

STUDY OF DYSLIPIDEMIA AND CARDIO VASCULAR RISK PATIENTS WITH RHEUMATOID ARTHRITIS

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ABSTRACT

Background: Rheumatoid arthritis (RA) is a disease of joints with an unknown etiology. Major co-morbidity among the RA patients is cardio-vascular risk due to elevation in lipid profile parameters. **Materials and Methods:** 75 RA, patients compared with 70 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. **Results:** The compared biochemical parameters have significant p value ($p < 0.001$) (except AST). The associated clinical manifestations were 18 (24%) angina, 6 (8%) MI, 23 (30.6%) type-2 DM, 14 (18%) IHD, 16 (21.3%) obesity. **Conclusion:** Present study of RA had prevalence of dyslipidemia. Apart from treating RA It is mandatory to treat atherosclerosis to prevent cardio vascular risk alarming for cardiologist, physician to treat such patients meticulously to prevent morbidity and mortality.

INTRODUCTION

Rheumatoid arthritis (RA) is a chronic autoimmune joint disease of unknown etiology which involves chronic inflammation. It primarily affects small to medium sized joints, causing bone erosions destruction of the joint and eventually loss of joint function.^[1] It also impacts other systems of the body, such as cardio vascular and haematologic systems. Hence cardio vascular disease (CVD) is the most common cause of mortality among the patients with RA due to elevated atherosclerosis or dyslipidemia.^[2] The risk of CVD among RA is estimated to be 50 to 70% patients.^[3]

The lipid profile among RA patients during active or inadequately treated hence it is characterised by decrease in high density lipoprotein (HDL) levels, total cholesterol and low density lipoprotein (LDL) and tracing of apolipoproteins and lipoproteins have been conflicting reports which indicates bad prognosis in patients with RA. Hence an attempt is made to evaluate the lipid profile and associated comorbidities in RA patients.^[4]

MATERIALS AND METHODS

75 adults patients aged between 21 years to 85 years regularly visited to General Medicine, PSP Medical College hospital and research institute, Tambaram, Panruti, Tamil Nadu-631604 were studied.

Inclusive Criteria: The patients diagnosed as RA as per the 1987 Revised American Rheumatology Association (ARA) criteria included in the study and 70 controlled group and the patients who gave their consent in writing for study were selected.

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: 75 RA patients given written consent for study and 70 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 minutes followed by centrifugation at 3000g 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).[5] All the analysis was done on Beckman synchron CX5 fully automated analyzer USA.

Duration of study was June 2025 to May 2026.

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) – 1.72 ($\pm$ 0.42) in RA patients, 2.10 ($\pm$ 0.20) in controlled group, t test was 6.87 and $p < 0.001$.
- Calcium (mg/dl) – 7.72 ($\pm$ 0.60) in RA patients, 9.95 ($\pm$ 0.62) in controlled group, t test was 22 and $p < 0.001$.
- Phosphorous (mg/dl) – 4.55 ($\pm$ 1.10) in RA patients, 3.62 ($\pm$ 1.12) in controlled group, t test was 5.04 and $p < 0.001$.
- Alkaline Phosphates IU/L – 345.5 ($\pm$ 95.55) in RA patients, 148.03 ($\pm$ 50.22) in controlled group, t test was 15.4 and $p < 0.001$.
- Asparate transaminase (IVL) – 29.36 ($\pm$ 10.40) in RA patients, 29.90 ($\pm$ 0.42) in controlled group, t test was 0.41 and > 0.85 (p value insignificant)
- Alaninine transferase (IU/L) – 29.33 ($\pm$ 10.45) in RA patients, 25.10 ($\pm$ 8.24) in controlled group, t test was 2.69 and $p < 0.001$.
- Total Cholesterol (mg/dl) – 245.72 ($\pm$ 9.82) in RA patients, 190.36 ($\pm$ 44.32) in controlled group, t test was 10.5 and $p < 0.001$.

- Triglyceride (mg/dl) – 193.42 ($\pm$ 0.48) in RA patients, 119.10 ($\pm$ 0.42) in controlled group, t test was 9.89 and $p < 0.001$.
- LDL – 189.55 ($\pm$ 13.5) in RA patients, 104.10 ($\pm$ 25.90) in controlled group, t test was 25.14 and $p < 0.001$.
- HDL (mg/dl) – 32 ($\pm$ 7.32) in RA patients, 41.50 ($\pm$ 8.00) in controlled group, t test was 7.35 and $p < 0.001$
- VLDL (mg/dl) – 29.98 ($\pm$ 8.25) in RA patients, 27.42 ($\pm$ 7.92) in controlled group, t test was 5.85 and $p < 0.001$.
- Total Protein – 7.99 ($\pm$ 1.10) in RA patients, 7.11 ($\pm$ 0.65) in controlled group, t test was 5.85 and $p < 0.001$
- Albumin (mg/dl) – 4.89 ($\pm$ 0.60) in RA patients, 4.03 ($\pm$ 0.45) in controlled group, t test was 3.11 and $p < 0.001$.
- S. Creatinine (mg/dl) – 0.92 ($\pm$ 0.02) in RA patients, 1.10 ($\pm$ 0.5) in controlled group, t test was 3.11 and $p < 0.001$.
- Uric Acid – 4.46 ($\pm$ 1.70) in RA patients, 3.73 ($\pm$ 1.42) in controlled group, t test was 2.79 and $p < 0.002$.
- DBP (mm Hg) – 86.22 ($\pm$ 0.95) in RA patients, 82.00 ($\pm$ 0.73) in controlled group, t test was 29.8 and $p < 0.001$.
- SBP (mm Hg) – 142.85 ($\pm$ 0.94) in RA patients, 130.60 ($\pm$ 1.32) in controlled group, t test was 65.1 and $p < 0.001$.

Table 2: Associated clinical manifestations in RA patients with dyslipedemia and cardio vascular risk – 18 (24%) patients had angina, 6 (8%) had Myocardial infarction, 23 (30.6%) had type-2 DM, 14 (18%) had IHD, 16 (21.3%) had obesity.

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

Biochemical parameters	Rheumatoid arthritis Group (75) Mean value $\pm$ SD	Control group (70) Mean value $\pm$ SD	t test	p value
Magnesium (mg/dl)	1.72 ($\pm$ 0.42)	2.10 ($\pm$ 0.20)	6.87	$P < 0.001$
Calcium (mg/dl)	7.72 ($\pm$ 0.60)	9.95 ($\pm$ 0.62)	22	$P < 0.001$
Phosphorous (mg/dl)	4.55 ($\pm$ 1.10)	3.62 ($\pm$ 1.12)	5.04	$P < 0.001$
Alkaline Phosphates IU/L	345.54 ($\pm$ 95.55)	148.03 ($\pm$ 50.22)	15.4	$P < 0.001$
Asparate transaminase	29.38 ($\pm$ 10.40)	29.90 ($\pm$ 0.42)	0.41	$p > 0.67$
Alanine transminase	29.33 ($\pm$ 10.45)	25.10 ($\pm$ 8.24)	2.69	$P < 0.001$
Total cholesterol mg/dl	245.72 ($\pm$ 9.82)	190.36 ($\pm$ 44.32)	10.5	$P < 0.001$
Triglyceride (mg/dl)	193.42 ($\pm$ 48.2)	119.10 ($\pm$ 38.2)	9.89	$P < 0.001$
HDL (mg/dl)	32.10 ($\pm$ 7.32)	41.50 ($\pm$ 8.00)	7.38	$P < 0.001$
VLDL (mg/dl)	22.96 ($\pm$ 8.20)	27.42 ($\pm$ 7.92)	3.32	$P < 0.001$
Total protein	7.99 ($\pm$ 1.10)	7.11 ($\pm$ 0.65)	5.85	$P < 0.001$
Albumin mg/dl	4.89 ($\pm$ 0.60)	4.03 ($\pm$ 0.45)	9.71	$P < 0.001$
Creatinine mg/dl	0.92 ($\pm$ 0.02)	1.10 ($\pm$ 0.5)	3.11	$P < 0.001$
Uric Acid	4.46 ($\pm$ 1.70)	3.75 ($\pm$ 1.42)	2.79	$P < 0.001$
DBP (mmHg)	86.22 ($\pm$ 0.95)	82.00 ($\pm$ 0.73)	29.8	$P < 0.001$
SP (mmHf)	142.85 ($\pm$ 0.92)	13.60 ($\pm$ 1.32)	65.1	$P < 0.001$

Table 2: Associated clinical manifestation in RA patient			(No. of patients: 75)
Sl. No	Clinical Manifestation	No. of patients	Percentage (%)
1	Angina	18	24
2	Myocardial Infarction	6	8
3	Type-2 DM	23	30.6
4	Ischemic Heart Disease	14	18.6
5	Obesity	16	21.3

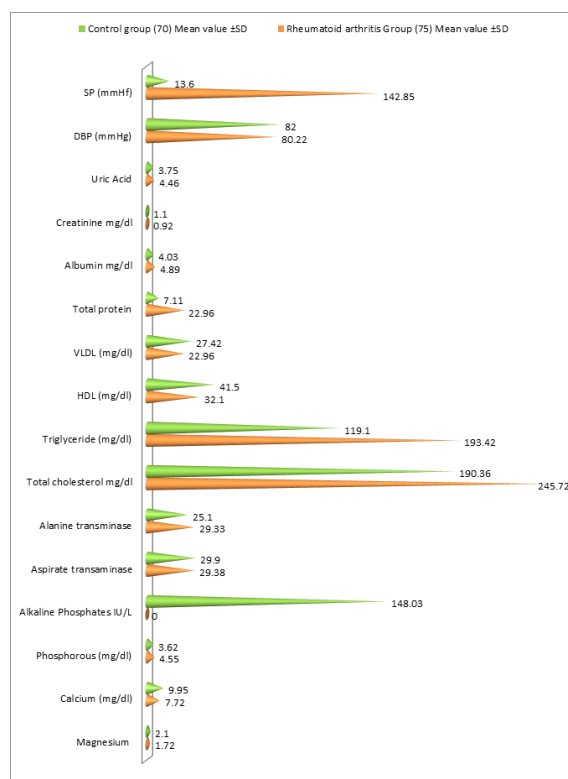


Figure 1: Comparative of Biochemical parameters in Rheumatoid Arthritis and control group

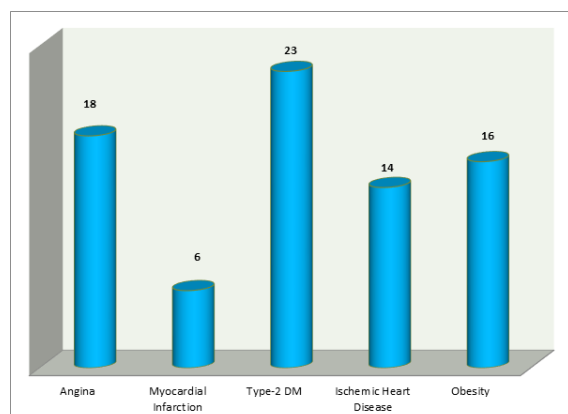


Figure 2: Associated clinical manifestation in RA patient

DISCUSSION

Present study of dyslipidemia and cardio vascular disease in patients with RA in Tamil 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 ($p < 0.11$) except Alamine transamines (ALT) [Table 1]. The Associated clinical manifestation in RA patients were 18 (24%) had angina, 6 (8%) had MI, 23 (30.6%) type-2 DM, 14 (18%) IHD, 16 (21.6%) were obese (Table-2). These findings are more or less in agreement with previous studies.^[6,7,8]

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.^[9] This is known as rheumatoid cachexia and important mediators are TNF α and other pro inflammatory enzymes. These mediators are also associated with lowered TC and HDL. Levels.^[10] 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).^[11] 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. This process elevates phospholipids which will be great risk to cardio-vascular diseases.

CONCLUSION

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

Limitation of study: Owing to remote location of research centre, small number of patients, lack of latest techniques, we have limited findings and results.

- This research work was approved by the ethical committee of PSP Medical College hospital and research institute, Tambaram, Panruti, Tamil Nadu-631604.
- No Conflict of Interest
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