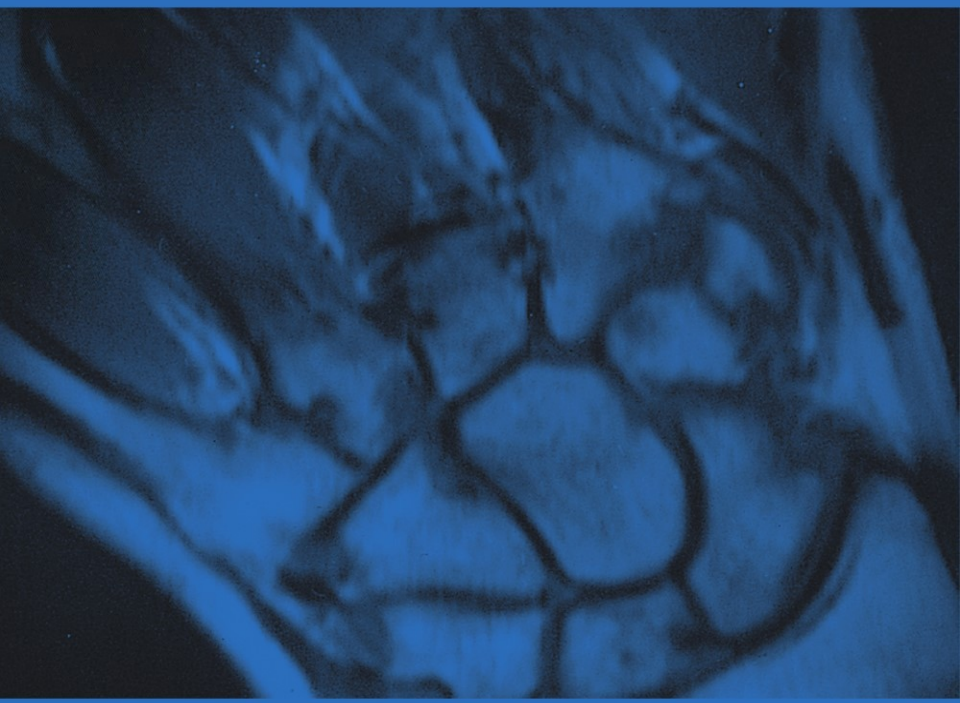
Pocket Reference to Early Rheumatoid Arthritis

Paul Emery



Springer Healthcare

Pocket Reference to Early Rheumatoid Arthritis

Pocket Reference to Early Rheumatoid Arthritis

Paul Emery
arc Professor of Rheumatology
Academic Unit of Musculoskeletal Disease, University of Leeds,
Leeds Teaching Hospitals Trust, UK

Springer Healthcare

Published by Springer Healthcare, 236 Gray's Inn Road, London, WC1X 8HB, UK
www.springerhealthcare.com

British Library Cataloguing-in-Publication Data.

A catalogue record for this book is available from the British Library.

ISBN 978 1 85873 448 4

Commissioning editor: Dinah Alam
Project editors: Hannah Cole and Alison Whitehouse
Designers: Joe Harvey and Taymoor Fouladi
Production: Marina Maher
Printed in Great Britain by Latimer Trend & Company Ltd.

Contents

Author biography

Paul Emery is Arthritis Research Campaign Professor of Rheumatology and Head of the Academic Unit of Musculoskeletal Medicine at the University of Leeds, as well as Clinical Director (Rheumatology) at the Leeds Teaching Hospitals Trust in the United Kingdom. He graduated in medicine from the University of Cambridge and completed specialist accreditation in internal medicine and rheumatology. He completed his thesis on the immunopathology of rheumatoid arthritis at Guy's Hospital, and then served as Head of Rheumatology at the Walter and Eliza Hall Institute in Melbourne. He was senior lecturer in rheumatology at the University of Birmingham, UK from 1987–1995 until his present appointment.

Professor Emery has served as a member of several education committees including the Senior Advisory Committees of the Royal College of Physicians, the MRCP Part 1 Board. He is currently the President of EULAR, a past member of the Scientific Committee and Chairs the MRI imaging group. He has served on the editorial boards of several journals, including *Rheumatology, Arthritis and Rheumatism, Annals of the Rheumatic Diseases, Clinical and Experimental Rheumatology, Clinical Rheumatology* and *Modern Rheumatology (Japanese Rheumatology Association Journal).*

He is a recipient of the Roche Biennial Award of Clinical Rheumatology, the Rheumatology Hospital Doctor of the Year award 1999 and the EULAR prize 2002 for outstanding contribution to Rheumatology research. Professor Emery's research interests centre around the immunopathogenesis and immunotherapy of rheumatoid arthritis and connective tissue diseases. He has a special interest in the factors leading to persistent inflammation. He has published over 650 peer-reviewed articles in this area.

Chapter 1

Rheumatoid arthritis: an overview

Rheumatoid arthritis is a chronic systemic inflammatory arthritis of auto-immune origin that affects primarily the synovial joints, usually in a symmetrical pattern. It is the most common and most serious of the inflammatory arthritides, and it dominates clinical rheumatological practice (Silman 2002). Current evidence shows that prompt diagnosis and early instigation of definitive disease-modifying treatment can delay or avoid progression and enable patients to retain function that would otherwise be lost in this progressive and frequently disabling disease.

Incidence

Rheumatoid arthritis is estimated to affect between about 0.5 and 2% of the population worldwide (Alamanos and Drosos 2005, Emery 2006, Sommer et al. 2005).

Broadly speaking the disease is equally common worldwide, although there is some evidence of variation, with lower prevalence rates in southern Europe than in northern Europe and North America (Alamanos et al. 2006). There may also be lower rates in developing countries than in the developed world, a finding that has been attributed to environmental effects of urbanisation, although the precise causes are not known (Kalla and Tikly 2003). Some studies have reported a general trend for a decrease in frequency (Doran et al. 2002), more specifically in countries with high rates of disease (Alamanos et al. 2006) although such a trend is far from certain (Silman 2002). However, the small number of studies for most areas of the world and the lack of incidence studies for developing countries means that knowledge of the global epidemiology of rheumatoid arthritis is limited (Alamanos et al. 2006). UK data are summarised in Figure 1.1.

Rheumatoid arthritis in the UK

- 24 new cases of rheumatoid arthritis per 100,000 of the population per year [a]
- > 580,000 people have rheumatoid arthritis [b]
- Women 2–3 times more likely to be affected than men [c]

Figure 1.1 Rheumatoid arthritis in the UK. [a]Wiles et al.1999, [b]National Audit Office 2009, [c]Symmons et al. 1994.

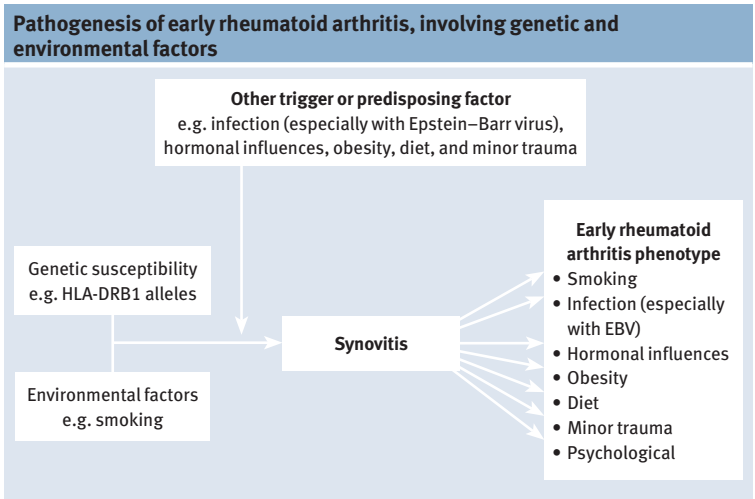


Figure 1.2 Pathogenesis of early rheumatoid arthritis, involving genetic and environmental factors. EBV, Epstein–Barr virus. Adapted from Pratt et al. 2009, with data from Pratt et al. 2009, Lee et al. 2007, Balandraud et al. 2004.

Aetiology and pathophysiology

The precise aetiology of rheumatoid arthritis is not known but it is complex and multifactorial, and involves both genetic and environmental components (Lee et al. 2007) (Figure 1.2).

Genetic factors

The disease is known to cluster in families, and those with a first-degree relation with rheumatoid arthritis are between two and 10 times more likely to have the disease than the general population (John et al. 1998). A genetic basis for this familial preponderance is confirmed by the observation that the concordance for rheumatoid arthritis in monozygotic twins is about 15%, up to five times the concordance in dizygotic twins (Pratt et al. 2009). Certain alleles at the human leukocyte antigen (HLA)-*DRB1* locus are known to be associated with susceptibility to rheumatoid arthritis and with the presence of autoantibodies, notably rheumatoid factor and antibodies against cyclic citrullinated peptide (anti-CCP antibodies or ACPA) (Gregersen et al. 1987). For example, *HLA-DRB1*401* and *DRB1*0404* are associated with radiographic erosions (Weyand et al. 1992).

Other genetic associations have also been identified (Pratt et al. 2009) (Figure 1.3). In Europeans, about 50% of genetic susceptibility to rheumatoid arthritis is contributed by two genes: *HLA-DRB1* and *PTPNN2 (*Pratt et al. 2009).