Statin Use May Lower Rheumatoid Arthritis Risk

BY MITCHEL L. ZOLER

PHILADELPHIA — Statin use was linked with a significant reduction in the incidence of rheumatoid arthritis in a retrospective, observational study of about 200,000 people in Israel.

The anti-inflammatory effect of statins is the hypothesized mechanism by which the drugs appear to have protected against development of rheumatoid arthritis (RA), Dr. Howard Amital said at the annual meeting of the American College of Rheumatology.

Added support for a causal relationship came from the observations that statin use had no impact on the incidence of osteoarthritis, and the protective effect for RA was greatest in people who received the highest statin dosages or the most potent agents.

"The degree of [RA] lowering with statin was remarkable," said Dr. Amital, head of the department of medicine at Meir Medical Center in Kfar-Saba, Israel.

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The investigators used medical records from patients enrolled in a large Israeli health maintenance organization that had 1.8 million enrollees during 1998-2007. The study excluded people who had an existing diagnosis of RA, osteoarthritis (OA), or rheumatic fever at the start of the study period, and also excluded people being treated with a corticosteroid, disease-modifying antirheumatic drug, or biological agent.

During the study period, about 200,000 people received statin treatment and did not have a preexisting diagnosis of RA or OA. During an average follow-up of 5 years, a total of 2,578 of these people received an initial diagnosis of RA and 8,906 received an initial diagnosis of OA.

To assess the impact of statin treatment, Dr. Amital and his associates broke the study population into five groups based on the percentage of days during follow-up that they were on statin treatment: 0%-19%, 20%-39%, 40%-59%, 60%-79%, and 80%-100%. The relative risk for developing RA was significantly related to statin use. Among people on a statin for 0%-19% of the follow-up period, the RA incidence rate was set as 1.0. The rate dropped with increased use, reaching a nadir of 0.6 in those on a statin for 80%-100% of follow-up, a statistically significant difference, compared with the reference subgroup.

The incidence rate for OA showed much less change across the range of statin use. In people on a statin for 80%-100% of follow-up, the rate of new OA was 85% of the rate among those who used a statin for 0%-19% of follow-up.

The impact of statin use on RA varied with the age of the patients. The statin effect appeared to be greatest among people aged 35-44 years. Among enrollees aged 75 or older, statin use had no significant association with RA incidence. The link between statin use and reduced RA incidence was very similar in women and men.

Dr. Amital and his associates also performed another analysis that stratified statin use into three tiers on the basis of dosage and drug potency. For example, a dosage of lovastatin of 40 mg/day or less was categorized as low efficacy (a dosage expected to reduce serum cholesterol by 30% or less), whereas an atorvastatin dosage of 10 mg/day was categorized as moderate efficacy (expected to cut serum cholesterol by 31%-40%). A lovastatin dosage of 80 mg/day or an atorvastatin dosage of 20 mg/day or greater were categorized as high efficacy (expected to reduce serum cholesterol by 41% or more).

This analysis showed that while all three statin categories were linked with significant reductions in RA incidence, the high-efficacy category was associated with the largest reduction in RA, and the low-efficacy category was linked with the smallest reduction.

Dr. Amital said that he and his associates had no conflicts of interest. ■