

## Correlation of Microalbuminuria and C - reactive protein in Normotensive and Hypertensive type 2 Diabetic Patients

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### ABSTRACT

**Background:** The present study was aimed to correlation of microalbuminuria and C-reactive protein in normotensive and hypertensive type 2 diabetic patients.

**Introduction:** In diabetes and hypertension blood pressure is one of the main determinant of microalbuminuria. Because CRP and microalbuminuria considered to be closely related with same disease processes. So, an attempt has been made to find out the association of CRP with microalbuminuria and whether this association varies in diabetic population with or without hypertension.

**Material & Methods:** The present study includes 500 type 2 diabetic subjects & further divided into two groups based on hypertension group A (Normotensive) and group B (hypertensive). Biochemical parameters analyzed in the department of biochemistry of Dr. Ulhas Patil Medical College & Hospital, Jalgaon. Hypertension was considered according to definition of WHO; as systolic blood pressure (SBP)  $\geq 140$  mmHg and diastolic (DBP)  $\geq 90$  mmHg.

**Statistical Analysis:** Statistical analysis was performed using SPSS version 20. All the data were presented as mean  $\pm$ SD for continuous variables and percentage for categorical variables. A comparison was done between variables using independent students 't'- test.

**Results:** A statistically significant positive correlation was found between C-reactive protein and UACR in hypertensive diabetic group (group B) but was not found in normotensive diabetic group (group A). The correlation was stronger in group B ( $r=0.48$ ;  $p<0.001$ ) as compared to group A ( $r=0.00$ ).

**Conclusion:** In our study high levels of C-reactive protein are found in hypertensive diabetic subjects as compared to non hypertensive diabetic subjects.

**Keywords:** Microalbuminuria, Normotensive, Hypertension, Diabetes Mellitus, IDF

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**Conflict of interest:** None

### INTRODUCTION

Many chronic diseases are now in pandemic proportions and increasingly a major cause of morbidity and mortality worldwide. Diabetes mellitus, especially type 2 diabetes, plays a starring role in this problem,<sup>1,2</sup> with its complications, being a very important public health issue.

There are more than 366 million diabetic subjects in the world, and these numbers are projected to increase to nearly 552 million by 2030.<sup>3</sup> The International Diabetes Federation (IDF) also reported that the total number of diabetic subjects in India was 61.3 million in 2011 and that this would rise to 101.2 million by the year 2030.<sup>4</sup>

Microalbuminuria may be a marker of generalized vascular disease, with arterial endothelial dysfunction being involved in the pathogenesis of atherothrombotic vascular disease. The exact pathophysiology regarding how microalbuminuria contributes to or accelerates the atherosclerotic process is uncertain. The current

understanding, however, suggests that mechanisms of vascular injury associated with microalbuminuria are different between those with and those without diabetes who also have hypertension.<sup>5,6,7</sup>

In individuals with microalbuminuria who do not have diabetes, both endothelial dysfunction and alterations in the extracellular matrix contribute to the increase in vascular permeability and ultimately promote the atherosclerotic process.<sup>8,9,10</sup>

Defective endothelial permeability permits lipid influx into the vessel wall causing atherosclerotic changes. Microalbuminuria may not be a direct pathogen in the genesis of nondiabetic hypertensive vascular or renal disease. This lack of pathogenicity may relate to the observation that the albumin moiety is not glycosylated.

CRP levels are found to be related to insulin resistance, obesity and endothelial dysfunction in a cross-sectional study by Yudkin et al.<sup>11</sup> Data derived from the Womens

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Health Study showed that elevated levels of CRP predict the development of T2DM.<sup>12</sup> This was further confirmed by data from the Nurses Health Study.<sup>13</sup> The antidiabetic thiazolidinedione drug rosiglitazone has been shown to lower CRP levels in patients with type 2 diabetes.<sup>14</sup> Pioglitazone also lowers CRP,<sup>15</sup> making this most likely a class effect. The thiazolidinediones are PPAR (peroxisome proliferator-activated receptor) gamma agonists; however the PPAR alpha agonist fenofibrate has also been shown to lower CRP levels.<sup>16</sup> CRP levels have also been associated with future development of hypertension,<sup>17</sup> and are known to play a direct role in atherosclerosis and thrombosis.<sup>18</sup>

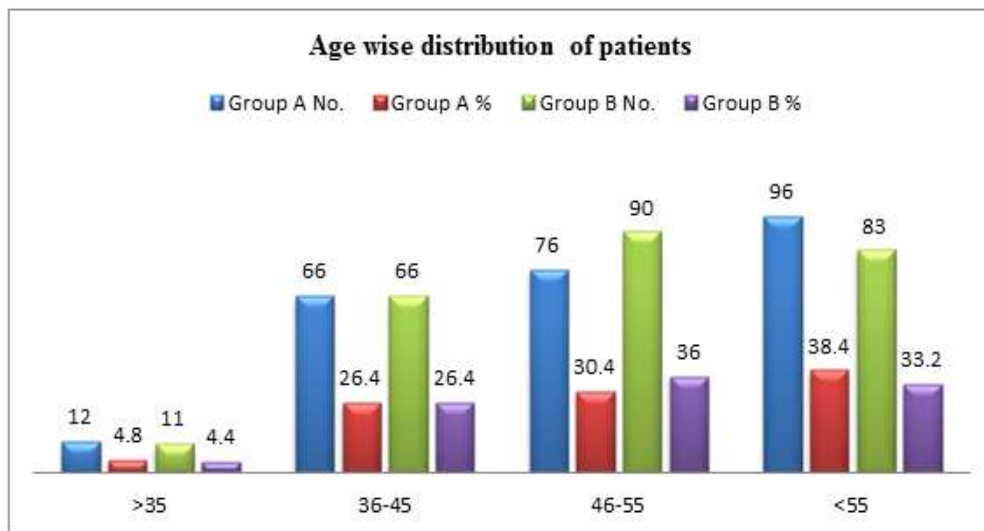
In diabetes and hypertension blood pressure is one of the main determinant of microalbuminuria. Because CRP and microalbuminuria considered to be closely related with same disease processes, there may be the possibility of some strong relationship. And this relationship may vary in condition of some other cardiovascular risk factors eg. in diabetes and hypertension. So, an attempt has been made to find out the association of CRP with microalbuminuria and whether this association varies in diabetic population with or without hypertension.

**Material & Methods:** The present study includes 500 type 2 diabetic subjects & further divided into two groups based on hypertension group A (Normotensive) and group B (hypertensive). Biochemical parameters analyzed in the department of biochemistry of Dr. Ulhas Patil Medical College & Hospital, Jalgaon. Hypertension was considered according to definition of WHO; as systolic blood pressure (SBP)  $\geq 140$  mmHg and diastolic (DBP)  $\geq 90$  mmHg.

**Statistical Analysis:** Statistical analysis was performed using SPSS version 20. All the data were presented as mean  $\pm$ SD for continuous variables and percentage for

categorical variables. A comparison was done between variables using independent students 't'- test. Correlation was performed between variables using Pearson's correlation coefficient analysis. A p-value  $< 0.05$  was considered as statistically significant.

**Results:** In this study the minimum age was 26 years old and maximum age was 70 years old. Out of total 500 patients, 58% were males while 42% were females. In normotensive diabetic group (Group A) maximum patients were in age group of  $>55$  years (38%) whereas in hypertensive diabetic group (Group B), maximum numbers of patients (36%) were in the age group of 46-55 years. In both the groups A and B, Systolic blood pressure was  $125 \pm 5.49$  Vs  $158.5 \pm 7.05$  mm/Hg and Diastolic blood pressure was  $82.9 \pm 4.59$  Vs  $90.1 \pm 6.055$  mm/Hg respectively. Comparison was statistically significantly different between both the groups for SBP and DBP ( $p < 0.001$ ). Comparison on the basis of Fasting blood sugar in between two groups was statistically highly significant  $188 \pm 34.4$  Vs  $231 \pm 44.31$  mg/dl ( $p < 0.001$ ). However the comparison between both the groups was non significant with respect to Post prandial blood sugar  $291.8 \pm 40.70$  Vs  $297 \pm 31.84$  mg/dl ( $p > 0.076$ ). The comparison on the basis of UACR in between two groups was statistically highly significant  $177.23 \pm 34.93$  vs.  $193.21 \pm 48.18$  mg/g ( $p < 0.001$ ). The comparison on the basis of CRP in between two groups was statistically highly significant  $5.75 \pm 2.34$  vs.  $8.45 \pm 2.57$  mg/l ( $p < 0.0001$ ). A statistically significant positive correlation was found between C-reactive protein and UACR in hypertensive diabetic group (group B) but was not found in normotensive diabetic group (group A). The correlation was stronger in group B ( $r = 0.48$ ;  $p < 0.001$ ) as compared to group A ( $r = 0.00$ ).



**Figure-1:** Frequency distribution of patients according to age

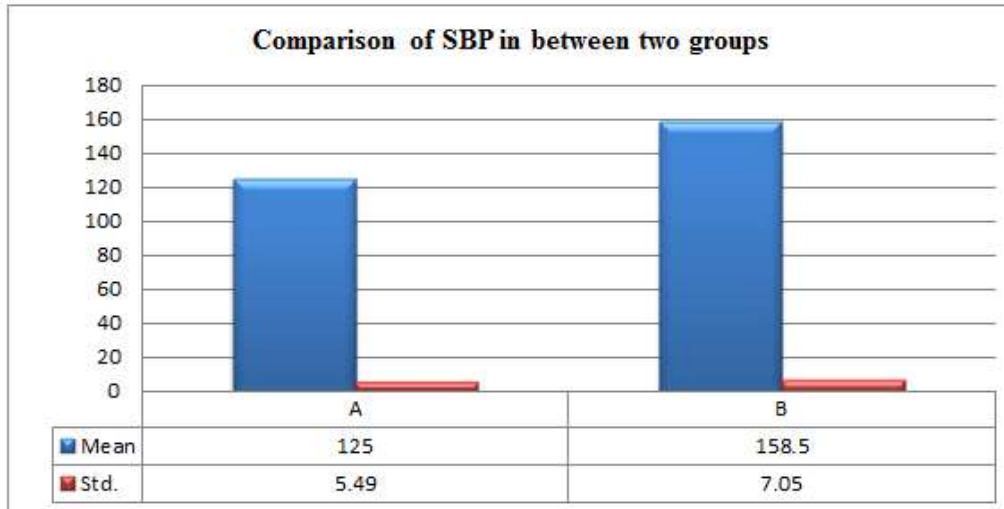


Figure- 2: Comparison of SBP in between two groups

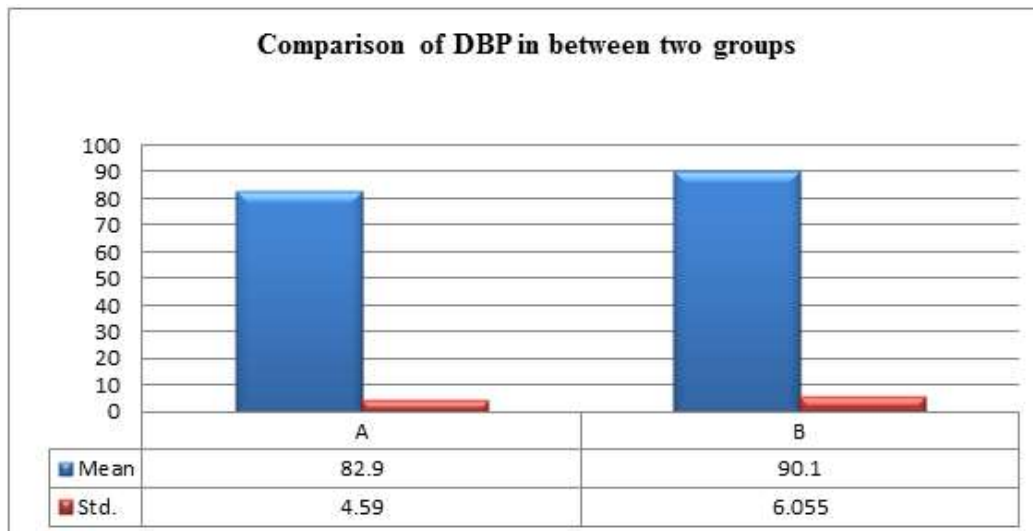


Figure- 3: Comparison of DBP in between two groups

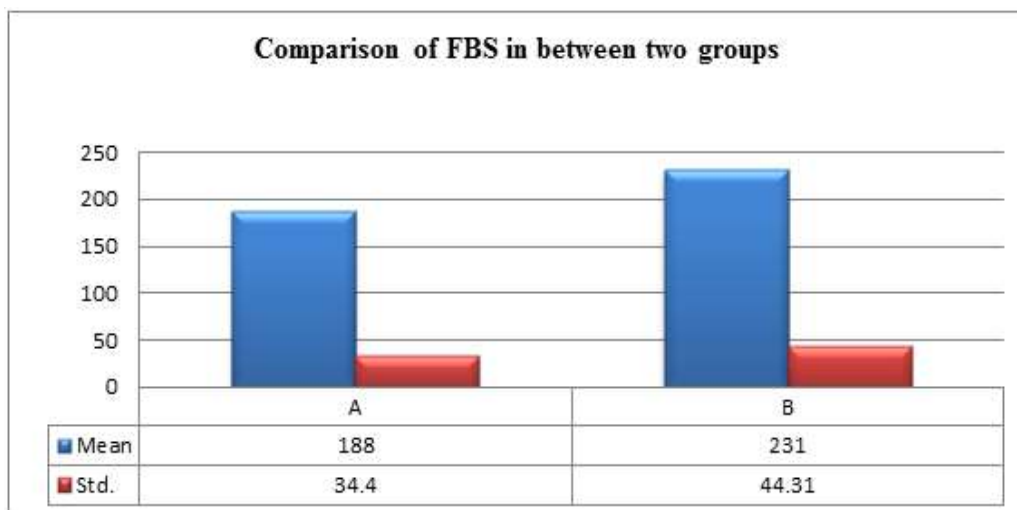
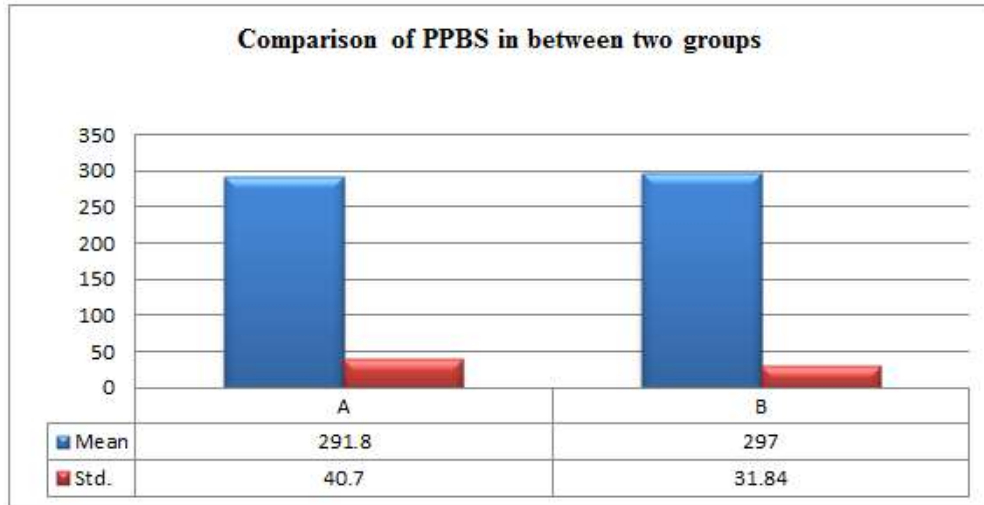
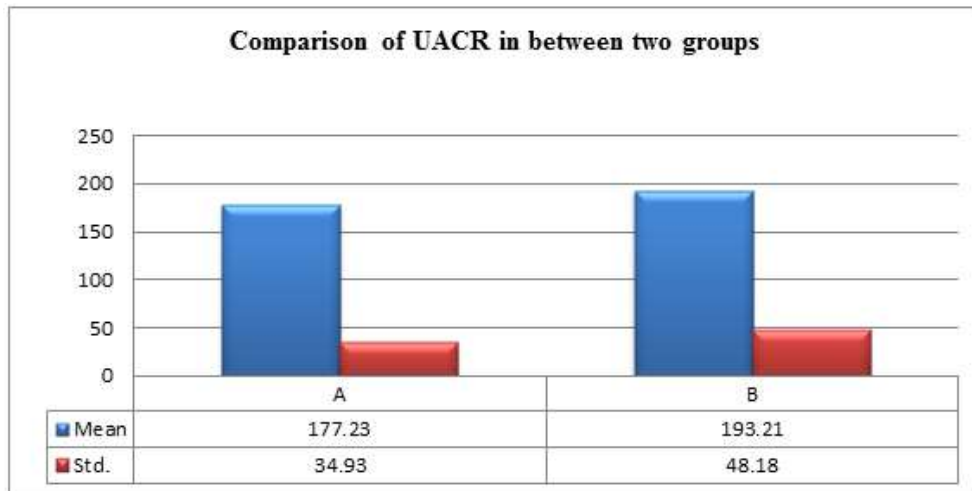


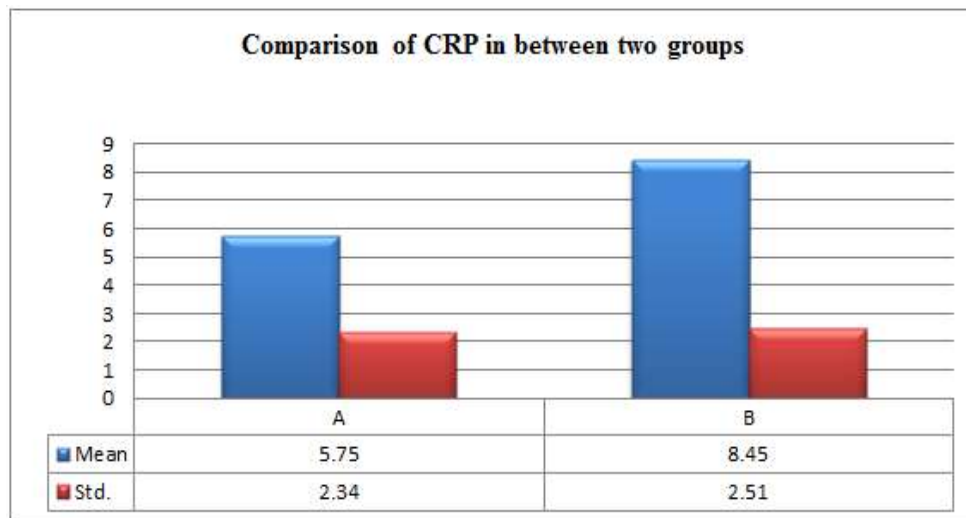
Figure-4: Comparison of FBS in between two groups



**Figure-5:** Comparison of PPBS in between two groups



**Figure-6:-**Comparison of UACR in between two groups



**Figure-7:** Comparison of CRP in between two groups

**DISCUSSION**

500 clinically diagnosed Type 2 diabetes mellitus patients with microalbuminuria attending outpatient department of medicine, Dr. Ulhas Patil Medical College & Hospital, Jalgaon were considered for the study. Further on the basis of blood pressure, they were divided into two groups. 250 normotensive patients were considered as Group A and 250 hypertensive patients were considered as Group B. C-reactive protein and microalbuminuria was studied in all of them to find the correlation between C-reactive protein and microalbuminuria.

The aim of present study was to determine whether serum C-reactive protein levels were associated with microalbuminuria in diabetic population with and without hypertension.

Microalbuminuria is a first clinical sign for the involvement of kidneys in patients with type 2 diabetes mellitus. It is also a known predictor of poor renal outcomes in diabetes mellitus and in essential hypertension.

Similar observations have also been made in other studies. Fotheringham J et al concluded that at lower eGFR, a given increase in systolic blood pressure is accompanied by a substantially greater relative increase in renal albumin permeability, consistent with a remnant nephron effect increasing transmission of systemic hydrostatic pressure to the glomerulus. An alternative explanation underlying the interactions of diabetes mellitus and excretory impairment with systolic blood pressure is that arterial stiffness accompanying renal disease and diabetes mellitus results in a higher central arterial pressure at any given brachial systolic blood pressure.

Stuveling et al had also shown that the association between microalbuminuria and C-reactive protein was more significant in subject with high mean arterial pressure. They concluded that BP positively modified the relationship between microalbuminuria and C-reactive protein.

Tsioufis et al had shown that microalbuminuria is accompanied by increase in C-reactive protein level in the view of hypertension, with atherosclerotic changes. So, the findings in the present study are consistent with the above mentioned studies.

In our study high levels of C-reactive protein are found in hypertensive diabetic subjects as compared to non hypertensive diabetic subjects. Elevated C-reactive protein levels indicate low grade inflammation. Inflammation seems to be related with increased albumin secretion from glomerulus in response to blood pressure. Our study shows the novel finding about the relationship between elevated C-reactive protein levels and microalbuminuria in hypertensive diabetics but not in normotensive diabetics.

**REFERENCES**

1. Beaglehole R, Yach D. Globalisation and the prevention and control of non-communicable disease:

the neglected chronic diseases of adults. *The Lancet*. 2003 Sep; 362(9387):903-908.

2. Yach D, Hawkes C, Gould CL, Hofman KJ. The global burden of chronic diseases: overcoming impediments to prevention and control. *JAMA*. 2004 Jun 2; 291(21):2616-2622.

3. Whiting DR, Guariguata L, Weil C, Shaw J. IDF Diabetes Atlas: Global estimates of the prevalence of diabetes for 2011 and 2030. *Diabetes Research and Clinical Practice*. 2011 Dec; 94(3):311-321.

4. Anjana RM, Pradeepa R, Deepa M, Datta M, Sudha V, Unnikrishnan R, et al. Prevalence of diabetes and prediabetes (impaired fasting glucose and/or impaired glucose tolerance) in urban and rural India: Phase I results of the Indian Council of Medical Research-India Diabetes (ICMR-INDIAB) study. *Diabetologia*. 2011 Dec 1;54(12):3022-3027.

5. Schmitz A. Microalbuminuria, blood pressure, metabolic control, and renal involvement: Longitudinal studies in white non-insulin-dependent diabetic patients. *Am J Hypertens*.1997;10:189S-197S.

6. Gosling P. Microalbuminuria: a marker of systemic disease. *BrJ Hosp Med*.1995;54:285-290.

7. Jensen JS. Renal and systemic transvascular albumin leakage in severe atherosclerosis. *ArteriosclerThrombVasc Biol*.1995;15:1324-1329.

8. Schmitz A. Microalbuminuria, blood pressure, metabolic control, and renal involvement: Longitudinal studies in white non-insulin-dependent diabetic patients. *Am J Hypertens*.1997;10:189S-197S.

9. Deckert T, Kofoed-Enevoldsen A, Norgaard K, Borch-Johnsen K, Feldt-Rasmussen B, Jensen T. Microalbuminuria. Implications for micro and macrovascular disease. *Diabetes Care*.1992;15:1181-1191.

10. Mogyorosi A, Ziyadeh FN. Update on pathogenesis, markers and management of diabetic nephropathy. *CurOpie Nephrol Hypertens*.1996;5:243-253.

11. John S. Yudkin; C.D.A. Stehouwer;J.J. Emeis; S.W. Coppack C-Reactive Protein in Healthy Subjects: Associations with Obesity, Insulin Resistance, and Endothelial Dysfunction. *ATVG*. 1999; 19: 972-978.

12. Aruna D P, JoAnn E; Nader R, Julie E B, Paul M R. C-Reactive Protein, Interleukin 6, and Risk of Developing T2DM. *JAMA*.2001; 286: 327-334.

13. Frank B. Hu, James B. Meigs, Tricia Y. Li 1, Nader Rifai, and JoAnn E. Manson Inflammatory Markers and Risk of Developing T2DM in Women Diabetes. 2004; 53: 693-700.

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14. Haffner SM, Greenberg AS, Weston WM, Chen H, Williams K, Freed MI. Effect of Rosiglitazone Treatment on Nontraditional Markers of Cardiovascular Disease in Patients with Type 2 Diabetes Mellitus. *Circulation*. 2002 Aug 6; 106(6):679-684.
15. Satoh N, Ogawa Y, Usui T, Tagami T, Kono S, Uesugi H, et al. Antiatherogenic effect of pioglitazone in type 2 diabetic patients irrespective of the responsiveness to its antidiabetic effect. *Diabetes Care*. 2003 Sep; 26(9):2493-9.
16. Staels B, Koenig W, Habib A, Merval R, Lebret M, Torra IP, et al. Activation of human aortic smooth-muscle cells is inhibited by PPAR alpha but not by PPAR gamma activators. *Nature*. 1998 Jun 25; 393(6687):790-3.
17. Sesso HD, Buring JE, Rifai N, Blake GJ, Gaziano JM, Ridker PM. C-reactive protein and the risk of development hypertension. *JAMA*. 2003 Dec10; 290(22):2945-51.
18. Ridker PM. Rosuvastatin in the Primary Prevention of Cardiovascular Disease Among Patients with Low Levels of Low-Density Lipoprotein Cholesterol and Elevated High -Sensitivity C-Reactive Protein Rationale and Design of the JUPITER Trial. *Circulation*. 2003 Nov 11; 108(19):2292-7.