

# STUDY OF DYSLIPIDEMIA AND CARDIO VASCULAR RISK IN PATIENTS WITH RHEUMATOID ARTHRITIS PATIENTS IN TELANGANA POPULATION

Veeranna Addanki<sup>1</sup>, C. Yadavendra Reddy<sup>2</sup>

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Corresponding Author:

**Dr. C. Yadavendra Reddy,**

Email: chamareddy\_07@yahoo.com

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<sup>1</sup>Assistant Professor, Department of General Medicine, ESIC Medical College, Hyderabad, Telangana, India.

<sup>2</sup>Associate Professor, Department of General Medicine, MRIMS Suraram, Hyderabad, Telangana, India

## Abstract

**Background:** Rheumatoid Arthritis (RA) is a chronic inflammatory disease associated with dyslipidemia hence it enhances atherosclerosis and causes morbidity and mortality in cardio vascular patients. **Materials and Methods:** 150 RA, patients compared with 100 controlled group. The bio chemical parameters included lipid profile, Magnesium calcium 'ALT', AST. Total protein, Uric acid was studied and compared. Moreover associated clinical manifestations were also noted and classified compared. **Result:** The compared biochemical parameters have significant p value ( $p < 0.001$ ) (except AST). The associated clinical manifestations were 35 (23.3%) angina, 12 (8%) MI, 46 (30.6%) type II DM, 27 (18%) IHD, 32 (21.3%) obesity. **Conclusion:** Present pragmatic study has proved that RA being chronic inflammatory disease closely associated with dyslipidemia and CVD risk factors. These value predict CVD risk factors hence these value are alarming for cardiologist, physician to treat such patients efficiently to prevent morbidity and mortality.

## INTRODUCTION

Patients with Rheumatoid Arthritis have an increased morbidity and mortality from cardio-vascular disease and untreated patients have an atherogenic lipid profile which can be positively influenced by the use of disease modifying anti rheumatic drugs (DMARD) therapy.<sup>[1]</sup>

It is observed that, use of gluco- corticoids which are known to cause hyper cholestremia in these patients has also been shown to increase high density lipoprotein (HDL) and reduce total cholesterol / HDL ratio, thus suggesting a causative role for the inflammatory response in generating this lipid abnormality.<sup>[2]</sup> Increased cardio vascular disease in RA patients could have several causes, i.e., the prevalence of new or established cardio vascular risk factors such as dyslipidemia, diabetes mellitus hypertension; higher body mass Index (BMI) chronic inflammatory component RA could be an independent cardio vascular risk factor.<sup>[3]</sup>

There is emerging evidence that, apolipoproteins and lipoprotein are useful predictors for cardio vascular disease,<sup>[4]</sup> as these are present in HDL cholesterol LDL cholesterol, triglycerides and chylomicron particles may increase the risk of cardio vascular diseases (CVD). Hence attempt is made to evaluate the various biochemical parameters and associated

clinical manifestations to rule out the risk factors associated with RA patients.

## MATERIALS AND METHODS

150 adults patients aged between 21 years to 85 years regularly visited to General Medicine, ESIC Medical College, Hyderabad-500038 were studied.

### Inclusive Criteria

The patients diagnosed as RA as per the 1987 Revised American Rheumatology Association (ARA) criteria included in the study.

### Exclusion Criteria

Patients with other forms of arthritis family history of coronary artery diseases diabetes mellitus hypertension thyroid disorders, Kidney and liver disease and patients already on the treatment of lipid lowering drugs were excluded from study.

### Method

150 RA patients given written consent for study and 100 controlled (normal) groups were also selected for study.

After an overnight fasting of 14-16 hours 5ml blood samples of each patient was collected in vacuum tubes and allowed to clot at room temperature for 60-120 minutes followed by centrifugation at 30000g for 10 minutes at 14°C serum was stored as 20°C for estimation of lipid profile and other bio chemical

parameters in aliquots Total cholesterol (As per Laboratory Pvt. Ltd. India) triglycerides (Futura system S. r. L. Italy) and HDL cholesterol (Be canon system pack USA) were estimated by enzymatic methods using kits LDL cholesterol was calculated using Friedewalds formula APO A-1 (Erba Diagnostics Germany).<sup>[5]</sup> All the analysis was done on Beckman synchron CX5 fully automated analyzer USA.

Duration of study was January-2022 to February-2023

### Statistical Analysis

Various biochemical parameters include lipid profile, total protein, magnesium, calcium, and Blood pressures were compared with controlled group with t test. Associated clinical manifestations were classified with percentage. The statistical analysis was carried out in SPSS software. The ratio of male and females was 2:1.

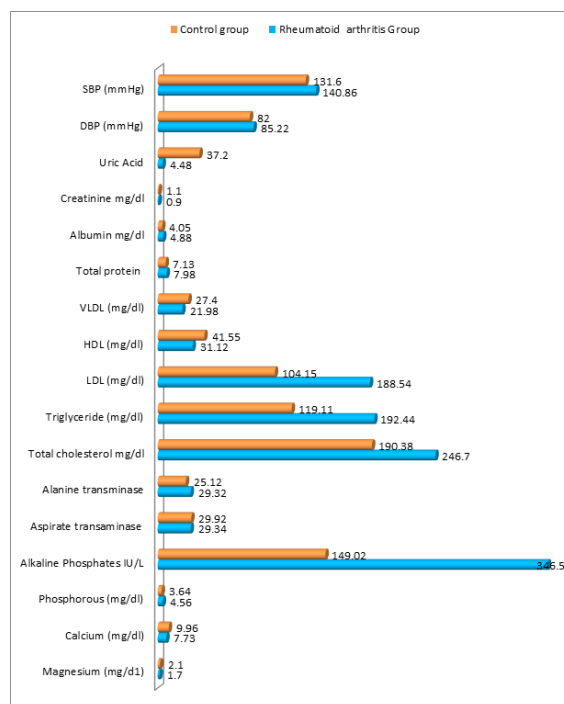
## RESULTS

[Table 1] Comparison of Biochemical parameters in Rheumatoid arthritis patients with dislipademia and cardio vascular risk patients.

- Magnesium (mg/dl) – 1.70 ( $\pm$  0.44) in RA patients, 2.10 ( $\pm$  0.23) in controlled group, t test was 9.37 and  $p < 0.001$ .
- Calcium (mg/dl) – 7.73 ( $\pm$  0.62) in RA patients, 9.96 ( $\pm$  0.64) in controlled group, t test was 27.3 and  $p < 0.001$ .
- Phosphorous (mg/dl) – 4.56 ( $\pm$  1.12) in RA patients, 3.64 ( $\pm$  1.10) in controlled group, t test was 9.37 and  $p < 0.001$ .
- Alkaline Phosphates IU/L – 346.5 ( $\pm$  96.48) in RA patients, 149.02 ( $\pm$  50.24) in controlled group, t test was 21.1 and  $p < 0.001$ .
- Asparate transaminase (IVL) – 29.34 ( $\pm$  10.48) in RA patients, 29.92 ( $\pm$  30.4) in controlled group, t test was 0.18 and  $> 0.85$  (p value insignificant)
- Alaninine transferase (IU/L) – 29.52 ( $\pm$  10.46) in RA patients, 25.12 ( $\pm$  3.31) in controlled group, t test was 3.31 and  $p < 0.001$ .
- Total Cholesterol (mg/dl) – 246.70 ( $\pm$  9.80) in RA patients, 190.58 ( $\pm$  44.33) in controlled group, t test was 12.4 and  $p < 0.001$ .
- Triglyceride (mg/dl) – 192.44 ( $\pm$  0.50) in RA patients, 119.11 ( $\pm$  0.40) in controlled group, t test was 247.5 and  $p < 0.001$ .
- LDL – 188.54 ( $\pm$  1.35) in RA patients, 104.15 ( $\pm$  26.98) in controlled group, t test was 31.2 and  $p < 0.001$ .
- HDL (mg/dl) – 31.12 ( $\pm$  7.30) in RA patients, 41.55 ( $\pm$  8.00) in controlled group, t test was 10.4 and  $p < 0.001$ .

- VLDL (mg/dl) – 29.98 ( $\pm$  8.25) in RA patients, 27.40 ( $\pm$  7.90) in controlled group, t test was 2.48 and  $p < 0.001$ .
- Total Protein – 7.98 ( $\pm$  1.11) in RA patients, 7.13 ( $\pm$  0.65) in controlled group, t test was 7.62 and  $p < 0.001$ .
- Albumin (mg/dl) – 4.88 ( $\pm$  0.68) in RA patients, 4.05 ( $\pm$  0.46) in controlled group, t test was 11.5 and  $p < 0.001$ .
- S. Creatinine (mg/dl) – 0.90 ( $\pm$  0.20) in RA patients, 1.10 ( $\pm$  0.32) in controlled group, t test was 5.56 and  $p < 0.001$ .
- Uric Acid – 4.48 ( $\pm$  1.72) in RA patients, 3.72 ( $\pm$  1.44) in controlled group, t test was 3.77 and  $p < 0.002$ .
- DBP (mm Hg) – 85.22 ( $\pm$  0.96) in RA patients, 82.00 ( $\pm$  0.72) in controlled group, t test was 30.2 and  $p < 0.001$ .
- SBP (mm Hg) – 140.86 ( $\pm$  0.94) in RA patients, 131.60 ( $\pm$  1.30) in controlled group, t test was 61.3 and  $p < 0.001$ .

[Table 2] Associated clinical manifestations in RA patients with dyslipedemia and cardio vascular risk – 35 (23.3%) patients had angina, 12 (8%) had Myocardial infarction, 46 (30.6%) had type-II DM, 27 (18%) had IHD, 32 (21.3%) had obesity.



**Figure 1: Comparative of Biochemical parameters in Rheumatoid Arthritis and control group**

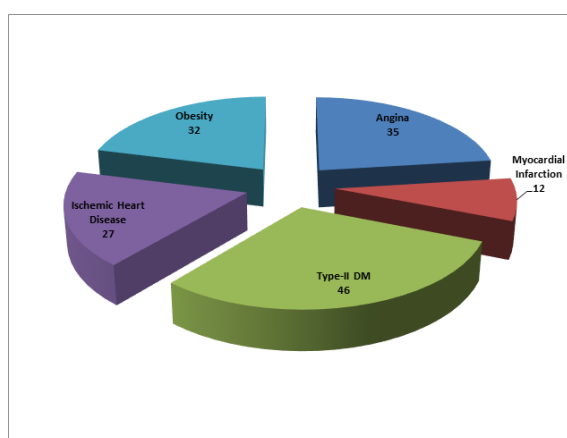
**Table 1: Comparative of Biochemical parameters in Rheumatoid Arthritis and control group**

Biochemical parameters	Rheumatoid arthritis Group (150) Mean value $\pm$ SD	Control group (100) Mean value $\pm$ SD	t test	p value
Magnesium (mg/dl)	1.70 ( $\pm$ 0.44)	2.10 ( $\pm$ 0.23)	9.34	$P < 0.001$
Calcium (mg/dl)	7.73 ( $\pm$ 0.62)	9.96 ( $\pm$ 0.64)	27.3	$P < 0.001$
Phosphorous (mg/dl)	4.56 ( $\pm$ 1.12)	3.64 ( $\pm$ 1.10)	6.43	$P < 0.001$
Alkaline Phosphates IU/L	346.52 ( $\pm$ 96.48)	149.02 ( $\pm$ 50.24)	21.1	$P < 0.001$

Aspartate transaminase	29.34 ( $\pm$ 10.48)	29.92 ( $\pm$ 0.40)	0.18	P>0.85
Alanine transaminase	29.32 ( $\pm$ 10.46)	25.12 ( $\pm$ 9.38)	3.31	P<0.001
Total cholesterol mg/dl	246.70 ( $\pm$ 9.80)	190.38 ( $\pm$ 44.33)	12.4	P<0.001
Triglyceride (mg/dl)	192.44 ( $\pm$ 0.50)	119.11 ( $\pm$ 0.40)	247.1	P<0.001
LDL (mg/dl)	188.54 ( $\pm$ 1.35)	104.15 ( $\pm$ 26.98)	31.2	P<0.001
HDL (mg/dl)	31.12 ( $\pm$ 7.30)	41.55 ( $\pm$ 8.00)	10.4	P<0.001
VLDL (mg/dl)	21.98 ( $\pm$ 8.25)	27.40 ( $\pm$ 7.90)	2.48	P<0.001
Total protein	7.98 ( $\pm$ 1.11)	7.13 ( $\pm$ 0.65)	7.62	P<0.001
Albumin mg/dl	4.88 ( $\pm$ 0.68)	4.05 ( $\pm$ 0.46)	11.5	P<0.001
Creatinine mg/dl	0.90 ( $\pm$ 0.20)	1.10 ( $\pm$ 5.56)	5.56	P<0.001
Uric Acid	4.48 ( $\pm$ 1.72)	37.2 ( $\pm$ 1.44)	3.77	P<0.002
DBP (mmHg)	85.22 ( $\pm$ 0.96)	82.00 ( $\pm$ 0.72)	30.2	P<0.001
SBP (mmHg)	140.86 ( $\pm$ 0.94)	131.60 ( $\pm$ 1.30)	61.3	P<0.001

**Table 2: Associated clinical manifestation in RA patients.**

Sl no	Clinical Manifestation	No of patients	Percentage
1	Angina	35	23.3
2	Myocardial Infarction	12	8
3	Type-II DM	46	30.6
4	Ischemic Heart Disease	27	18
5	Obesity	32	21.3



**Figure 2: Associated clinical manifestation in RA patients**

## DISCUSSION

Present study of dyslipidemia and cardio vascular disease in patients with RA in Telangana population. The biochemical parameters were compared in RA patients with controlled group. The parameters included Magnesium control calcium (mg/dl) Phosphorous, Alkaline phosphate (IU/L) AST, Total cholesterol, triglyceride, LDL, HDL, VLDL, Total protein, Albumin (mg/dl), Cratinine (mg/dl), Uric acid and Blood pressure (DBP, SBP) and all parameters very highly significant p value ( $p < 0.001$ ) except Alamine transamines (ALT) [Table 1]. The Associated clinical manifestation in RA patients were 35 (23.3%) had angina, 12 (8%) had MI, 46 (30.6%) type-II DM, 27 (18%) IHD, 32 (21.6%) were obese (Table-2). These findings are more or less in agreement with previous studies.<sup>[6-8]</sup>

The RA and heart disease share common under pinning involving inflammation. The high levels of inflammation, that characterize rheumatic diseases. The important metabolic feature of RA is the catabolic state leading to loss of body cell mass due to accelerated loss of skeletal muscle.<sup>[7]</sup> This is known as rheumatoid cachexia and important

mediators are TNF  $\alpha$  and other pro inflammatory enzymes. These mediators are also associated with lowered TC and HDL. Levels.<sup>[8]</sup> As a higher activity in RA is accompanied higher TNF level might explain the relationship between disease activity and lipid levels Apolipoprotein A-1, Apolipoprotein-B and lipoprotein is the protein present in HDL-C particles where as apo B found on the LDL-C, VLDL and chylomicrons particles. Hence assessment of plasma apo A.1 and apo lipo protein B allow an assessment of total number of anti-atherogenic and atherogenic particles respectively. LP(a) is modified from LDL in which apo A is bound to apo B. It is reported that apo B is a better predictor of cardio vascular events than LDL-C and apo B and apo-B ratio is an accurate risk factor for cardio vascular diseases (CVD).<sup>[9]</sup> The relation between lipid profile and RA was determined in 1078. When donors later developed RA, and when RA patients displayed higher lipid profiles. It is also hypothesized that, rising lipid profile might be related to the development of RA by a common or linked background. This could be socio- economic (including dietary) or a genetic background. Alternately lipids might modulate the susceptibility to the development of inflammatory diseases such as RA.

The LDL levels were quite lower in RA which associated with risk factors of cardio vascular diseases. It could be due to increased levels of secretory group of phospholipase A2, an acute cardio vascular risk factor. When LDL becomes dense and concentrated, there begins atherosclerosis, LDL in filters the artery wall and oxidized by reactive oxygen to oxidize LDL leads to release of phospholipids activation endothelial cells, there by initiating inflammatory process. This leads to formation of foam cells and subsequent fatty streaks. The HDL acts as pro inflammatory factor to promote inflammation.<sup>[11]</sup> This process elevates phospholipids which will be great risk to cardio-vascular diseases.

## CONCLUSION

The present study of dyslipidemia and cardiovascular risk in RA patients of Telangana population will be quite useful to physician to correlate the lipid profile and associated clinical manifestations to prevalent cardiovascular risk in RA patients. Though Rheumatoid arthritis is an idiopathic inflammatory disease and aggravated by elevation of lipids which ultimately cause risk to cardiovascular system. This study demands further genetic, biochemical, angiological study, because little is known about relation between inflammation and lipoprotein levels which determine the risk to cardiovascular disease in RA patients.

### Limitation of Study

Owing tertiary location of research centre, small number of patients and lack of latest techniques, we have limited findings and results

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