documented over time. Determinants of health care resources utilisation and of cost of illness are being investigated.

A better knowledge of predictors of RA impact, if modifiable by a medical or a social intervention, helps decrease the severity of the condition, decrease the frequency of deleterious consequences, increase patients quality of life and reduce costs to society. An investigation of societal and economic aspects of the disease impact is still needed over the long term. Condition for impact prediction may well change with the RA condition recently entering a new therapeutic era.

Cardiovascular complications in R. D. – Thursday 14 June, 16.00-17.30/Congress Hall

SP0042 ENDOTHELIAL ACTIVATION IN INFLAMMATION AND ATHEROSCLEROSIS

¹D Haskard, ²Dorian O Haskard. ¹National Heart and Lung Institute, Imperial College School of Medicine, London, UK; ²BHF Cardiovascular Medicine Unit, Imperial College School of Medicine, Hammersmith Hospital, London W12 0NN, UK

10.1136/annrheumdis-2001.3

Endothelial activation is an integral component of inflammatory rheumatic diseases, and also of atherosclerosis. Leukocytes emigrate from the blood into inflamed tissues through a series of adhesion events ("the adhesion cascade"), each of which is dependent upon the state of endothelial cell activation. Initial rolling of neutrophils on vascular endothelium is mediated by transient interactions between selectins (L-selectin on leukocytes, E-selectin on cytokine-activated endothelial cells (EC) and P-selectin on both activated EC and activated platelets) and carbohydrate-bearing counter-structures on the opposing cell. Whilst rolling, leukocytes become exposed to activating signals (such as chemokines), resulting in an upregulation of the capacity of b2 integrins (eg LFA-1, Mac-1) to bind ligands (eg ICAM-1, -2) on EC. This integrin-mediated secondary adhesion results in leukocyte arrest and is followed by their transmigration into the tissues. In the case of mononuclear cells, adhesion of a4 integrins to EC ligands (eg VCAM-1) may contribute to both the rolling and firm adhesion stages of their interaction with EC. The talk will focus on comparisons between inflammatory rheumatic diseases and atherosclerosis, and will discuss recent technical advances that have allowed an improved understanding of the patterns of endothelial activation and adhesion molecule expression that occur in acute and chronic inflammatory settings.

SP0043 EPIDEMIOLOGY OF CARDIOVASCULAR COMPLICATIONS IN SYSTEMIC LUPUS ERYTHEMATOSUS

M Petri. *Medicine, Division of Rheumatology, Johns Hopkins University, School of Medicine, Baltimore, USA*

10.1136/annrheumdis-2001.4

Cardiovascular complications are increasingly recognised as a major cause of morbidity and mortality in SLE. The incidence of myocardial infarction is elevated 8 to 50-fold compared to age and gender matched controls. Traditional cardiovascular risk factors are increased in SLE, and are associated with both

preclinical and clinical atherosclerosis. However, even after adjustment for traditional risk factors, a large difference in the frequency of cardiovascular disease exists between SLE patients and controls. In the general population, atherosclerosis is now recognised as a chronic inflammatory condition, with elevations of C-reactive protein and IL-6. TNF has a role in the pathogenesis of congestive heart failure. These and other inflammatory cytokines likely play a role in SLE. Furthermore, SLE patients are hypercoagulable secondary to antiphospholipid antibodies and other factors. Thus, the pathogenesis of cardiovascular complications in SLE is likely multifactorial, with both SLE and traditional risk factors playing a role.

Abstract SP0043 Table 1

Variable	OR	95% CI	p-value
Smoking	1.87	1.1–3.1	0.02
Renal insuff.	2.21	1.1–4.5	0.04
Obesity	2.13	1.3–3.6	0.005
Hypertension	4.19	2.4–7.3	0.0001
Diabetes	3.44	1.6–7.4	0.002
Cholesterol	1.79	1.04–3.1	0.045
Aspirin Use	0.54	0.3–0.96	0.04
Premarin	2.48	1.4–4.5	0.004
Continuous Variables			P-value
Weight			0.0005
BP-systolic			0.0001
BP-diastolic			0.019
Age			0.0001
Yrs. Duration of Lupus			0.04
Highest Prednisone Dose			0.04

SP0044 THE EPIDEMIOLOGY OF VASCULAR DISEASE IN RHEUMATOID ARTHRITIS

¹S Wållberg-Jonsson, ²ML Öhman, ¹S Rantapää-Dahlqvist. ¹Department of Rheumatology, University Hospital; ²Department of Statistics, University of Umeå, Umeå, Sweden

10.1136/annrheumdis-2001.5

Cardiovascular disease (CVD) is the most common cause of death in RA as in the general population. According to most epidemiological reports, death due to CVD is more frequent in RA than in the general population with standardised mortality ratios (SMR:s) ranging from 1.3–2.4. Several of the studies are community based. The largest increase, SMR 3.64, was reported in women aged 15–49. Some studies have reported increased death due to ischaemic heart disease (IHD) while reports on cerebrovascular disease are contradictory. In 606 patients with RA followed-up after 16 years we found an SMR for CVD of 1.57 and for IHD 1.54. Cardiovascular morbidity in RA is less investigated. However, current epidemiological data implicate increased prevalence of cardiovascular (CV) events and increased IHD morbidity in RA. These findings are supported by physiological studies. In 39 RA patients with medium-term disease, we found indications of increased atherosclerosis as measured by intima media thickness (IMT) of the carotid artery, presence of plaques and aortic cusp sclerosis compared to controls. Established cardiovascular risk factors do not contribute prominently to CVD in RA. Recent studies have reported increased prevalence of hypertension and higher blood pressure in RA compared to controls. Hypertension could also predict CV events and death. The