Work characteristics, demographic factors and clinical variables could predict work disability in rheumatoid arthritis

Authors: F. Wolfe and D.J. Hawley
Title: The longterm outcomes of rheumatoid arthritis. Work disability: A prospective 18-year study of 823 patients
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Aim: Work disability is a frequent and serious outcome of rheumatoid arthritis (RA), accounting for the greatest indirect costs of this condition. A longitudinal, long-term prospective study was conducted on a large cohort of RA patients (pts) to investigate the rate/risk of work disability and the factors that may be considered concomitant with and therefore predictors of work disability, using a broad series of clinical, laboratory and self-reported measures in all pts and in a subgroup of early RA pts.

Methods: RA pt data from 1974 were recorded in a database at the Wichita Arthritis Center in which demographic, clinical, and pt self-reported variables were registered at every pt visit. In 1994, 823 RA pts were examined and interviewed about their employment and work history. The pts were asked for occupation data - the start and stop dates of each job, the specific job title, the reason for which the job was stopped; demographic variables (sex, age, ethnic origin, education level, number of children, smoking history, total income, and current marital status); and clinical data [tender joint count, grip strength, health assessment questionnaire (HAQ), pain on a visual analogic scale (VAS), VAS global severity, AIMS anxiety and depression, co-morbid conditions, body mass index (BMI), and rheumatoid factor (RF)]. Values used in the prediction of work disability were those detected at the first visit. 2-year average values, and average values over the length of the study. Average variables were considered as full predictors when work disability could not be considered to have caused the predictor value. The primary analyses were Kaplan-Meier survival statistics and Cox regression. Proportional hazard assumptions were verified.

Results: 79 out of the 823 RA pts (9.6%) never worked for a salary. All the males and 87.3% of the females had been employed with a salary. 509 pts were employed at the time they had RA: the following statistical analyses were limited to these subjects. Work discontinuation was reported by 37.7%. The median survival time or time to work disability was 20.9 years, but the 25% survival time was 6.4 years. Out of 456 pts working at the first visit, 23% were work disabled in the first 5 years (8.6 in the first 2.5 years), 19.4% in the next 5 years and 57.6% beginning after 10 years of follow-up. The 25% survival time (time to work disability) was 10.05 years. Fewer years of formal education, a high BMI (showing a late effect on work disability), VAS pain scores, high HAQ scores, high RF and erythrocyte sedimentation rate (ESR) values, and high physical demands of the job were the covariates independently associated with work disability. Over the course of the illness, the work disabled patients had a 35% reduction in family income, and more abnormal scores for the joint count, grip strength, ESR, depression and anxiety. The results, except for BMI, were essentially similar in a subset of 156 pts in whom RA duration was less than one year.

Conclusions: Work disability in RA pts was frequent and could be predicted by almost all of the demographic and clinical variables and by the physical characteristics of the work process present at the first visit. The best predicting factors were a persistent elevation of ESR, pain, and disability according to HAQ detected in longitudinal follow-up. Thus, the optimal management of RA pts may differ based on their clinical, demographic, and work variables. The physician should try to treat the clinical variables medically and to modulate the demographic and work variables by active interaction with the patient, calling in other specialists (e.g., psychiatrists or psychologists) where necessary.

Comment

Work disability is a key outcome, and arguably the greatest economic cost, resulting from rheumatoid arthritis. Factors that influence work disability include disease activity variables, as shown in this large prospective study by Wolfe and Hawley. In addition, workplace factors and their interaction with disease are often difficult to disentangle. Thus, an employer who is unsympathetic to lost work time in relation to clinic visits and drug monitoring may be influential. Age is an important predictor and those with an older age at onset, and hence nearing retirement, are more likely to cease work permanently than those who are younger, though this was not covered in this paper. One problem in the interpretation of studies such as this is selection. In this paper by Wolfe et al. the mean disease duration at entry was 5 years and only about one-sixth of their patients were seen within a year of onset. Recent studies from both Finland (1) and the UK (2) suggest that work disability occurs early and may even pre-date the first hospital attendance. Thus, being in employment at the first clinic visit is likely to be a marker of a good prognosis. It is perhaps not surprising that clinical activity predicts both current and future work disability. The unanswered question, which may be answerable by data sets such as that from Wichita, is: If disease activity can be suppressed, could permanent work disability be prevented?

There are a number of non-clinical issues that influence work disability which make it difficult to extrapolate the results from one population more widely. As Wolfe et al. demonstrated (and as might be expected) physical demands are highly predictive. Other factors that vary between countries include...
social security and insurance provision, underlying unem-
ployment in the population and the willingness of society to accept disa-
blement in the workplace. It is thus of interest to note that in the recent UK study work disability was 32 times more likely in RA patients than in the local underlying popu-
lation (2).

Thus although the report by Wolfe et al. underlines the im-
portance of disease control in reducing work disability, such action alone may not have the impact desired.

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References

Opioids safely control chronic rheumatic disease pain

Authors: S.R. Ytterberg et al.
Title: Codeine and oxycodone use in patients with chronic rheumatic disease pain

Aim: The efficacy, toxicity, tolerance, dependence and addiction/abuse of opioids usually raises concerns about its use as an analgesic for chronic pain in rheumatic disease. A retrospective study was conducted to examine the effect of codeine and oxycodone in a large cohort of patients (pts) with defined rheumatologic diseases treated for a short or long period of time with opioids.

Methods: The prescription and use of codeine and oxycodone were retrospectively studied in a cohort of 644 rheumatology clinic pts by examining computerized pharmacy records, interviews, and medical record reviews to identify those who had been prescribed opioids during the previous 3 years. 354 pts were identified as never having taken opioids and 76 of these were used as the control group for the study. 290 pts had received prescriptions for opioid analgesics. They were divided into 2 groups: long-term opioid (LTO) use if prescriptions were filled for >3 consecutive months and short-term opioid (STO) use if prescriptions were filled for <3 months. Statistical analysis was carried out considering 3 groups: LTO, STO, and controls. Interview data included information about demographics, diagnosis, disease duration, type of opioid used, the patient’s assessment of opioid efficacy and toxicity, and a series of questions about substance abuse (alcohol, “street drugs”). Doses of opioid medications were converted to the equivalence of 30 mg of codeine (C-30 equivalents) to calculate a comparable mean daily dose for every pt. Dosage escalation was defined as an increase of ≥ 2C-30 equivalents per day. The records of pts whose dosage increased by ≥ 2C-30 were reviewed to search for the cause of the dose escalation (increased pain severity or development of tolerance and/or loss of control of opioid use) and to determine if the escalation was transient or per-
manent. Analysis of variance (ANOVA) was used to compare data among the 3 groups. “Post hoc” comparisons of groups were carried out by means of t-tests.

Results: 290/644 pts received codeine and oxycodone pres-
scriptions over the previous 3 years: 153 pts filled the opioid prescriptions for < 3 months (STO) and 137 for ≥ 3 consecutive months (LTO). For 266 pts in the opioid group complete data were obtained: 133 of them were included the STO group and 133 in the LTO group. Demographic data, diagnoses and disease duration were not significantly different among the three groups. Codeine was the most prescribed opioid (127/133 pts in the STO group, and 133/133 pts in the LTO group, alone or together with oxycodone).

Opioids significantly reduced the rheumatic disease pain severity scores from 8.2 to 3.6 (evaluated on a 0-10 scale) (P < 0.001). In 38% pts mild side effects (mainly nausea, dyspepsia, constipation and sedation) were reported. The mean ± SD initial dosage was 2.1 ± 1.7 30-mg codeine equivalents/day, the mean peak was 3.4 ± 3.3 mg/day, and the mean current dose was 2.7 ± 2.0/day. Dosage escalations occurred in 32 pts due to a worsening of the underlying rheumatic condi-
tion or to medical complications in all but 4 pts (3%), who developed tolerance and abuse behavior. Abuse behavior was not more frequent in the pts with or without a history of abuse/addiction.

Conclusions: Treatment of chronic rheumatic pain with co-
deine was effective and was associated only with mild side effects. Doses remained stable for long periods of time and escalations were mainly linked to a worsening of the under-
lying conditions or to complications, as already demonstrat-
ed for other diseases, rather than to the development of toler-
ance. For this reason, opioids may be considered an effective long-term or short term treatment, not associated with any severe side effects, for well-defined rheumatic diseases.

Comment
This retrospective study confirms the analgesic properties of codeine and oxycodone in rheumatic pain. Unfortunately, the authors did not examine the relationship between the efficacy of opioids, control of pain and the daily dose. The minimal efficacious dose of codeine for controlling chronic pain is 30 mg, three times a day, a dosage that can be increased up to 60 mg three times a day depending on the patient’s response. Therefore, the lack of pain control in this study may have been due to an under-dose of the drug.

Further improvement cannot be achieved with a daily dose exceeding 180 mg per day, as opioids have a plateau of effi-
cacy. Side effects are more frequent without any benefit for the patient if this plateau is exceeded. Side effects were indeed observed in those patients who received a dose of 120-360 mg per day, and it is likely that the side effects occurred mainly in those patients who received more than
180 mg of codeine per day. The mechanisms of codeine-induced analgesia are both central and peripheral. Nociceptive pain, in the course of inflammatory diseases, induces an inhibition of the central opioidergic pathways. On the other hand, inflammation of the joints and muscles upregulates the opioid receptors of the thin peripheral afferent endings, which consequently overdischarge. It is well known that codeine, when used to control pain, does not induce tolerance and addiction. Thus, codeine is a good analgesic drug with few side effects, and can be safely used when non-steroidal anti-inflammatory drugs are contraindicated.

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