure of ≥120mmHg to ≤140mmHg and diastolic pressure of ≥80mmHg to ≤ 90mmHg. The criteria for the selection of controls was age and sex matched healthy normotensive individuals (systolic pressure ≤ 120mmHg and diastolic pressure ≤ 80mmHg ) without any family history of hypertension.

### Exclusion criteria

Hypertensive patients who were already on anti-hypertensive treatment were excluded from the study. Study subjects were examined systematically to exclude any disease (Secondary hypertension) or factors known to cause or those that were associated with hypertension. Subjects with any underlying condition or taking any drugs like steroids, oral contraceptive pills, and thyroxin were also excluded from the study. Similarly, subjects with any underlying condition or taking any drug known to alter serum lipid levels were excluded from the study. Subjects who were smokers, suffering from any inflammatory condition, malignancy, obese and pregnant women, were also excluded from the study.

### Methods for Analysis of Test Parameters

### Blood pressure measurement [11]

In a quiet and comfortably seated study subject, two BP readings were taken five minutes apart, on both arms, with a mercury sphygmomanometer (cuff size, 12.5 x 40 cm). The SBP and DBP were read to the nearest 2 mm Hg. The first and fifth phases of Korotkoff's sounds were taken as the criteria for SBP

and DBP respectively. The average of two consecutive readings was recorded.

**Serum hsCRP [12]**

For the estimation of serum hsCRP, 2 ml of fasting, venous, nonhaemolysed blood sample was withdrawn without the aid of a tourniquet, in a plain sterile bulb. The blood samples were analysed immediately. The estimation of serum hsCRP was done on XL-600 Automatic Analyzer with the kit (Erba Mannheim) based on the measurement of antigen-antibody reaction by the end-point Method.

**Estimation of Serum hs C - reactive protein** [13-15]

This was done by using Latex – immunoturbidimetric high sensitivity method.

Principle Serum C-reactive protein (CRP) causes agglutination of the latex particles coated with anti-human C-reactive protein. The agglutination of the latex particles is proportional to the CRP concentration and can be measured by turbidimetry.

Reference Range Serum hs-CRP in adults – 2.68 to 8.5 mg/L

**Determination of HDL-Cholesterol in Serum or Plasma Method [16]**

Precipitation and enzymatic determination by AutoZyme Cholesterol Reagent Kit: Accurex biomedical Principle: Phosphotungstate /Mg2+precipitates chylomicrons, LDL and VLDL fraction. High Density Lipoprotein (HDL) fraction remains uneffected in supernatant. Cholesterol content of HDL fraction is assayed using AutoZyme Cholesterol.

**Measurement of LDL Cholesterol in Serum or Plasma [17]**

**Method**

Indirect using Friedewald equation LDL Cholesterol was measured by indirect method using the Friedewald equation. The Friedewald equation: In the most widely used indirect method, cholesterol, triglyceride, and HDL cholesterol are measured and LDL cholesterol is calculated from the primary measurements using the empirical equation of Friedewald and colleagues. [LDL chol] = [Total chol] – [HDL chol] – [Triglyceride / 5] where all concentrations are given in milligrams per deciliter. The factor [Triglyceride / 5] is an estimate of VLDL cholesterol concentration, and is based on the average ratio of triglyceride to cholesterol in VLDL.

**Determination of Triglyceride in Serum / Plasma [17]**

Method: Enzymatic.

Kit: Accurex

Normal reference value: Desirable triglyceride ≤150 mg/dl

**RESULTS**

A total of 150 cases in which 50 newly diagnosed hypertensives (age and sex matched) were selected for the study. Subjects between the age group of 40-60 years were selected. The criteria for diagnosis of hypertension were systolic pressure of ≥140 mm of Hg and diastolic pressure of ≥90 mmHg. Pre-hypertension were systolic pressure of 120 to 140 mmHg and diastolic pressure of ≥80 mmHg to ≤90 mmHg. The criteria for the controls were age and sex matched healthy normotensive individuals (systolic pressure ≤120 mmHg and diastolic pressure ≤80 mmHg) without any family history of hypertension.

**Table-1: Distribution of study groups with different grades of hypertension and Controls as per the categorization of serum CRP levels**

| | Pre-hypertensive | | Hypertensive | | Control | |
|---|---|---|---|---|---|---|
| | No. | % | No. | % | No. | % |
| <1 mg/litre | 20 | 40% | 38 | 76% | 47 | 94% |
| 1-3 mg/litre | 21 | 42% | 11 | 22% | 3 | 6% |
| >3 mg/litre | 9 | 18% | 01 | 2% | 0 | 0 |
| Total | 50 | 100% | 50 | 100% | 50 | 100% |

Table 1 show 18% of pre-hypertensive was having > 3mg/litre serum CRP levels. In hypertensive groups majority of study participants 76% were having <1mg/litre serum CRP levels. Within the patient group (n=50), the male hypertensive subjects does not show any significant difference in hs-CRP levels as compared to female hypertensive subjects (P>0.1).

When compared with the hsCRP levels in control subjects, the serum hs-CRP levels vary significantly, with most significant difference found in patients with prehypertension (P<0.001) followed by hypertensives.

**Table-2: Distribution of study groups with different grades of hypertension and controls as per the categorization of serum total cholesterol levels**

| | Pre-hypertensive | | Hypertensive | | Control | |
|---|---|---|---|---|---|---|
| | No. | % | No. | % | No. | % |
| < 200 mg/dl | 25 | 50% | 24 | 48% | 36 | 72% |
| 200-239 mg/dl | 17 | 34% | 19 | 38% | 8 | 16% |
| ≥ 240 mg/dl | 8 | 16% | 07 | 14% | 6 | 12% |
| Total | 50 | 100% | 50 | 100% | 50 | 100% |

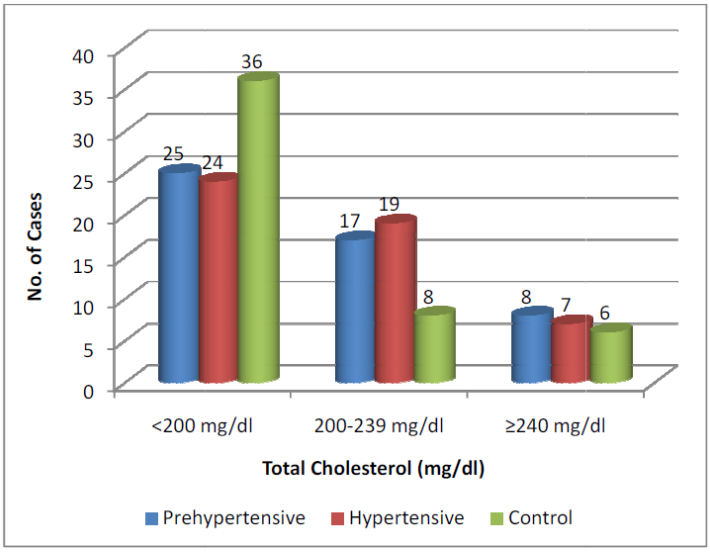


**Fig-1: Distribution of study groups with different grades of hypertension and controls as per the categorization of serum total cholesterol levels**

**Table-3: Distribution of study groups with different grades of hypertension and controls as per the categorization of serum LDL levels**

| | Pre-hypertensive | | Hypertensive | | Control | |
|---|---|---|---|---|---|---|
| | No. | % | No. | % | No. | % |
| < 100 mg/dl | 17 | 34% | 14 | 28% | 23 | 46% |
| 100-129 mg/dl | 7 | 14% | 18 | 36% | 13 | 26% |
| 130-159 mg/dl | 9 | 18% | 9 | 18% | 6 | 12% |
| 160-189 mg/dl | 11 | 22% | 6 | 12% | 4 | 8% |
| >189 mg/dl | 2 | 4% | 1 | 2% | 4 | 8% |
| Total | 50 | 100% | 50 | 100% | 50 | 100% |

**Table-4: Distribution of study groups with different grades of hypertension and controls as per the categorization of serum HDL levels**

| | Pre-hypertensive | | Hypertensive | | Control | |
|---|---|---|---|---|---|---|
| | No. | % | No. | % | No. | % |
| < 40 mg/dl | 36 | 72% | 23 | 46% | 26 | 52% |
| 40-59 mg/dl | 14 | 28% | 27 | 54% | 24 | 48% |
| Total | 50 | 100% | 50 | 100% | 50 | 100% |

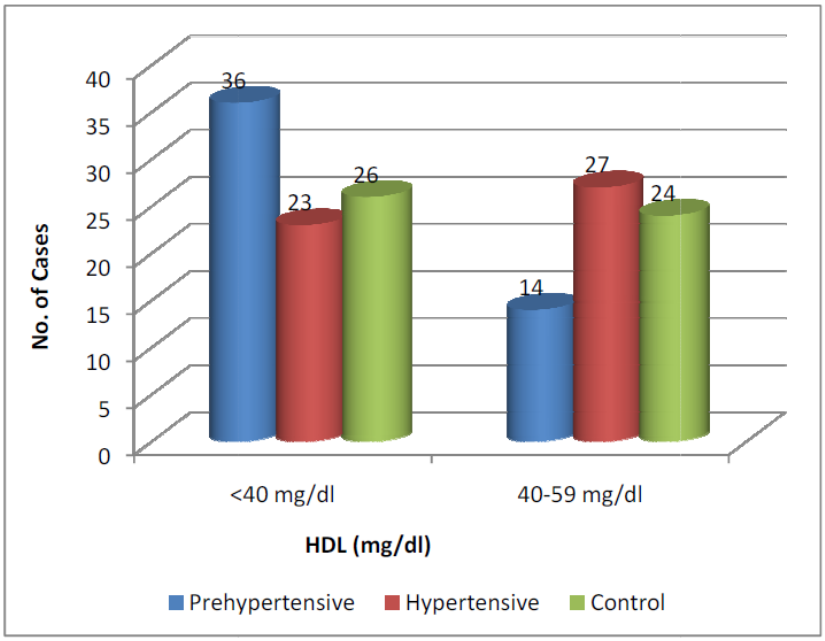


**Fig-2: Distribution of study groups with different grades of hypertension and controls as per the categorization of serum HDL levels**

**Table-5: Distribution of study groups with different grades of hypertension and controls as per the categorization of serum triglycerides levels**

| | Pre-hypertensive | | Hypertensive | | Control | |
|---|---|---|---|---|---|---|
| | No. | % | No. | % | No. | % |
| < 150 mg/dl | 25 | 50% | 21 | 42% | 34 | 68% |
| 150-199 mg/dl | 20 | 40% | 15 | 30% | 15 | 30% |
| 200-499 mg/dl | 5 | 10% | 14 | 28% | 1 | 2% |
| Total | 50 | 100% | 50 | 100% | 50 | 100% |

**Table-6: Comparison of mean values of various parameters in study groups with different grades of hypertension and control group**

| | Control | | Prehypertensive | | Hypertensive | | ANOVA |
|---|---|---|---|---|---|---|---|
| | Mean | ±SD | Mean | ±SD | Mean | ±SD | p value |
| Age | 37.98 | 6.717 | 40.46 | 5.939 | 40.86 | 7.039 | .063 |
| SBP | 110.60 | 4.92 | 130.44 | 5.13 | 148.28 | 7.84 | 000 |
| DBP | 73.20 | 3.23 | 85.08 | 2.22 | 92.32 | 3.88 | 000. |
| CRP | 0.36 | 0.31 | 1.77 | 1.35 | 0.82 | 0.72 | .000 |
| CHL | 180.04 | 43.60 | 197.86 | 51.51 | 198.72 | 35.39 | .060 |
| LDL | 112.75 | 46.24 | 130.58 | 48.88 | 119.51 | 36.63 | .130 |
| HDL | 40.08 | 3.22 | 36.86 | 6.16 | 41.08 | 6.02 | .000 |
| TG | 136.06 | 30.42 | 152.10 | 41.91 | 190.66 | 85.66 | .000 |
| Non HDL cholesterol | 139.96 | 43.99 | 161.00 | 48.13 | 157.64 | 35.68 | .034 |

**Table-7: Correlation between BP and CRP in Normal healthy subjects**

| Characteristics | r value | P value |
|---|---|---|
| SBP and CRP | 0.004 | 0.979 |
| DBP and CRP | 0.172 | 0.231 |

**Table-8: Correlation between BP and Lipid Profile in Normal healthy subjects**

| | r value | p value |
|---|---|---|
| SBP and CHL | .169 | .241 |
| DBP and CHL | -.037 | .798 |
| SBP and LDL | .182 | .206 |
| DBP and LDL | -.017 | .909 |
| SBP and HDL | -.093 | .519 |
| DBP and HDL | .038 | .795 |
| SBP and TG | -.123 | .396 |
| DBP and TG | -.160 | .268 |
| SBP and Non HDLCHL | .174 | .226 |
| DBP and NON HDL CHL | -.040 | .785 |

**Table-9: Correlation between CRP and Lipid Profile in Normal healthy subjects**

| Characteristics | r value | P value |
|---|---|---|
| CRP and CHL | 0.162 | 0.262 |
| CRP and LDL | 0.159 | 0.271 |
| CRP and HDL | 0.189 | 0.188 |
| CRP and TG | -0.149 | 0.303 |
| CRP and Non HDL CHL | 0.146 | 0.311 |

**Table-10: Correlation between BP and CRP in pre-hypertensive subjects**

| Characteristics | r value | P value |
|---|---|---|
| SBP and CRP | -0.42 | 0.770 |
| DBP and CRP | 0.240 | 0.093 |

**Table-11: Correlation between BP and lipid profile in pre-hypertensive subjects**

| | r value | p value |
|---|---|---|
| SBP and CHL | .240 | .093 |
| DBP and CHL | .114 | .432 |
| SBP and LDL | .254 | .076 |
| DBP and LDL | .162 | .260 |
| SBP and HDL | .099 | .495 |
| DBP and HDL | -.051 | .723 |
| SBP and TG | -.076 | .600 |
| DBP and TG | -.210 | .143 |
| SBP and Non HDLCHL | .244 | .087 |
| DBP and NON HDL CHL | .128 | .375 |

**Table-12: Correlation between CRP and lipid profile in pre-hypertensive subjects**

| | r value | p value |
|---|---|---|
| CRP and CHL | .017 | .909 |
| CRP and LDL | -.030 | .836 |
| CRP and HDL | -.068 | .639 |
| CRP and TG | .327 | 0.20 |
| CRP and Non HDLCHL | .027 | .855 |

## DISCUSSION

There are several potential mechanisms that may account for the observed relationship between blood pressure and CRP levels. Increased blood pressure may promote vascular inflammation by modulation of mechanical stimuli from pulsatile blood flow. Cyclic strain has been shown to increase the expression of soluble intercellular adhesion molecule1 (sICAM-1) and vascular cell adhesion molecule-1(VCAM-1) by endothelial cells [18] and also upregulate the secretion of monocyte chemoattractant protein-1 (MCP-1) [19,20], that promote monocyte adhesion to endothelium. Furthermore, elevated blood pressure is also known to promote generation of reactive oxygen species (ROS) [21] as evident from a study where a significant correlation was observed between levels of CRP and mononuclear oxidative stress [22].

The association of inflammatory markers with pre-hypertension and hypertension is not very clear. Few studies, however, have explored interrelations between levels of CRP and hypertensive risk factors and data from these reports have been inconsistent. Hence, we hypothesized that the pre-hypertensive condition is associated with a pro-inflammatory condition that can be linked to a significant increase in the levels of hsCRP in plasma. Elevated CRP concentrations may exacerbate the underlying proatherothrombotic environment of hypertension. CRP has been independently associated with increases in BP and incident hypertension, although these associations may be confined to older populations [23-26].

Hypertension is an important cause of cardiovascular and kidney disease [1,2]. Due to its high mortality and morbidity early diagnosis and effective prevention is important. It is an established risk factor for development of atherosclerosis and various cardiovascular diseases (CVDs) like coronary heart disease (CHD), renal failure, congestive heart failure (CHF), ischemic and haemorrhagic stroke and peripheral vascular disease [27,28].

In this regard the concept of pre-hypertension defined as a systolic blood pressure of 120-139 mmHg and/or a diastolic blood pressure of 8—89 mmHg was introduced as the new guideline for the management of blood pressure [3]. There are many established risk factors for development of hypertension which reiterates the importance of an early diagnosis preferably at the stage of pre-hypertension.

Many studies demonstrated that the pre-hypertensive had higher levels of blood glucose, insulin resistance, total cholesterol, low density lipoprotein cholesterol and triglycerides, higher body mass index, abnormalities of glucose metabolism and lower levels of high-density lipoprotein cholesterol than the normotensive group [3, 7, 29].

The association of inflammatory markers with pre-hypertension and hypertension is not very clear. Few studies however, have explored interrelations between levels of CRP and hypertensive risk factors and data from these reports have been inconsistent [10-12]. The mean hsCRP value of pre-hypertensive group was higher than the control group and hypertensive group. The mean hsCRP value of pre-hypertensive group was also significantly higher than the hypertensive group.

Inflammation, common in hypertensives, decreases endothelium dependent relaxation, possibly by decreased capacity of the endothelium to generate vasodilatory factors, particularly nitric oxide (NO) which inturn raises blood pressure. This is substantiated by several studies which have shown inflammatory markers such as CRP as an independent determinant of endothelium dependent vascular function among patient with coronary heart disease (CHD) and this situation may also exist in patients with hypertension [30]. Our findings are in agreement to that one reported by Sesso *et al*., who also have shown a link between elevated CRP and increased risk of developing hypertension in a cohort study, including people with baseline blood pressure in prehypertensive range [26].

CRP inhibits formation of nitric oxide by endothelial cells which in turn promote vasoconstriction, leukocyte adhesion, platelet activation, oxidation and thrombosis. Moreover, high levels of CRP may upregulate angiotensin receptors and enhance expression of plasminogen activator inhibitor-1 by endothelial cells. Both these changes could raise blood pressure and promote atherogenesis [11, 29].

On comparing the parameters of lipid profile among the three study groups, mean cholesterol levels was highest in hypertensive group followed by pre-hypertensive and lowest in control group. These differences however were not significant statistically. Similarly the differences in mean values of LDL were also statistically non significant. Mean HDL level was highest in hypertensive group followed by normal group and lowest in pre-hypertensive group. The difference in mean HDL levels on comparing control group with pre-hypertensive groups and pre-hypertensive group with hypertensive group was highly significant ($p<0.001$). But there was no significant difference in mean HDL values of control and hypertensive groups. Thus HDL was lowest in prehypertensive group.

The difference in mean TG levels on comparing control group with hypertensive groups and pre-hypertensive group with hypertensive group was highly significant ($p<0.001$). But there was no significant difference in mean TG values of control and pre-hypertensive groups. Thus TG was highest in hypertensive group.

The difference in mean non HDL cholesterol levels on comparing control group with pre-hypertensive groups was statistically significant ($p<0.046$). But there was no significant difference in mean non HDL cholesterol values of control and hypertensive groups and pre-hypertensive and hypertensive groups. Thus non HDL cholesterol was higher in pre-hypertensive and hypertensive group.

No correlation was established between systolic or diastolic blood pressure values and hsCRP or lipid profile parameters in normal healthy subjects. No association was found values of hsCRP and lipid profile parameters in normal healthy subjects.

On applying Pearson correlation no significant p value was seen on comparing SBP and DBP with hsCRP and lipid profile parameters in prehypertensive subjects. On comparing hsCRP with individual lipid profile parameters in pre-hypertensive subjects, significant correlation was found only between hsCRP and TG levels ($P=0.020$).

Similar comparison of hsCRP with lipid profile parameters in hypertensive subjects showed positive correlation with total cholesterol and LDL, while no significant correlation was established between hsCRP and HDL, TG or non HDL cholesterol.

Pre-hypertension and hypertension both are having an inflammatory pathology. Pre-hypertension not only developed in hypertension but also increases the chances of cardiovascular diseases. However, because of the cross-sectional nature of our study these findings should be confirmed in prospective cohort studies, aimed at elucidating the role of CRP in the prediction, diagnosis and management of hypertension.

## CONCLUSION

Among the lipid profile parameters cholesterol and LDL values show no significant difference among normal healthy subjects, pre-hypertensive and hypertensive subjects. HDL levels were significantly lower in pre-hypertensive subjects as compared to normal healthy subjects. The triglycerides were deranged with higher values in hypertensive subjects as compared to the normal and pre-hypertensive subjects. Non HDL cholesterol levels were significantly higher in pre-hypertensive and hypertensive subjects as compared to normal healthy subjects.

hsCRP values are highest among the pre-hypertensive subjects as compared to normal and hypertensive subjects. Thus it signifies its role as marker of chronic inflammatory process involved in evolution of hypertension.

Hypertensive subjects have significantly higher hsCRP level as compared to normal healthy subjects. Thus high hsCRP levels support the hypothesis of chronic inflammation underlying the pathogenesis of hypertension. hsCRP levels showed positive correlation with TG levels in pre-hypertensive patients. hsCRP levels showed positive correlation with total cholesterol and LDL levels in hypertensive subjects.

Thus dyslipidemia raised hsCRP levels are seen pre-hypertensive and hypertensive subjects. These parameters may be used to catch early stages of pre-hypertensive cases developing into hypertensive over a span of time. It can guide for timely intervention to avoid and delay long term effects and morbidity associated with hypertension.

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