The aims of this study were to investigate the relationship between cir-
que (OR 6.22 [95% CI 1.08 – 35.59]) at baseline and
1.0003], adjusted for carriage of the minor
alleles of rs4588, in a multiple-adjusted model (p<0.05).

**Conclusions:** To our knowledge, this is the first time that smoking is shown to be
a negative predictor for clinical response to pred in RA patients. Cessation of
smoking needs to be encouraged in patients initiating bDMARDS, MTX and pred and
in those already on these drugs.

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**THU0121**

**MORTALITY RATE AND PREVALENCE OF CARDIOVASCULAR DISEASE IN RHEUMATOID ARTHRITIS: 5-YEAR KARRA PROSPECTIVE STUDY**

N.R. Kim, J.W. Kang, J.S. Eun, J.H. Kim, J.Y. Kang, J.S. Seo, G.B. Bae, S.J. Lee,
E.J. Nam, Y.M. Kang. Division of Rheumatology, Department of Internal Medicine,
Kyungpook National University School of Medicine, Daegu, Korea, Republic of

**Background:** Rheumatoid arthritis (RA), which is an autoimmune chronic arthri-
tis, leads to elevated rates of disability and mortality.

The main causes of mortality identified among RA patients are increased inciden-
ces of cardiovascular (CV) disease, which accounts for one-third to one-half of the
premature deaths, infection and cancer.

In our previous study, we identified that cumulative inflammatory burden contrib-
utes to the development of carotid atherosclerosis through a synergistic interac-
tion with conventional CV risk factors in patients with RA.

During the 2 years follow-up period, the mortality rate was 2.4% (10/412), and the
main causes of death were infection (4/10) and CV disease (3/10).

**Objectives:** To investigate the incidences of mortality and CV disease in patients
with RA in the 5 year Kyungpook National University Hospital Atherosclerosis
Risk in Rheumatoid Arthritis (KARRA) prospective study.

**Methods:** A total of 372 patients with RA and 162 healthy controls were followed
up for 5 years or until deaths in a prospective KARRA cohort study (412 patients
and 221 controls at baseline).

To detect the presence and progression of carotid atherosclerosis, we performed
carotid ultrasound at baseline and 5 year.

We analysed the incidence of CVD, conventional CV risk factors, RA disease
activity and severity markers, medication histories, mortality rate, and causes of
death.

**Results:** During 5 year follow-up period, the mortality rate was 10.7% (44/412) in
RA patients and 1.4% (3/221) in healthy controls (p<0.001), while the incidence of
CVD were 11.4% (47/412) in RA patients and 0.9% (2/221) in healthy controls
(p<0.001).

Among CVD in RA patients, cerebrovascular accident (CVA) and cardiovascular
event (CVE) were 17 (36.2%) and 30 (63.8%) events, respectively.

Major causes of death included infection (21/44, 47.7%), CVD (12/44, 27.3%),
and others (11/44, 25%).

The mean age, presence and number of carotid plaques, functional class, modified
Korean version of the HAQ (mHAQ), tender joint count (TJC), swollen joint
count (SJC), ESR and CRP, and conventional CV risk factors at baseline and
cumulative ESR (ESR area under the curve), DAS28-ESR and DAS28-CRP at
year 5 were significantly associated with mortality in RA patients.

Multivariate logistic regression analysis showed that the presence of carotid pla-
que (OR 6.22 [95% CI 1.08 – 35.59]; p=0.031), mHAQ (OR 1.04 [95% CI 1.01 –
1.12; p=0.014], and ESR (OR 1.09 [95% CI 1.03 – 1.16; p<0.001]) at baseline and
cumulative ESR (ESR area under the curve) (OR 1.07 [95% CI 1.01 –1.13;
p=0.048]) and DAS28-ESR (OR 1.55 [95% CI 1.08 –2.21; p<0.016]) at year 5 were
independent risk factors for mortality of RA patients.

**Conclusions:** During the follow-up period of 5 years, the mortality rate and preva-
ience of CV disease were significantly increased in RA patients, compared to the
controls. Furthermore, main causes of death were infectious disease and CV disease.
Furthermore the risk factor for CVD and mortality is carotid plaque which is
determined by disease activity and CV risk factors.

**Disclosure of Interest:** None declared

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**THU0122**

**IMAGING CHARACTERISATION OF REMISSION IN RHEUMATOID ARTHRITIS**

N. Purkayastha1, W.S. Tari1, A. Mahto1, M. Lewis1, G. Liljo1, A. Nerviani1,
V. Rocher1, F. Humby1, C. Pitzalis2, S. Kelly2. 1Experimental Medicine and
Rheumatology, William Harvey Research Institute, 2Rheumatology, Bart’s Health
NHS Trust, London, UK

**Background:** Remission in rheumatoid arthritis (RA) is now achievable in a sig-
nificant proportion of patients using a combination of a treat to target strategy
and biologic therapy. A number of clinical assessment tools exist for assessing remis-
sion. Several reports have shown that ultrasound (US) may have a role in better
characterising this group of patients suggesting that subclinical synovitis
increases the risk of erosive disease and flares in patients in DAS28 remission.1-3

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**REFERENCES:**


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Västerbotten Intervention Program, the Northern Sweden MONICA Study,
and the mammary screening program of Västerbotten. Staff of the Department of
Biobank Research, Umeå University, aided in acquisition of samples and data.
These observations hint at a stratum of patients where remission is incomplete and may require a more tailored approach to therapy.

Objectives: In an early RA cohort we examine the relationship between US imaging and histological synovitis in the context of clinical remission and examine the predictive value of US to confirm synovial inflammation.

Methods: A prospective, observational study of 122 DMARD naïve, early RA patients classified according to the 1987 ACR criteria, with a maximum disease duration of 12 months (MRC PEAC study www.peacr.mds.qmul.ac.uk). One hundred and three synovial biopsies were analysed at baseline and 75 at 6 months, with 85 paired US 12 joint scores (US 12: 10 metacarpophalangeal joints (MCPJ) and 2 wrists). US images were analysed using a using a semi-quantitative score for synovial thickness (ST) and power Doppler (PD). Synovial inflammation was assessed using the Krenn synovitis scoring system. Fisher exact statistical test and Spearman’s rank was used to determine the association and correlations.

Results: Demographics of this cohort are listed in table 1. There was a good correlation between US ST and PD scores with the Krenn histology score at the level of the single biopsied joint (ST: r=0.47, p<0.001, PD: r=0.5, p<0.001). An association continued to be demonstrated when extending the US data set to 12 joints (ST r=0.27 p<0.01, PD r=0.28 p<0.01). Twenty-two patients with paired histology and ultrasound data were in DAS28 remission at 6 months and were eligible for analysis. A significant association between low PD (<36) signal (but not ST) and low Krenn score (<4) was demonstrated (Fisher exact test p<0.03) with a predictive value of 90%. This reduced to 80% in patients not in DAS remission at 6 months (n=42). Lastly, a clear relationship was noted between patients with US PD score recorded after 6 months of DMARD therapy (n=62, flares n=19). No subsequent flares were recorded during the course of the follow up period of 6 months with a low US PD score (p<0.002, negative predictive value 76%) and a high US PD score had a 86% positive predictive value for disease flare within this time course.

Conclusions: This study demonstrates that there is considerable validity in the use of US to assess disease activity, which reflects histological synovitis in patients in low disease activity states and remission. Ultrasound imaging may demonstrate a distinct clinical and histological remission cohort of patients and may be a useful predictive tool in terms of predicting subsequent clinical disease activity.

REFERENCES:

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