Elderly Receive Suboptimal RA Treatment

BY SARA FREEMAN

FROM THE ANNUAL MEETING OF THE BRITISH SOCIETY FOR RHEUMATOLOGY

VITALS

Major Finding: For every 10-year increase in age, the chance of a patient with RA receiving more intensive treatment reduces by approximately 22%.

Data Source: Repeat cross-sectional study of 290 patients with RA.

Disclosures: Dr. Ma and Dr. Deighton declared no conflicts.

BIRMINGHAM, ENGLAND — Elderly patients with rheumatoid arthritis are treated less intensively than their younger counterparts, despite experiencing similar levels of disease activity.

Data from a cross-sectional study that was conducted at two centers in the United Kingdom show that for every 10-year increase in age, the chance of an RA patient's receiving more intensive treatment is reduced by approximately 22%.

"Unfortunately, the elderly population is not well represented in clinical studies," said Dr. Margaret H.Y. Ma, a clinical research fellow at King's College Hospital in London.

"In routine clinical practice, we see a much larger proportion of elderly patients, and it is unclear currently how well we treat this population," Dr. Ma said at the annual meeting of the British Society for Rheumatology.

The incidence and prevalence of RA increases with age, and it is in the elderly (aged 65 years and older) that disease-related disabilities usually have the greatest impact. Therefore, the aim of the study was to examine the effects of age and other variables on the treatment of RA.

Dr. Ma reported that the study, performed in 2009-2010 and involving 290 participants, was a repeat of a similar investigation that was performed in 2007-2008 and involved 236 people. The original and repeat cohorts of patients were similar in terms of age (58 and 59 years, respectively), sex (79% vs. 81% female), and ethnicity (70% vs. 72% white; 20% vs. 19% Afro-Caribbean). Treatment plans also were similar between the cohorts (80% vs. 81% taking disease-modifying antirheumatic drugs [DMARDs]; 11% vs. 11% taking steroids; 15% vs. 17% taking biologics). Patients in the repeat study, however, were more likely to have longer disease duration (10 years vs. 8.3 years) as well as a lower 28-joint disease activity score, or DAS28 (4.1 vs. 3.78), than did those who took part in the original study.

Both studies showed that there was a significant effect of age and disease activity on the chances that patients would be given more intensive therapy. While older patients were less likely to receive treatment increases (odds ratio, 0.83 in the original study and 0.82 in the repeat study), higher disease activity was associated with more intensive therapy (OR, 2.15 and 2.39, respectively).

Adjustment for possible confounding factors revealed that age and disease activity were the only determinants of treatment changes.

In the 2009-2010 cohort, the percentage of patients aged 65 years and older on DMARDs, steroids, and biologics was 77%, 11%, and 11%, whereas the percentage of those younger than 65 years who took these drugs was 83%, 19%, and 19%. When investigators compared patients aged 65 years or older vs. those younger than 65 years, they found that there were no differences in disease activity, with both age groups exhibiting a similar spectrum of disease activity in both the original and repeat studies. However, for the same DAS28, elderly patients were less likely than younger patients to receive an increase in therapy if they had more moderate disease, Dr. Ma reported.

Physicians treat very active disease more aggressively, Dr. Chris Deighton, consultant rheumatologist at the Derbyshire Royal Infirmary, Derby, England, commented. "If they have moderate disease, then there is probably more of a negotiation that takes place" between the patient and physician, he said. ■

Resistance Exercise Protects Muscle Mass in Arthritis

BY M. ALEXANDER OTTO

EXPERT ANALYSIS FROM A RHEUMATOLOGY SEMINAR SPONSORED BY UCLA

MARINA DEL REY, CALIF. — Rheumatoid arthritis patients with well-controlled disease may benefit from performing fat-burning exercises accompanied by resistance training—such as weight lifting—to preserve or even build muscle mass, according to Dr. Joan M. Bathon.

A seemingly fit patient with well-controlled rheumatoid arthritis (RA) and a normal body mass index may still have excess body fat, elevated C-reactive protein (CRP) levels, and increased coronary artery disease risk, said Dr. Bathon, professor of medicine and director of the Johns Hopkins Arthritis Center in Baltimore.

RA's chronic inflammation can waste muscles, she explained at a rheumatology seminar sponsored by the University of California, Los Angeles. Appendicular fat correlates with disability, and visceral fat correlates with coronary artery disease, she said. When patients have well-controlled RA, their high CRP levels might be coming not from the inflamed joints, but rather from fat deposits, and might signal an increased risk of coronary artery disease.

Dr. Bathon and her colleagues performed anthropomorphic measurements and dual-energy x-ray absorptiometry (DXA) scanning to assess fat:muscle ratio in 72 men and 117 women with RA and moderate disability. A single CT image of the abdomen in the axial plane was used to assess the amount of visceral fat.

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A seemingly fit RA patient with a normal body mass index may have a number of heart disease risk factors.

Women with RA and BMIs below 25 kg/m^2 or between 25 and 30 were more likely than controls to have sarcopenic obesity. Men with RA had increased levels of visceral fat (Arthritis Rheum. 2008;59:807-15). ■

Disclosures: *Dr. Bathon said she had no relevant disclosures.*

Biomarkers Sought to Match RA Patients With Drug Therapy

BY M. ALEXANDER OTTO

EXPERT ANALYSIS FROM A RHEUMATOLOGY SEMINAR SPONSORED BY UCLA

MARINA DEL REY, CALIF. — Like rheumatologists everywhere, Dr. Joan M. Bathon, director of the Johns Hopkins Arthritis Center, sometimes has to rely on clinical trial and error to find the right medications for her patients.

It would be a tremendous help to be able to predict treatment response, and ways to do that may be coming to the clinic in the not-too-distant future, she said at rheumatology seminar sponsored by the University of California, Los Angeles.

Dr. Bathon said she usually starts RA patients on methotrexate. It generally takes 2-4 months to tell if the drug is working.

If methotrexate fails to bring RA under full control, Dr. Bathon said she will add a second agent. If a low to moderate level of disease activity remains, she'll add sulfasalazine, leflunomide, or hydroxychloroquine, depending on patient preference. With more severe residual disease activity, she's likely to add a tumor necrosis factor (TNF) inhibitor.

If one TNF inhibitor doesn't work, she'll try another. If the patient fails two TNF therapies, abatacept or rituximab are the next options, although rituximab is being used more cautiously these days since being linked with progressive multifocal leukoencephalopathy, she said.

Tocilizumab (Actemra)—which was approved by the Food and Drug Administration earlier this year as the first interleukin-6 receptor-inhibiting monoclonal antibody—is also an option if TNF inhibitors fail. The rheumatology community is still figuring out its place in the treatment paradigm, she said.

Research into biomarkers to predict treatment response may make that possible, Dr. Bathon said during her presentation.

While biomarkers for disease progression, treatment toxicity, and other aspects of RA care are also being sought, the biggest efforts are going into finding biomarkers for treatment response, according to Dr. Bathon.

Knowing, for instance, the particular molecular pathway—TNF, B cell, or T cell—that is most active in an individual RA patient would indicate if that patient would benefit from a TNF inhibitor such as etanercept, a B-cell depleter such as rituximab, or some other therapy.

Research into biomarkers for TNF inhibitor response is particularly active.

One study recently found that polymorphism in a TNF-alpha promoter gene (-308 G greater than A SNP) is associated with higher serum levels of TNF-alpha in RA patients, suggesting that the polymorphism is a weak predictor of response to anti-TNF therapy (Rheumatology Reports 2009 [doi:10.4081/rr.2009.e1]).

"If you have this, a TNF [inhibitor] may be the drug of choice," Dr. Bathon said.

Another recent study measured an array of autoantibodies and cytokines in 93 patients from three different ethnic groups. A 24-biomarker signature was discovered that predicted good to excellent response, as well as lack of response, to etanercept (Arthritis Res. Ther. 2009;11:115). ■

Disclosures: *Dr. Bathon said she had no relevant financial disclosures.*