(RESEARCH ARTICLE)

A study of carotid intima media thickness in patients with rheumatoid arthritis and its correlation with acute phase reactants and disease activity

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Abstract

Introduction: There is increased risk of cardiovascular morbidity and mortality in patients of rheumatoid arthritis (RA) due to accelerated atherosclerosis. In this study we measured Carotid artery intima media thickness (CIMT) as a surrogate marker for subclinical atherosclerosis in RA patients without pre-existing cardiovascular disease. This study also analyzed the correlation between CIMT, disease duration, Simplified Disease Activity Index (SDAI) and acute phase reactants.

Methods: A cross-sectional study was conducted on 90 patients divided into two groups of 45 patients of RA and another 45 control subjects. Subjects with RA and age and sex matched healthy controls were included in the study without pre-existing CV risk factors. Complete clinical evaluation, hematological and biochemical profile done. ESR, CRP and platelet count were used as markers of acute phase reactants. All the RA patients included in the study were evaluated for their disease activity using Simplified Disease Activity Index (SDAI). All the subjects including the controls evaluated for carotid intima media thickness by using carotid ultrasonography. CIMT measured in common carotid artery bilaterally by examining throughout common carotid artery up to 2 cm proximal to bifurcation. CIMT measurement taken at the site of greatest thickness. All measurements taken in diastole, measured in phase when lumen diameter is at its smallest and IMT at its largest. Mean value of 6 readings (3 from each side) taken as final CIMT for evaluation. B mode (linear probe) USG was used to determine carotid IMT.

Results: 45 cases and similar control subjects included in the study with mean age (SD) as 45.07 (14.81) years. We found female preponderance with 37 females out of 45 cases. Mean (SD) disease duration in male subjects was 5.38(2.40) years while in females was 5.09 (2.78) years. CRP was found to be significantly increased in cases group with mean (SD) of 5.75(8.85) mg/dl. ESR significantly raised to 35.22(13.55) mm/hour in cases group compared to control group where it was 16.16(5.71) mm/hr. Mean (SD) platelets ($x10^3/mm^3$) were 229.24(69.80) in cases while controls had 290.33(83.85), so platelets correlation with disease could not be established. CIMT was significantly raised in cases group. Mean (SD) CIMT in cases was 0.95(1.06) mm whereas in control group it was 0.60(0.16) mm with p value of 0.002. Moderate positive correlation was found between CIMT (mm) and SDAI and this correlation was statistically significant with p=0.001. For every 1 unit increase in CIMT, the SDAI increases by 2.92 units. A strong positive correlation between CIMT (mm) and Disease Duration (Years), and this correlation was statistically significant (rho = 0.9, p = <0.001). For every 1 unit increase in CIMT (mm), the Disease Duration (Years) increases by 1.26 units. Conversely, for every 1 unit increase in Disease Duration (Years), the CIMT (mm) increases by 0.19 units.

Conclusion: A strong correlation was observed between CIMT and disease duration in rheumatoid arthritis. Hence, CIMT can be a useful surrogate marker for detecting atherosclerosis in patients with RA. In view of the relation to duration of disease, established RA patients should regularly be screened to identify the evidence of atherosclerosis and manage it earlier. Prevention of cardiovascular disease in RA requires an integrated approach encompassing

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cardiovascular risk factors screening and management, effective and sustained control of RA disease activity, high index of suspicion and prompt investigation of suspected cardiac disease.

1. Introduction

Rheumatoid arthritis (RA) is a chronic systemic inflammatory, auto-immune disease of unknown origin with characteristic persistent symmetric polyarthritis (synovitis) and extra articular involvement of skin, heart, lungs and eye. [1] Although a disease of joints, abnormal immunological responses can lead to a variety of extra-articular manifestations including the involvement of blood vessels and heart. [2]

The risk of death is two-fold higher in RA patients than in general population and the main cause of mortality is cardiovascular disease (CVD), accounting for about a half of premature deaths observed. The global mortality incidence in RA patients attributable to CVD is approximately 40%. [3]

Many studies have previously shown that patients with RA have premature atherosclerosis as measured by increased intima media thickness (IMT) of the common carotid artery compared with controls.[4] Carotid IMT is a convenient, non-invasive marker, which usually can be used to assess subclinical atherosclerosis and the progression of CVD in clinical practice.[5] Carotid intima-media thickness (CIMT) is a simple, reliable, inexpensive, non-invasive marker that is increasingly being used to detect subclinical atherosclerosis and has been recommended by the American Heart Association (AHA), American Society of Echocardiography (ASE) and Society for Vascular Medicine (SVM) as a screening test for heart disease in apparently healthy individuals.[6] CIMT has been used in several clinical trials as a surrogate end point for evaluating the regression and/or progression of atherosclerotic cardiovascular disease.

Increased atherosclerosis in carotid arteries holds true for atherosclerosis for multiple vascular beds including coronaries, and so measurement of carotid IMT is an important marker of increased cardiovascular risk including acute coronary syndromes. [6]

Because of the high incidence of cardiovascular events observed in patients with RA, an important step forward might be to identify high-risk individuals who would benefit from active therapy to prevent clinical disease. [7]

2. Materials and methods

2.1. Place of study

The study was conducted in the Department of Medicine and Department of Radiology of NDMC Medical College & Hindu Rao Hospital, Delhi.

2.2. Study design

Cross-sectional study conducted between Jan 2021-July 2021.

2.3. Inclusion criteria

- Patients of RA diagnosed on basis of 2010 revised American college of Rheumatology/ European League Against Rheumatism criteria attending Rheumatology Clinic of Hindu Rao hospital was taken up as cases.
- Adult patients (Age > 18 years)

2.4. Exclusion criteria

- Patients with diabetes mellitus
- Patients with hypertension (BP > 140/90 mmHg) or use of antihypertensive medications.
- Patients with pre-existing CVD
- Patients on high dose steroids > 10mg/dl
- Patients with kidney disease
- Patients with dyslipidemia (Total cholesterol>240mg/dl, LDL >160 mg/dl, triglycerides >200 mg/dl) or use of lipid lowering medication.

- Smokers
- Patients with liver disease
- Arthritis of any other cause including Juvenile Rheumatoid Arthritis

3. Methodology

- Known case of RA diagnosed on basis of 2010 revised American college of Rheumatology /European League Against Rheumatism criteria attending Rheumatology Clinic of Hindu Rao hospital was taken up as cases.
- Complete clinical evaluation, hematological and biochemical profile was performed. ESR, CRP and platelet count were used as markers of acute phase reactants.
- Two groups made, 45 patients of RA and another 45 healthy (age and sex matched) subjects were taken into other group as controls.
- All the RA patients included in the study were evaluated for their disease activity using Simplified Disease Activity Index (SDAI)

Figure 1 Simple Disease Activity Index scoring using swollen and tender joint in both sides
MCP- metacarpophalangeal Joint, PIP- proximal interphalangeal Joint

All the subjects including the controls were evaluated for carotid intima media thickness by using carotid ultrasonography. Carotid ultrasonography was carried out by skilled radiologist by using grey scale ultrasonography and then followed by color flow imaging.

CIMT was measured in common carotid artery bilaterally by examining throughout common carotid artery up to 2 cm proximal to bifurcation. CIMT measurement was taken at the site of greatest thickness, and three readings were taken from each side at different points within region of interest. All measurements were taken in diastole, measured in phase when lumen diameter is at its smallest and IMT at its largest. Mean value of 6 readings (3 from each side) were taken as final CIMT for evaluation.

5. B mode (linear probe) USG was used to determine carotid IMT. Following table shows normal CIMT which is based on many studies, according to age and gender:

Table 1 Normal CIMT Values as per age and sex

AGE (Years)	Right CIMT (mm)		Left CIMT (mm)	
	Men	Women	Men	Women
<30	0.39-0.48	0.39-0.43	0.42-0.49	0.30-0.47
31-40	0.42-0.50	0.42-0.49	0.44-0.57	0.44-0.51
41-50	0.46-0.57	0.44-0.53	0.50-0.61	0.46-0.57
>50	0.46-0.62	0.50-0.59	0.53-0.70	0.52-0.64

3.1. AGE (Years)

According to Homa et al, intima media thickness of common carotid artery (measured at areas devoid of plaque) increases linearly with age from 0.48 mm at 40 years of age to 1.02 mm at 100 years of age (following a formula 0.009 x age + 0.116 mm).[8]

6. CIMT was then compared with disease activity, duration of disease and level of acute phase reactants.

3.2. Observations and Results

We included 45 patients of Rheumatoid Arthritis and similar number of age and sex matched controls

Table 2 Comparison of the 2 sub-group of variables in terms of age in Years (n = 90)

Age (Years)	Group		t-test	
	Case	Control	t	p value
Mean (SD)	45.07 (14.81)	44.93 (14.66)	0.043	0.966
Median (IQR)	45 (35-53)	45 (35-53)		
Range	18 - 85	18 – 85		

There was no significant difference between the groups in terms of Age (Years)0.043, p = 0.966).

Table 3 Association Between Group and Age (n = 90)

Age	Group			Fisher's Exact Test	
	Case	Control	Total	χ2	P Value
18-30 Years	7 (15.6%)	7 (15.6%)	14 (15.6%)	0.090	1.000
31-40 Years	9 (20.0%)	9 (20.0%)	18 (20.0%)		
41-50 Years	14 (31.1%)	13 (28.9%)	27 (30.0%)		
51-60 Years	9 (20.0%)	10 (22.2%)	19 (21.1%)		
61-70 Years	4 (8.9%)	4 (8.9%)	8 (8.9%)		
71-80 Years	1 (2.2%)	1 (2.2%)	2 (2.2%)		
81-90 Years	1 (2.2%)	1 (2.2%)	2 (2.2%)		
Total	45 (100.0%)	45 (100.0%)	90 (100.0%)		

There was no significant difference between the various groups in terms of distribution of Age (χ2 = 0.090, p = 1.000).

Table 4 Association Between Group and Gender (n = 90)

Gender	Group			Chi-Squared Test	
	Case	Control	Total	χ2	P Value
Male	8 (17.8%)	8 (17.8%)	16 (17.8%)	0.000	1.000
Female	37 (82.2%)	37 (82.2%)	74 (82.2%)		
Total	45 (100.0%)	45 (100.0%)	90 (100.0%)		

There was no significant difference between the various groups in terms of distribution of Gender (χ2 = 0.000, p = 1.000).

Table 5 Comparison of the 2 Subgroups of Variable Group in Terms of CRP (mg/L) (n = 90)

CRP	Group			Chi-Squared Test	
	Case	Control	Total	χ2	P Value
≤6 mg/L	30 (66.7%)	45 (100.0%)	75 (83.3%)	18.000	<0.001
>6 mg/L	15 (33.3%)	0 (0.0%)	15 (16.7%)		
Total	45 (100.0%)	45 (100.0%)	90 (100.0%)		

There was a significant difference between the 2 groups in terms of CRP (mg/L) (W = 1665.000, p = <0.001), with the median CRP (mg/L) being highest in the Group: Case group.

Table 6 Association Between Group and CRP (n = 90)

CRP	Group			Chi-Squared Test	
	Case	Control	Total	χ2	P Value
≤6 mg/L	30 (66.7%)	45 (100.0%)	75 (83.3%)	18.000	<0.001
>6 mg/L	15 (33.3%)	0 (0.0%)	15 (16.7%)		
Total	45 (100.0%)	45 (100.0%)	90 (100.0%)		

There was a significant difference between the various groups in terms of distribution of CRP (χ2 = 18.000, p = <0.001).

Table 7 Comparison of the 2 Subgroups of the Variable Group in Terms of ESR (mm/Hr) (n = 90)

ESR (mm/Hr)	Group		Wilcoxon-Mann-Whitney U Test	
	Case	Control	W	p value
Mean (SD)	35.22 (13.55)	16.16 (5.71)	1802.000	<0.001
Median (IQR)	36 (24-45)	14 (12-20)		
Range	10 - 62	5 – 32		

There was a significant difference between the 2 groups in terms of ESR (mm/Hr) (W = 1802.000, p = <0.001), with the median ESR (mm/Hr) being highest in the Group: Case group.

Table 8 Association Between Group and ESR (n = 90)

ESR	Group			Chi-Squared Test	
	Case	Control	Total	χ2	P Value
≤20 mm/Hr	6 (13.3%)	34 (75.6%)	40 (44.4%)	35.280	<0.001
>20 mm/Hr	39 (86.7%)	11 (24.4%)	50 (55.6%)		
Total	45 (100.0%)	45 (100.0%)	90 (100.0%)		

There was a significant difference between the various groups in terms of distribution of ESR (χ2 = 35.280, p = <0.001).

Table 9 Comparison of the 2 Subgroups of the Variable Group in Terms of Platelet count ($x10^3$/mm^3) (n = 90)

Platelet count ($x10^3$/mm^3)	Group		Wilcoxon-Mann-Whitney U Test	
	Case	Control	W	p value
Mean (SD)	229.24 (69.80)	290.33 (83.85)	571.000	<0.001
Median (IQR)	214 (184-273)	289 (243-351)		
Range	115 - 375	101 – 450		

There was a significant difference between the 2 groups in terms of Platelet count ($x10^3$/mm^3) (W = 571.000, p = <0.001), with the median Platelet count ($x10^3$/mm^3) being highest in the Group: Control group.

Table 10 Comparison of the 2 Subgroups of the Variable Group in Terms of CIMT (mm) (n = 90)

CIMT (mm)	Group		Wilcoxon-Mann-Whitney U Test	
	Case	Control	W	p value
Mean (SD)	0.95 (1.06)	0.60 (0.16)	1403.000	0.002
Median (IQR)	0.8 (0.53-0.98)	0.6 (0.45-0.75)		
Range	0.24 - 7.5	0.31 - 0.91		

There was a significant difference between the 2 groups in terms of CIMT (mm) (W = 1403.000, p = 0.002), with the median CIMT (mm) being highest in the Group: Case group.

Table 11 Correlation between Age (Years) and Disease Duration (Years) (n = 45)

Correlation	Spearman Correlation Coefficient	P Value
Age (Years) vs Disease Duration (Years)	0.3	0.045

There was a moderate positive correlation between Age (Years) and Disease Duration (Years), and this correlation was statistically significant (rho = 0.3, p = 0.045).
For every 1 unit increase in Age (Years), the Disease Duration (Years) increases by 0.06 units. Conversely, for every 1 unit increase in Disease Duration (Years), the Age (Years) increases by 1.90 units.

Table 12 Comparison of the 2 Subgroups of the Variable Gender in Terms of Disease Duration (Years) (n = 45)

Disease Duration (Years)	Gender		Wilcoxon-Mann-Whitney U Test	
	Male	Female	W	p value
Mean (SD)	5.38 (2.40)	5.09 (2.78)	163.500	0.654
Median (IQR)	5 (3.88-6.62)	5 (3-7)		
Range	2 - 9	1.5 – 11		

There was no significant difference between the groups in terms of Disease Duration (Years) (W = 163.500, p = 0.654).
Strength of Association (Point-Biserial Correlation) = 0.04 (Little/No Association)

Table 13 Correlation between CRP (mg/L) and Disease Duration (Years) (n = 45)

Correlation	Spearman Correlation Coefficient	P Value
CRP (mg/L) vs Disease Duration (Years)	0.2	0.264

There was no statistically significant correlation between CRP (mg/L) and Disease Duration (Years) (rho = 0.17, p = 0.264).

Table 14 Correlation between ESR (mm/hour) and Disease Duration (Years) (n = 45)

Correlation	Spearman Correlation Coefficient	P Value
ESR (mm/Hr) vs Disease Duration (Years)	0.2	0.116

There was no statistically significant correlation between ESR (mm/Hr) and Disease Duration (Years) (rho = 0.24, p = 0.116).

Table 15 Correlation between SDAI and Disease Duration (Years) (n = 45)

Correlation	Spearman Correlation Coefficient	P Value
SDAI vs Disease Duration (Years)	0.4	0.010

There was a moderate positive correlation between SDAI and Disease Duration (Years), and this correlation was statistically significant (rho = 0.38, p = 0.010).
For every 1 unit increase in SDAI, the Disease Duration (Years) increases by 0.07 units.
Conversely, for every 1 unit increase in Disease Duration (Years), the SDAI increases by 1.00 units.

Table 16 Correlation between CIMT (mm) and Disease Duration (Years) (n = 45)

Correlation	Spearman Correlation Coefficient	P Value
CIMT (mm) vs Disease Duration (Years)	0.9	<0.001

There was a strong positive correlation between CIMT (mm) and Disease Duration (Years), and this correlation was statistically significant (rho = 0.9, p = <0.001).
For every 1 unit increase in CIMT (mm), the Disease Duration (Years) increases by 1.26 units.
Conversely, for every 1 unit increase in Disease Duration (Years), the CIMT (mm) increases by 0.19 units.

Table 17 Correlation between Age (Years) and CIMT (mm) (n = 45)

Correlation	Spearman Correlation Coefficient	P Value
Age (Years) vs CIMT (mm)	0.3	0.029

There was a moderate positive correlation between Age (Years) and CIMT (mm), and this correlation was statistically significant (rho = 0.33, p = 0.029).
For every 1 unit increase in Age (Years), the CIMT (mm) increases by 0.01 units.
Conversely, for every 1 unit increase in CIMT (mm), the Age (Years) increases by 1.21 units.

Table 18 Comparison of the 2 Subgroups of the Variable Gender in Terms of CIMT (mm) (n = 45)

CIMT (mm)	Gender		Wilcoxon-Mann-Whitney U Test	
	Male	Female	W	p value
Mean (SD)	0.84 (0.27)	0.98 (1.16)	167.000	0.583
Median (IQR)	0.9 (0.74-0.95)	0.74 (0.53-0.98)		
Range	0.4 - 1.25	0.24 - 7.5		

There was no significant difference between the groups in terms of CIMT (mm) (W = 167.000, p = 0.583).

Table 19 Correlation between CRP (mg/L) and CIMT (mm) (n = 45)

Correlation	Spearman Correlation Coefficient	P Value
CRP (mg/L) vs CIMT (mm)	0.3	0.074

There was no statistically significant correlation between CRP (mg/L) and CIMT (mm) (rho = 0.27, p = 0.074).

Table 20 Correlation between ESR (mm/hour) and CIMT (mm) (n = 45)

Correlation	Spearman Correlation Coefficient	P Value
ESR (mm/Hr) vs CIMT (mm)	0.3	0.064

There was no statistically significant correlation between ESR (mm/Hr) and CIMT (mm) (rho = 0.28, p = 0.064).

Table 21 Correlation between SDAI and CIMT (mm) (n = 45)

Correlation	Spearman Correlation Coefficient	P Value
SDAI vs CIMT (mm)	0.5	0.001

There was a moderate positive correlation between SDAI and CIMT (mm), and this correlation was statistically significant (rho = 0.47, p = 0.001).
For every 1 unit increase in SDAI, the CIMT (mm) increases by 0.03 units.
Conversely, for every 1 unit increase in CIMT (mm), the SDAI increases by 2.92 units.

Table 22 Correlation between Platelet count ($x10^3/mm^3$) and CIMT (mm) (n = 45)

Correlation	Spearman Correlation Coefficient	P Value
Platelet count ($x10^3/mm^3$) vs CIMT (mm)	0.3	0.025

There was a moderate positive correlation between Platelet count ($x10^3/mm^3$) and CIMT (mm), and this correlation was statistically significant (rho = 0.33, p = 0.025).
For every 1 unit increase in Platelet count ($x10^3/mm^3$), the CIMT (mm) increases by 0.00 units.
Conversely, for every 1 unit increase in CIMT (mm), the Platelet count ($x10^3/mm^3$) increases by 19.48 units.

4. Discussion

Rheumatoid Arthritis is a characterized by symmetric polyarthritis. It is a chronic inflammatory disease with no known etiology. Most common cause of death in patients with RA is cardiovascular disease. The incidence of coronary artery disease and carotid atherosclerosis is higher in RA patients than in general population even when controlling for traditional risk factors, such as hypertension, obesity, hypercholesterolemia, diabetes, and cigarette smoking. [9]

Atherosclerosis, a disease of arterial system of our body, in which the blood vessel wall will become thickened and hardened by "plaques". Atherosclerotic plaque is composed of cholesterol and other lipids, inflammatory cells, and calcium deposits. Atherosclerosis is an inflammatory disease. There are many similarities between the inflammatory and immunological mechanisms operating in atherosclerotic plaque and in rheumatoid synovitis. The common pathophysiological features in the affected tissues include an abundance of activated macrophages which release or induce release of inflammatory mediators, including cytokines (e.g., IL-1 and TNF), adhesion molecules with matrix metalloproteinases, growth factors and T-cell infiltrates.

Both atherosclerosis and Rheumatoid arthritis are associated with elevated levels of acute phase reactants – C-reactive protein (CRP), serum amyloid A, ESR, fibrinogen, and secondary phospholipase 2. [10]

CIMT is a reliable marker for coronary atherosclerosis and peripheral vascular disease. [11]

Total sample size of 90 with 45 cases and 45 age and sex matched control was taken in the study. Mean (SD) age for cases group 45.07(14.81) years which was comparable with that of control group of 44.93(14.66). Cases group in the study includes 17.8% Male and 82.2% Female. We got female preponderance of the disease as supported by various previous studies. [6] In a study by Gonzalez-Juanatey et al they also mentioned that from 118 patients they recruited for the study, 75.4% were women.[7] In our study the mean CIMT was found to be significantly higher in Case group when compared to control group with p value of 0.002 which is statistically significant. The mean (SD) of CIMT (mm) in the Case group was 0.95 (1.06) while in the control group was 0.60 (0.16). This clearly suggests that CIMT increases in patients with Rheumatoid arthritis compared to normal population. Saigal R et al [1] also found significantly higher CIMT in RA patients (0.68_+ 0.06mm). Singh et al also reported higher CIMT in RA patients compared to controls, [12] Patel S, Bhatt K, Patel A et al found mean CIMT in RA group to be 0.86_+0.18 mm and that of control group as 0.53+_0.15 mm .[6]

There was a significant difference between the cases and control groups in terms of distribution of ESR ($\chi2$ = 35.280, p = <0.001). 86.7% of the participants in cases group had ESR: >20 mm/hour while only 24.4% of the participants had ESR: >20 mm/hour in control group. So, Control had the larger proportion of ESR: ≤20 mm/hour and Cases had the larger proportion of ESR: >20 mm/Hr. Saigal R et al [1] and Mandal et al [13] also found significantly higher ESR in RA patients (51.75+_24.13 mm/hour) in comparison to control group.

The mean (SD) of CRP (mg/L) in the Case group was 5.75 (8.85) while in control group was 0.60 (0.00). There was a significant difference between the 2 groups in terms of CRP (mg/L) (W = 1665.000, p = <0.001), with the median CRP (mg/L) being highest in the Case group.

We found significantly high CRP and ESR in RA cases compared to control group. This indicates that age alone is not enough to explain the increase in CIMT in RA cases, but inflammation also had some contributing factor to it. Many previous studies also support that inflammation is a contributing factor in atherosclerosis development. [14-16] RA patients who receive TNF-alfa blockers had reduction in CIMT most probably due to reduction in inflammation. [17] All these reports support that inflammation has the contributing role in development of atherosclerosis. Elevated levels of cytokines such as tumor necrosis factor-α (TNF-α), interleukin-17 (IL-17), interleukin-6 (IL-6) and interleukin-1β (IL-1β) are found in both entities, namely, RA and CVD, with higher levels being present in RA. [3]

However, the mean (SD) of Platelet count ($x10^3/mm^3$) in the Case group was 229.24 (69.80) and the mean (SD) of Platelet count ($x10^3/mm^3$) in the Control group was 290.33 (83.85). There was a significant difference between the 2 groups in terms of Platelet count ($x10^3/mm^3$) with the median Platelet count ($x10^3/mm^3$) being highest in the Control group. Mandal SK, Sarkar P, Sarkar RN, et al [13] demonstrated platelet count ($x10^3/mm^3$) as 383 in cases group while 311 in control group. We found that platelet count failed to be associated with atherosclerosis.

SDAI (Simplified Disease Activity Index) have been developed to overcome the major practical limitations of the DAS-based indices. The SDAI is a valid and sensitive assessment of disease activity and treatment response that is comparable with the DAS 28 and ACR response criteria; it is easy to calculate and therefore a viable tool for day-to-day clinical assessment of RA treatment. Overall results indicate that the SDAI has content, criterion and construct validity. [18] SDAI, DAS, and DAS28 all require lab parameters (CRP or ESR), which constitutes limitation in clinical practice as it often prevents immediate assessment because of waiting time for lab results. Though DAS28 has not been tested using clinical variables only, on the other hand SDAI has shown to convey similar results even when CRP was eliminated from the formula. This simplified index, based solely on clinical measures termed Clinical Disease Activity Index (CDAI).

The mean Patient Global Activity was 4.93 ± 2.91, mean Provider Global Activity was 4.27 ± 2.86, mean SDAI was 16.00 ± 10.10. Out of 45 cases, 9 (20.0%) of the participants had Disease Activity as High, 21 (46.7%) had Disease Activity as Moderate, 14 (31.1%) had Disease Activity as Low and 1 (2.2%) of the participant had Disease Activity in Remission.

The mean (SD) of SDAI in the CIMT: ≤0.8 mm group was 11.54 (9.61) while in the CIMT: >0.8 mm group was 20.67 (8.50). There was a significant difference between the 2 groups in terms of SDAI (W = 111.000, p = 0.001), with the median SDAI being highest in the CIMT: >0.8 mm group. Patients with high and moderate disease activity had CIMT> 0.8mm.

The study depicts the correlation between CIMT (mm) and SDAI.

There was a moderate positive correlation between CIMT (mm) and SDAI, and this correlation was statistically significant (rho = 0.47, p = 0.001). For every 1 unit increase in CIMT (mm), the SDAI increases by 2.92 units. Conversely, for every 1 unit increase in SDAI, the CIMT (mm) increases by 0.03 units. Maldar et al [19] found a strong correlation between CIMT and disease activity in patients with RA. Therefore CIMT can be a useful surrogate marker for detecting atherosclerosis in patients with RA.

There was a moderate positive correlation between SDAI and Disease Duration (Years), and this correlation was statistically significant (rho = 0.38, p = 0.010). For every 1 unit increase in SDAI, the Disease Duration (Years) increases by 0.07 units. Conversely, for every 1 unit increase in Disease Duration (Years), the SDAI increases by 1.00 units. The mean (SD) Disease Duration (Years) in the high SDAI group was 5.72 (2.61), Moderate SDAI group was 5.79 (2.36), Low SDAI group was 3.96 (3.02), Remission SDAI group was 3.00 (NA).

There was a strong positive correlation between CIMT (mm) and Disease Duration (Years), and this correlation was statistically significant (rho = 0.9, p = <0.001). For every 1 unit increase in CIMT (mm), the Disease Duration (Years) increases by 1.26 units. Conversely, for every 1 unit increase in Disease Duration (Years), the CIMT (mm) increases by

0.19 units. The mean Disease Duration (Years) found to be highest in the CIMT: >0.8 mm group. Mandal S K et al also found that carotid atherosclerosis in RA patients is increased with age and with longer duration of disease. Tiwari et al [20] and Maldar et al [19] also found that as the duration of disease progresses the CIMT tends to increase. Disease duration is one of the best predictors for the development of atherosclerotic disease. Moreover, the CIMT increases significantly with duration of disease. Studies conducted also observed a statistically significant association of disease duration with CIMT and atherosclerotic plaques. [12][21]

In view of the relation to duration of disease, physicians should regularly screen the established RA patients so that to identify the evidence of atherosclerosis and manage it earlier. Prevention of cardiovascular disease in RA requires an integrated approach encompassing cardiovascular risk factors screening and management, effective and sustained control of RA disease activity, high index of suspicion and prompt investigation of suspected cardiac disease.

5. Conclusion

A strong correlation was observed between CIMT and disease duration in rheumatoid arthritis. Hence, CIMT can be a useful surrogate marker for detecting atherosclerosis in patients with RA. In view of the relation to duration of disease, physicians should regularly screen the established RA patients so that to identify the evidence of atherosclerosis and manage it earlier. Prevention of cardiovascular disease in RA requires an integrated approach encompassing cardiovascular risk factors screening and management, effective and sustained control of RA disease activity, high index of suspicion and prompt investigation of suspected cardiac disease.

Compliance with ethical standards

Disclosure of conflict of interest

There are no conflict of interest.

Reference

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