Which patients stop working because of RA?

The paper by Young et al is a useful contribution to the question of work disability related to rheumatoid arthritis. However, one of the areas in which intervention is theoretically possible to reduce disability—namely, that of work place intervention—was not discussed. In the study by Young et al the type of work performed by the study cohort are grouped into four categories—namely, manual, semi-manual, semi-sedentary, and mainly sedentary. Presumably, the patients were allocated to a group based on job title using the British classification of occupations and coding index.

It is well known to occupational physicians and others taking occupational histories that a job title does not adequately reflect the true nature of work. In addition, problems likely specifically to affect patients with rheumatoid arthritis are not usually classified in a subjective ordinal scale such as that used in the study. Examples of such problems might be fine repetitive movements of the hand or work starting times.

To gain an accurate insight into work factors affecting work disability, a study is required in which information on the nature of the work tasks is obtained. Ideally this should be gathered prospectively by direct analysis of the work place by trained observers. Tools have been developed that can assist with this type of data gathering.

This would reduce recall and misclassification bias of previous studies. Once this has proved resistant to a variety of treatments. Mab (Revellex) in the treatment of adult systemic lupus erythematosus (SLE) has proved resistant to a wide variety of agents over a 10 year period and who has had several, very serious complications of this condition, including the development of cardiac amyloidosis.

The patient, a 35 year old nursing sister, was initially diagnosed with AOSD at the age of 23 and was treated with non-steroidal anti-inflammatory drugs, salicylates, methotrexate, antimalarial drugs (chloroquine), and p-aminosalicylic acid. On this regimen she developed frequent flares and side effects to most of the disease modifying antirheumatic drugs to which she had been exposed—for example, antimalarial drugs resulted in severe loss of peripheral vision in her right eye. Intramuscular gold injections (Mycosprin) were then given but also to no avail. High dose steroids (3g daily over five days intravenously (IV) given at 6–8 weekly intervals caused weight gain and Cushing’s syndrome. Attacks of myalgic pain affecting the neck, shoulders, and mid-back areas were common, and were present throughout her illness. Eventually this was diagnosed as fibromyalgia.

At the age of 29 she was admitted to hospital with severe dyspnoea and chest pain. She tested positive for cytomegalovirus and coxsackievirus B. Steroids were ineffective and an emergency tracheotomy was performed in 1996. She was admitted to the intensive care unit with congestive emphysema, bilateral pulmonary oedema, pleural effusions and a pericardial effusion. She was kept sedated for most of her admission. The tracheotomy tube was removed but had to be reinserted owing to the collapse of both arytenoid cartilages. Eventually a Montgomery stent (permanent tracheotomy) was inserted and this remained in place for some three years later. Treatment was started with daily oral cyclophosphamide. This resulted in severe neutropenia which required Neupogen as the white cell count had fallen to 1x10^9/l. Extensive alopecia also developed. Steroids were again given in a dose of 1 g twice weekly. Then she consulted a different rheumatologist (RAA) when she was admitted to the intensive care unit again with pericarditis and effusion, pleural effusions, peritonitis (polyserositis), hepatosplenomegaly, and a restrictive cardiomyopathy, which was later diagnosed as being due to amyloid. Gastric copy showed reflux oesophagitis with ulceration and severe candidiasis. Severe bone marrow depression with thrombocytopenia (15x10^9/l) necessitated intravenous gammaglobulin (Polygam) treatment, which was ineffective.

It was decided to attempt plasmapheresis combined with cyclophosphamide at 6–8 weekly intervals. Although the frequency of relapses was markedly reduced, she developed pseudomomas infection and the line had to be removed. Myocarditis was treated with β blockers (Sotacor 160 mg twice a day).

In February 1999 treatment with cyclosporin 125 mg twice a day and methotrexate 50 mg weekly was started. This resulted in fair but not complete control. Liver functions were measured weekly, but the high doses of methotrexate were well tolerated. Because of side effects, the cyclosporin had to be discontinued. The relapses were not as severe as or as frequent as previously. IV cyclophosphamide, together with mesna and zofran were given every 6–8 weeks. However, relapses occurred more frequently again requiring high dose steroids. Thalidomide was then attempted but even with minimal doses peripheral neuropathy ensued and it had to be discontinued. Premature ovarian failure was then diagnosed.
The use of Enbrel revolutionised control of the disease. However, a reduction of the dose to one injection weekly instead of the recommended two was ineffective and major relapses occurred. The use of TNFα antagonists in the treatment of AOSD dates back to 1997 when it was first suggested by Stambe and Wicks, who documented the use of thalidomide in a 44 year old woman unresponsive to the usual treatment, although in this particular patient the TNFα levels were low. Hoshino et al found high levels of TNFs as well as interferon γ and interleukin 6 in 12 patients with AOSD.

Elliott et al in 1998 used infliximab effectively in a patient with juvenile rheumatoid arthritis and systemic features. The first paper demonstrating its usefulness in AOSD was that of Cavagna et al in 2001. It was shown that long intervals between infusions resulted in relapses and that reduction of the interval of administration from eight to four weekly was effective. Skin reactions (for example, urticaria) were prevented by the administration of antihistamines before the infusions. Kraetsch et al also introduced the use of infliximab in 2001 treated six patients with a diagnosis of AOSD with infliximab. The fevers, arthralgias, myalgias, hepatosplenomegaly, and rash resolved in all six patients after the first course of treatment with the compound. All serological abnormalities also returned to normal. Up to the time of publication of that series, treatment had been given for between five and 28 months. It seems now that infliximab and entanercept have revolutionised the treatment of recalcitrant AOSD and should not be withheld from any patients who do not initially respond to conventional treatments which may have included cyclosporin, cyclophosphamide, both of which have been used in the treatment of this condition. High dose steroids combined with cyclophosphamide, on occasion, aborted the acute episodes in this patient but resulted in side effects of steroid overdosage.

Our patient had a number of unusual features of the disease and her response to anti-TNF treatment has been nothing short of dramatic.

References

1 Burcoglu A, Church C, Bontempo F. Therapeutic use of single-stranded naked denatured DNA (deformol DF) in antiphospholipid antibody syndrome (APLAS) [abstract]. Lupus 1996;5(S1):8


Authors’ response

The authors report an interesting case of a patient with severe adult onset Still’s disease refractory to multiple conventional treatments. A reduction of disease activity was not achieved with treatment with tumour necrosis factor (TNF) antagonist etanercept at a dose of 25 mg twice weekly was started. After a period of treatment with the dose approved for the treatment of rheumatoid arthritis a marked improvement of the patient’s symptoms was seen. Subsequent reduction of the dose to a weekly administration of 25 mg etanercept was followed by a relapse of the disease. Treatment had to be re-escalated to the original dose of 25 mg etanercept twice weekly. Again, a relevant reduction of disease activity was achieved. Up to now treatment with etanercept 25 mg twice weekly is effective and well tolerated by the patient.

The case demonstrates again that treatment directed against TNFα is an effective treatment for adult onset Still’s disease. Up to now, promising data have been presented only for treatment with infliximab. To our knowledge, this is the first published case of successful treatment of adult onset Still’s disease (AOSD) with the TNF receptor construct etanercept, suggesting that the use of TNFα is a potential approach to treatment of this disease. Additionally, as seen with several patients treated with infliximab, the case again underlines the need to give continuously a “minimum dose” of this drug to maintain the achieved remission of AOSD.

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References


Epidemiology of the rheumatic diseases


Eight years after the first edition, this revision reflects the extraordinary growing importance of epidemiology of the rheumatic diseases. The present volume illustrates the complexity of the discipline and its many links with other specialties, particularly with genetics. It makes it very clear that the epidemiology of rheumatic diseases deserves more than a sentence or a section at the beginning of each chapter in textbooks. This second edition has been largely rewritten and augmented by new contributions. It expands the scope to new aspects of rheumatology, including syndromes and the particular burden of overall musculoskeletal diseases, which is of major importance at the beginning of the Bone and Joint Decade (BJD).

The aims are to understand the basic concept of, and specific difficulties related to, the epidemiology of rheumatic diseases. Each chapter has a useful format which describes the methodological difficulties in the disease considered from the standpoint of diagnosis and classification criteria, incidence, prevalence and time trends, and exposure factors classified into genetic and non-genetic, particularly environmental, factors. Minor departures from this scheme are sometimes justified by the particular aspects of some diseases. Chapters are well referenced.

There is a supplementary thematic index with well organised and comprehensive information. Although up-to-date in the presentation and taxonomy of diseases, the book will probably require periodic revision because of continuous progress in the field, an undertaking which should be encouraged. As a comprehensive worldwide perspective of the rheumatic diseases as currently described in the BJD, the new chapter on osteoarthritis includes a prognostic section, which provides an understanding of outcomes. A tabular summary of occurrence data on osteoarthritis would have been welcome. This chapter is an important contribution to information about one of the most common conditions.

A clear synthesis of complicated data is given in the chapter on osteoporosis. New insights from contributors with innovative views are given on back pain, arm disorders, and fibromyalgia.

Epidemiology of the rheumatic diseases reflects the evolution of taxonomy, conceptualisation, knowledge, and increasingly documented epidemiology in the broad area of the rheumatic diseases and is abundantly documented. It is useful for both clinicians and epidemiologists. Authors and editors are to be commended for producing this second edition and should be encouraged to keep pace with changes in the field by preparing a third edition when opportune. Why not as a measure of progress by the end of the BJD?

F Guillemin

Spondyloarthropathies and related seronegative or B27 related diseases are covered in three chapters on psoriasis, ankylosing spondyloarthropathies (inflammatory bowel disease and acute anterior uveitis), and reactive arthritis and Reiter's syndrome. Although this forms a single topic, the presentation used by the authors is thoughtful. Further evolution in classification may be necessary. Chapters on other rare diseases like lupus, scleroderma, polymyalgia rheumatica, myositis, and systemic vasculitis provide in-depth information about recent advances in familial clustering, twin studies, genetic models, immunogenetics, etc.

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**FORTHCOMING EVENTS**

24th Annual Meeting of the American Society for Bone and Mineral Research
20–24 Sep 2002; San Antonio, TX, USA
Contact: ASBMR, 2025 M. Street, NW, Suite 800, Washington DC 20036-3309, USA
Tel: +1 202 367 1161
Fax: +1 202 857 1880
Email: asbmr@dc.sba.com

**Translational Research in Autoimmunity**
21–22 Sep 2002; Pavia, Italy
Contact: Organising secretariat: eventi S.R.L., Corso Cavour, 18/20 - 27100 Pavia, Italy
Email: tra@e20pr.com
Website: www.e20pr.com
Congress website: www.medicine.ucsd.edu/albani/2001 meeting

**OsteoArthritis Research Society International (OARSI) World Congress**
22–25 Sep 2002; Sydney, Australia
Contact: OsteoArthritis Research Society International (OARSI), 2025 M Street, NW, Suite 800, Washington DC 20036, USA
Tel: +1 202 367 1177
Fax: +1 202 367 2177
Email: oarsi@oarsi.org
Website: www.oarsi.org

**7th EULAR Postgraduate Course in Rheumatology**
22–27 September 2002; Budapest, Hungary
The course will cover clinical aspects of rheumatic diseases, concentrating on outcome, assessment and evidence based management, and the scientific basis of rheumatology. It is aimed at junior rheumatologists at the end of at least four years of postgraduate training but is open to all rheumatologists. The course will be conducted in English.
Course fee 550 euros, including tuition, accommodation, and full board.
Details: The preliminary programme and registration information are available on the EULAR website: www.eular.org

**Third International Conference on Familial Mediterranean Fever and Hereditary Inflammatory Disorders**
23–27 September 2002; La Grande Motte, France
Contact: Dr Isabelle Touitou, Laboratoire de Génétique Moléculaire et Chromosomique, Hôpital A de Villeneuve, Montpellier, France
Tel: 33 4 67 33 58 59
Fax: 33 4 67 33 58 62
Email: isabelle.touitou@igh.cnrs.fr
Website: www.congres.igh.cnrs.fr.FMF/2002

**10th International Congress on Antiphospholipid Antibodies**
29 Sep–3 Oct 2002; Sicily, Italy
Contact: Secretariat, 10th International Congress on Antiphospholipid Antibodies, c/o Kenes International, PO Box 50006, Tel Aviv 61500, Israel
Tel: 972 3 5140018/9
Fax: 972 3 5140077 or 972 3 5172484
Email: aps@kenes.com
Website: www.kenes.com/aps

**Third International Congress on Spondyloarthropathies**
2–5 Oct 2002, Gent, Belgium
Topics covered will be:
- Innate immunity
- Genetics and HLA-B27
- Animal models and pathogenesis
- Clinical research and therapy
Contact: Organisation and secretariat, Medicongress, Waalpoel 28-34, B-9960 Assenede, Belgium
Tel: +32 9 344 39 59
Fax: +32 9 344 40 10
Email: congresses@medicongress.com
Website: www.medicongress.com

**7th International Conference on Eicosanoids and Other Bioactive Lipids in Cancer, Inflammation and Related Diseases**
14–17 Oct 2002; Nashville, TN, USA
Contact: Lawrence J Marnett, Biochemistry Department, Vanderbilt University, School of Medicine, Nashville TN 37232-0146, USA
Tel: (615) 343 7329
Fax: (615) 343 7354
Email: www.eicosanoids.science.eayne.edu

**3rd International Conference on Sex Hormones, Pregnancy, and the Rheumatic Diseases**
21–24 Oct 2002; New Orleans, LA, USA
Contact: Anne Parke
Tel: 860 679 8190
Fax: 860 679 1287
Email: parke@nso.uchc.edu

**66th American College of Rheumatology AGM**
25–29 Oct 2002; New Orleans, USA
Contact: ACR, Ronald F Olejko, Director of Conferences and Meetings, 1800 Century Place, Suite 250, Atlanta, Georgia 30045–4300, USA
Tel: +1 404 633 3777
Fax: +1 404 633 1870
Email: acr@rheumatology.org
Website: www.rheumatology.org

**Third International Meeting on Social and Economic Aspects of Osteoporosis and Osteoarthritis**
7–9 November 2002; Barcelona, Spain
Contact: Yolande Piette Communication, Boulevard Kleyer 108, 4000 Liège, Belgium
Tel: 32 4 254 12 25
Fax: 32 4 254 12 90
Email: ypc@compuserve.com

**Certifying Examination in Pediatric Rheumatology**
18 Nov 2002
Contact: American Board of Pediatrics, 111 Silver Cedar Court, Chapel Hill, NC 27514-1513, USA
Tel: 919 929 0461
Fax: 919 918 7114 or 919 929 9255
Website: www.abp.org

**10th APLAR Congress of Rheumatology**
1–6 Dec 2002; Bangkok, Thailand
Contact: APLAR 2002 Secretariat
Fax: 66 2 716 6525
Email:secretariat@aplar2002.com
Website: www.aplar2002.com

**Future EULAR congresses**
18–22 June 2003; EULAR 2003 Lisbon, Portugal
9–12 June 2004; EULAR 2004 Berlin, Germany
8–11 June 2005; EULAR 2005 Vienna, Austria
21–24 June 2006; EULAR 2006 Amsterdam, The Netherlands