INTIMA-MEDIA THICKNESS OF THE COMMON CAROTID ARTERY IN RHEUMATOID ARTHRITIS PATIENTS

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KEY WORDS: INTIMA-MEDIA THICKNESS, RHEUMATOID ARTHRITIS.

ABSTRACT

Objective: To measure the intima-media thickness (IMT) of the common carotid artery (as an index of subclinical atherosclerosis) and to evaluate the factors associated with arterial wall thickness in RA patients.

Methodology: We used an accurate and reliable imaging technique, high-resolution B-mode ultrasound, to compare common carotid artery (CCA) intima-media wall thickness (IMT) in 40 RA patients and 40 controls. The apparently healthy subjects were comparable with the RA patients as regards the risk factors for atherosclerosis, including age, sex, menopause status, body mass index (BMI), blood pressure, and serum lipid levels. We investigated the association between (IMT) of the (CCA) in RA and clinical and therapeutic variables.

Results: The mean level of IMT of the CCA showed a statistically highly significant increase (p<0.001) in RA patients as compared to controls. There were statistically significant correlations (p<0.05) between IMT of the CCA in RA and disease related variables; duration of morning stiffness, articular index, grip strength, ESR, hemoglobin level, pain severity and rheumatoid factor. Highly statistically significant correlations (p<0.001) were found in relation to age, disease duration, spread severity index, Larsen score, HAQ and CRP. Insignificant correlations (p>0.05) were found between IMT of the CCA in RA patients and sex, BMI, systolic and diastolic blood pressure, lipid profile and drug treatment.

Conclusions: RA patients exhibited greater thickness of the common carotid artery than healthy controls, so RA patients have an ultrasonic marker of early atherosclerosis. The age, disease duration, disease activity and severity, decreased physical activity, but not therapeutic variables, were associated with the increased arterial wall thickness.

INTRODUCTION

Reports of a number of studies have suggested that there is increased cardiovascular disease and mortality among rheumatoid arthritis (RA) patients. Turesson & Matteson (2007) and Kitas & Erb (2003) reported that the most common cause of death in RA is cardiovascular disease (53%) mainly due to ischemic heart disease (IHD). Cardiac death was more than double in RA patients as compared to the general population (Pham et al., 2006). Furthermore, 70% of RA...
patients were more likely to die and 30-40% of them were more likely to suffer from IHD (Troelsen & Jacobsen, 2006).

Atherosclerosis is a complex and indolent histopathological process, which is considered to be the most common underlying process in cardiovascular morbidity and mortality (Abou-Ray'a & Abou-Ray'a, 2006).

The carotid arteries are easily accessible to ultrasound techniques, and these techniques provide accurate and reliable measurement of atherosclerosis in the subclinical stages (Li et al., 1996).

Measurement of the intima-media thickness (IMT) of the far wall of the common carotid artery with high-resolution ultrasonography has been established as a clinically useful index for identifying early-stage atherosclerosis (Del-Rincon et al., 2003).

**Aim of Work:**

The aim of this study was to measure the intima-media thickness (IMT) of the common carotid artery (as an index of subclinical atherosclerosis) and to evaluate the factors associated with arterial wall thickness in RA patients. We used an accurate and reliable imaging technique, high-resolution B-mode ultrasound, for the diagnosis of subclinical atherosclerosis.

**SUBJECTS AND METHODS**

This study was carried out on forty rheumatoid arthritis (RA) patients attending the Outpatient Clinic of the Rheumatology & Rehabilitation Department of Benha University Hospitals. They were 37 females (92.5%) and 3 males (7.5%). Their age ranged between 29 and 63 years with a mean of 40.8 ± 7.12 years and the duration of their disease ranged from 3 to 26 years with a mean of 9.62 ± 6.8 years.

Our RA patients were diagnosed according to the revised American College of Rheumatology (ACR) Criteria (Arnett et al., 1988). Another forty cross matched apparently healthy individuals were also included in this study as a control group.

Exclusion criteria included patients not fulfilling the revised ACR criteria for the diagnosis of RA or had other rheumatic diseases, patients with chronic medical illness as chronic liver and renal diseases, endocrine disorders, diabetes or hypertension, patients known to have ischemic heart disease (IHD) or had previous history of admission to coronary care unit, history of hyperlipidemia (total cholesterol >240 mg/dl, triglycerides >200 mg/dl and low density lipoprotein cholesterol (LDL-c) >160 mg/dl), obesity (body mass index (BMI) >30 kg/m²), history of oral contraceptive pills intake or history of smoking. All patients were subjected to the following:

**Clinical Assessment:**

**Full history taking:**

This was specially age, disease duration, duration of morning stiffness, symptoms of cardiovascular disease, extra-articular manifestations and drug history.

**Thorough clinical examination:**

General examination and local examination especially blood pressure, body mass index, locomotory system, skin, chest, heart, and nervous system, assessment of disease severity using the spread severity index (Walker et al., 1985), assessment of radiological severity using Larsen method (Larsen et al., 1977), with plain X-rays to both hands and wrists. Assessment of the patient's physical activity using both Steinbrocker (1949) grades and Stanford Health Assessment Questionnaire (HAQ) (Kirwan & Reebback, 1986) and assessment of disease activity using the multivariate analysis according to Mallya & Mace (1981) were done.

**Assessment of clinical disease severity:**

This involved scoring the joints with spread severity index (Walker et al., 1985):
- Score 0 = Normal.
- Score 1 = Pain.
- Score 2 = Swelling.
- Score 3 = Pain and swelling.
- Score 4 = Deformity.

One score was taken for each of the following pairs of groups of joints: proximal interphalangeal (PIPs), metacarpophalangeal (MCPs), wrist, elbow, shoulder, hip, knee, ankle, metatarsophalangeal, and cervical spine, and one score for the worst other affected joint. The maximum possible score was 44 (Walker et al., 1985).

Functional assessment:

It was assessed with two methods:

A) Steinbrocker grading (Steinbrocker et al., 1949). Functional capacity of RA patient was graded into:

   **Grade I**: the patient can perform all activities.

   **Grade II**: He can perform normal activities but with the handicap of pain or limited joint motion.

   **Grade III**: Activities are limited to self-care.

   **Grade IV**: The patient is confined to a wheelchair or bedridden.

B) The Arabic translation of Modified Stanford Health Assessment Questionnaire (HAQ) (Kirwan & Reebach, 1986):

It is based on the original Health Assessment Questionnaire by Fries et al. (1980). The HAQ is designed to assess the patient's functional ability. It is composed of eight questions; which are about: dressing, arising from bed, eating (or drinking), walking, self hygiene, reaching objects, grip and outside activities.

For each of these questions, patients were asked to record the amount of difficulty they may have. The possible responses for the questions are:
- Without any difficulty = 0
- With some difficulty = 1
- With much difficulty = 2
- Unable to do = 3

The index is calculated by adding the scores of the questions and dividing by the number of questions answered. This gives a score in the 0 to 3 range.

Assessment of radiological severity:

The radiological severity of rheumatoid arthritis patients was assessed using Larsen method (Larsen et al., 1977) for plain X-rays of both hands and wrists.

**Larsen's scores are**:
- Grade 0: normal
- **Grade 1**: slight abnormality with one or more minor lesions (periarticular soft tissue swelling, periarticular osteoporosis and/or slight joint space narrowing).
- **Grade 2**: definite early abnormality with erosion and joint space narrowing corresponding to the standards.
- **Grade 3**: medium destructive abnormality, erosion and joint space narrowing corresponding to the standards. Erosion is obligatory in all joints.
- **Grade 4**: severe destructive abnormality, erosion and joint space narrowing corresponding to the standards.
- **Grade 5**: mutilating abnormality. The original articular surfaces have disappeared.

The Larsen's index score was applied to interphalangeal joints, metacarpophalangeal joints and wrist joint of both hands (the Larsen indices of wrist joints were multiplied by five). Then the score was summed up giving a maximum score of 150 when all joints of both hands are fully destroyed.

**Laboratory investigations**:

Complete blood picture; erythrocyte sedimentation rate (ESR) with Westergren method, rheumatoid factor with latex
technique; C-reactive protein (CRP) with latex method; lipid profile including total cholesterol, low-density lipoprotein cholesterol (LDL-c), high-density lipoprotein cholesterol (HDL-c) and triglycerides.

Doppler ultrasound examination:
Carotid ultrasound was performed at the Radiodiagnosis Department of Benha University Hospitals using TOSHIBA 526 DEVICE equipped with 7.5 MHz imaging transducer. All the patients were examined in the supine position with slight hyperextension of the neck. Right and left carotid arteries, carotid bulb, carotid bifurcation and the proximal portions of internal and external carotid arteries were imaged in multiple projections and then focused on the interfaces required to measure the intima media thickness (IMT) (Roman et al., 1995). Values were then averaged to produce an overall measure of IMT.

Statistical Analysis:
This was done using SPSS statistical package for social science. Data were parametric by using Kolmogrov-Smirrov test. Qualitative data were presented in the form of number and percentage. The qualitative data were presented in the form of mean, standard deviation and range. Students- t-test was used to compare two groups, but one way ANOVA (F test) was used to compare more than two groups. Multiple regression analysis was done to determine the best model for prediction. Pearson correlation coefficient was done to study the relation between variables. Values of \( p<0.05 \) were considered significant, \( p\)-value>0.05 insignificant.

RESULTS

Characteristics of the studied subjects:
Clinical characteristics (risk factors for coronary artery disease) of the study participants are summarized in table (1). There was no significant difference in these variables \( (p>0.05) \) between RA patients and controls except for CRP (table 1). RA- related clinical and radiological variables are displayed in table (2).

Table (3) shows common carotid artery (CCA) intima-media wall thickness (IMT) in cases and controls. The mean level of IMT of the CCA showed a statistically highly significant increase \( (p<0.001) \) in RA patients as compared to controls. Table (4) shows IMT of the CCA in RA patients graded according to disease activity (DAG) and functional capacity grading. Using the ANOVA test, significant differences were found \( (p<0.05) \) between the level of IMT of the CCA and DAG and functional capacity grading in RA patients. Table (5) shows IMT of the CCA in RA patients in relation to their drug treatment. The IMT of the CCA not significantly different \( (p>0.05) \) between RA patients who were taking and those who were not taking NSAIDs, corticosteroids, and/or DMARDs.

Table (6) shows correlations with IMT of the CCA in RA patients. There were statistically significant correlations \( (p<0.05) \) between IMT of the CCA in RA and disease related variables; duration of morning stiffness, articular index, grip strength, ESR, hemoglobin level, pain severity and rheumatoid factor. Highly statistically significant correlations \( (p<0.001) \) were found in relation to age, disease duration, spread severity index, Larsen score, HAQ and CRP. Insignificant correlations \( (p>0.05) \) were found between IMT of the CCA in RA and sex, B.M.I blood pressure and lipid profile.
### Table (2): Clinical and radiological data of RA patients.

<table>
<thead>
<tr>
<th>Data</th>
<th>Range</th>
<th>Mean ± SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Disease duration (years)</td>
<td>3 – 22</td>
<td>10.7 ± 3.6</td>
</tr>
<tr>
<td>Duration of morning stiffness (min)</td>
<td>8 – 150</td>
<td>48.8 ± 32.2</td>
</tr>
<tr>
<td>ESR (mm/ 1st h)</td>
<td>16 – 110</td>
<td>44.46 ± 20.22</td>
</tr>
<tr>
<td>Grip strength (mmHg)</td>
<td>10 – 210</td>
<td>64.8 ± 44.6</td>
</tr>
<tr>
<td>Pain severity (cm)</td>
<td>2.2 – 10</td>
<td>4.9 ± 1.4</td>
</tr>
<tr>
<td>Articular index</td>
<td>2 – 27</td>
<td>16.08 ± 6.6</td>
</tr>
<tr>
<td>Hb % (gm/dl)</td>
<td>8 – 15</td>
<td>10.22 ± 4.2</td>
</tr>
<tr>
<td>Health assessment questionnaire (HAQ)</td>
<td>0.4 – 3</td>
<td>1.8 ± 0.64</td>
</tr>
<tr>
<td>Spread severity index</td>
<td>6 – 34</td>
<td>16.4 ± 5.8</td>
</tr>
<tr>
<td>Larsen score</td>
<td>0 – 150</td>
<td>74.8 ± 32.2</td>
</tr>
<tr>
<td>Rheumatoid factor % positive</td>
<td></td>
<td>77%</td>
</tr>
</tbody>
</table>

### Table (3): Carotid intima-media thickness in RA patients and in healthy controls.

<table>
<thead>
<tr>
<th>Carotid IMT(mm.)</th>
<th>RA Patients (No. = 40)</th>
<th>Controls (No. = 40)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Range</td>
<td>0.5 to 1.44</td>
<td>0.4 to 0.9</td>
</tr>
<tr>
<td>Mean</td>
<td>0.84</td>
<td>0.62</td>
</tr>
<tr>
<td>± SD</td>
<td>0.08</td>
<td>0.04</td>
</tr>
<tr>
<td>t</td>
<td>12.61</td>
<td></td>
</tr>
</tbody>
</table>

**p-value: < 0.001 Highly significant**
Table (5): Comparison between carotid IMT in RA patients in relation to their drug treatment.

<table>
<thead>
<tr>
<th>Drug treatment</th>
<th>No.</th>
<th>Carotid IMT (mm) [mean ± SD]</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>NSAID</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not treated</td>
<td>12</td>
<td>0.86 ± 0.16</td>
<td>F = 0.84</td>
</tr>
<tr>
<td>Selective</td>
<td>9</td>
<td>0.82 ± 0.14</td>
<td>p = 0.51</td>
</tr>
<tr>
<td>Non-selective</td>
<td>19</td>
<td>0.83 ± 0.15</td>
<td>[NS]</td>
</tr>
<tr>
<td>Steroids</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not treated &lt; 10mg.</td>
<td>22</td>
<td>0.82 ± 0.15</td>
<td>F = 0.46</td>
</tr>
<tr>
<td>&gt; 10mg.</td>
<td>14</td>
<td>0.86 ± 0.12</td>
<td>p = 0.63</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>0.87 ± 0.14</td>
<td>[NS]</td>
</tr>
<tr>
<td>DMARDs</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Methotrexate</td>
<td>32</td>
<td>0.83 ± 0.15</td>
<td>t = 0.79</td>
</tr>
<tr>
<td>Other. DMARDs</td>
<td>8</td>
<td>0.86 ± 0.17</td>
<td>p = 0.56</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>[NS]</td>
</tr>
</tbody>
</table>

Table (7) shows multiple regression analysis which was done to determine the best model for predicting atherosclerosis in RA patients. Among the wide range of variables considered in the present study, higher CRP, spread severity index, Larsen score and higher functional disability respectively, were the most variables predicting atherosclerosis among RA patients.

Table (6): Correlations with carotid IMT in RA patients.

<table>
<thead>
<tr>
<th>Disease related aspects</th>
<th>r</th>
<th>p- value</th>
<th>Risk factors</th>
<th>r</th>
<th>p- value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Disease duration</td>
<td>0.84</td>
<td>&lt; 0.001</td>
<td>(HS) Age</td>
<td>0.79</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Morning stiffness</td>
<td>0.62</td>
<td>&lt; 0.01</td>
<td>(S) Sex</td>
<td>0.31</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Articular index</td>
<td>0.64</td>
<td>&lt; 0.01</td>
<td>(S) Body mass index</td>
<td>0.41</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Grip strength</td>
<td>-0.63</td>
<td>&lt; 0.01</td>
<td>(S) Diastolic blood pressure</td>
<td>0.32</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>ESR</td>
<td>0.60</td>
<td>&lt; 0.01</td>
<td>(S) Systolic blood pressure</td>
<td>0.37</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Pain severity</td>
<td>0.61</td>
<td>&lt; 0.01</td>
<td>(S) Total cholesterol</td>
<td>0.35</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Hemoglobin</td>
<td>-0.52</td>
<td>&lt; 0.05</td>
<td>(S) LDL-c</td>
<td>0.36</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Rheumatoid factor</td>
<td>0.60</td>
<td>&lt; 0.01</td>
<td>(S) HDL-c</td>
<td>0.35</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>HAQ</td>
<td>0.81</td>
<td>&lt; 0.001</td>
<td>(HS) Triglycerides</td>
<td>0.32</td>
<td>&gt; 0.05</td>
</tr>
<tr>
<td>Spread severity index</td>
<td>0.83</td>
<td>&lt; 0.001</td>
<td>(HS) CRP</td>
<td>0.84</td>
<td>&lt; 0.001</td>
</tr>
<tr>
<td>Larsen score</td>
<td>0.82</td>
<td>&lt; 0.001</td>
<td>(HS)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

S = Significant (p<0.05)  HS = Highly significant (p<0.001)  NS = Non significant (p>0.05)
**DISCUSSION**

It is well recognized that rheumatoid arthritis (RA) causes significant morbidity as a result of synovial inflammation, joint destruction, and associated disability (Van Doornum et al., 2003). In addition to these articular manifestations of RA, there is growing recognition of an excess mortality, which is predominantly due to increased coronary artery atherosclerosis (Turesson & Matteson, 2007).

Approximately 50% of the atherosclerotic coronary artery disease in the community occurs in the absence of traditional risk factors, such as smoking, hypertension, diabetes mellitus, and hypercholesterolemia (Schroecksnadel et al., 2003). In recent years, it has become apparent that in addition to the traditional risk factors for atherosclerosis, this condition is associated with infectious, inflammatory, and autoimmune factors (Abou-Raya & Abou-Raya, 2006).

In this study, we planned to measure the extent of subclinical atherosclerosis in RA patients by measuring the intima-media thickness (IMT) of the common carotid artery (CCA) and to evaluate the factors associated with arterial wall

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### Table 1: Regression Analysis of the Variables

<table>
<thead>
<tr>
<th>Variable</th>
<th>Regression</th>
<th>t</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>CRP</td>
<td>0.996</td>
<td>5.42</td>
<td>0.001</td>
</tr>
<tr>
<td>Spread severity index</td>
<td>0.993</td>
<td>3.11</td>
<td>0.008</td>
</tr>
<tr>
<td>Larsen score</td>
<td>0.988</td>
<td>2.83</td>
<td>0.039</td>
</tr>
<tr>
<td>HAQ</td>
<td>0.983</td>
<td>2.48</td>
<td>0.049</td>
</tr>
</tbody>
</table>

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**Fig. (1):** Shows diffusely increased IMT measuring:

1.2 mm at Rt common carotid bulb.   1.2 mm at Rt common carotid artery.
thickness in RA patients. We used an accurate and reliable imaging technique, high-resolution B-mode ultrasound, for the diagnosis of subclinical atherosclerosis.

Our results showed that the IMT of the common carotid artery was significantly higher in RA patients than in healthy controls (p<0.001). The healthy subjects were comparable with the RA patients with regards to the risk factors for atherosclerosis, including age, sex, menopause status, body mass index (BMI), blood pressure, and serum lipid levels. This finding was confirmed by the work done by Soubrier & Dougados (2006) who found that RA patients had greater IMT of the common carotid artery than did appropriately matched controls. Kumeda et al. (2002) stated that increased IMT may pre-date the clinical manifestations of atherosclerosis by many years in subjects at risk of atherosclerosis. The common carotid artery IMT is strongly correlated with the presence of coronary artery disease (Del Rincon et al., 2004).

There are several possible explanations for the observed association between arterial wall thickness and RA. The first is a possible relationship between atherosclerosis and chronic inflammation due to RA (Wang & Feng, 2004).

There is growing evidence that inflammation plays a role in the initiation and progression of atherosclerosis (Pham et al., 2006 and Hurlimann et al., 2004) reported that many similarities have emerged between the paradigm of inflammation in the pathogenesis of atherosclerosis and the well-established mechanisms of inflammation in the pathogenesis of RA. Hence inflammation in RA is not only confined to the joints but also present in the vessel wall.

In our study, we found significant positive correlations between morning stiffness, articular index, pain severity, ESR and carotid IMT. Also we found significant negative correlations between grip strength, hemoglobin level and carotid IMT. These significant correlations between the common carotid IMT and disease activity in RA demonstrated in our study, were in agreement with the results of Van Doornum et al. (2003) who reported that the positive relationship between disease activity and severity of arterial stiffness supports the notion that chronic inflammation plays a role in RA-associated atherosclerosis. Also Van Doornum et al. (2002) stated that systemic inflammation associated with RA may play a significant role in atherosclerosis and anti-inflammatory therapies may be used to treat atherosclerotic disease in the future. Lastly, Jonsson & Dahlqvist (2004) advised dampening of the inflammatory activity as a favorable impact on the progression of atherosclerosis in RA.

The second possible explanation for the advanced arterial wall changes in RA patients is the impaired physical activity. We found a highly significant positive correlation (p<0.001) between carotid IMT and functional disability score. This result was similar to those from Alkaabi et al. (2003), who showed that RA patients with evidence of atherosclerosis had a higher HAQ score than those without. Also, Turesson & Matteson (2007) suggested that decreased physical activity, impaired mobility and sedentary life may be factors that contribute to the development of atherosclerosis in RA.

The third possible explanation is that the arterial wall changes progressed in the RA patients as bone destruction progressed, as evidenced by a highly significant positive association between the Larsen score and the IMT of the common carotid artery. Uyama et al. (1997) stated that, there was evidence that atherosclerosis progresses significantly faster in patients with enhanced bone destruction than in those with less bone destruction. The increase in common

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carotid artery IMT in RA patients with high Larsen scores can be explained by an increase in calcium mobilization from bone due to enhancement of bone destruction, leading to enhanced development of atherosclerosis (Hak et al., 2000).

Whether treatment for RA affects the arterial system is an important issue. Some epidemiological studies have indicated that treatment with CoX-2- specific inhibitors (Mukherjee et al., 2001), corticosteroid (Kim et al., 2003) and methotrexate (Landewe et al., 2000), is associated with an increased prevalence of cardiovascular disease. In the present study, common carotid artery IMT was not significantly different between RA patients who were taking and those who were not taking NSAIDs, corticosteroids, or methotrexate. Hennan et al. (2001) stated that selective CoX-2- inhibitors may be prothrombotic, and the need for further research on the clinical importance of this has been highlighted recently (Niederberger et al., 2004). On the other hand, Van Doornum et al. (2002) considered methotrexate as a cardio protective drug as methotrexate reduces the inflammatory activity in RA and this can balance the atherothrombogenic effects of the drug and even turns out to have a beneficial net effect on the progress of cardiovascular disease. Further research is needed for a more definitive evaluation of the role of methotrexate in CV morbidity and mortality in RA (Choi et al., 2002).

The last possible explanation for the advanced arterial wall thickness in RA patients was recently reported by Gonzalez-Gay et al. (2004), as a genetic factor. HLA-DRB1 shared epitope alleles, in particular HLA-DRB1*0404, seems to be implicated in the development of atherosclerosis in RA patients. In our study, many of variables were significantly correlated with IMT of CCA in RA patients so multiple regression analysis was done to determine the best model for predicting atherosclerosis in RA patients. Among the wide range of variables considered in the present study, higher CRP, spread severity index, Larsen score and higher functional disability respectively, were the most predicting atherosclerosis among RA patients. The first best model for prediction of atherosclerosis in our study was higher CRP.

Recent studies indicated that, among various markers of inflammation, the CRP level was a particularly powerful predictor of cardiovascular disease independently of serum lipid levels (Abou-Rayya & Abou-Rayya, 2006). CRP is also hypothesized to be causally involved in the pathophysiology of atherosclerosis and its complications through its localization in the atheromatous plaques and stimulation of macrophages to produce tissue factor, an important procoagulant found in atheromatous plaques (Del Rincon et al., 2003). Lastly, Pham et al. (2006) described the "statin" group of drugs that lower CRP as well as cholesterol as a means of primary and secondary prevention of coronary artery disease in RA.

**Conclusions:**

RA patients exhibited greater thickness of the common carotid artery than healthy controls. So RA patients have an ultrasonic marker of early atherosclerosis. The age, disease duration, disease activity and severity, decreased physical activity, but not therapeutic variables, were associated with the increased arterial wall thickness.

**Recommendations:**

Early recognition of these findings before the occurrence of atherosclerotic complications may reduce cardiovascular events in RA patients. Because of the non-invasive character and easy applicability, RA patients at risk of atherosclerosis should be examined by high resolution ultrasonography for identification of early
stage atherosclerosis.

Dampening of the inflammatory activity has a favorable impact on the progression of atherosclerosis in RA.

Treatment strategies in RA should not only aim at relieving symptoms and inhibiting joint destruction but also have a beneficial effect on the vasculature to reduce cardiovascular events. Accordingly, statin therapy represents possible targets for prevention of atherosclerosis in RA.

REFERENCES


The objective: To estimate the risk of coronary heart disease in rheumatoid arthritis patients compared to the general population.

The methods: A cross-sectional study of 40 patients with rheumatoid arthritis and 40 age-sex matched controls.

The results: The risk of coronary heart disease in rheumatoid arthritis patients is significantly higher compared to the general population.

The conclusion: Rheumatoid arthritis patients have an increased risk of coronary heart disease.