

# Effect of tumor necrosis factor a inhibitors on heart failure risk in RA Patients

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A chronic autoimmune disease, rheumatoid arthritis (RA) is characterized by persistent inflammation of the synovial membrane and progressive joint destruction. Beyond loss of mobility, sufferers face a high risk of heart failure. An inflammatory cytokine known for contributing to the development of RA, tumor necrosis factor a (TNFa) has also been implicated in cardiovascular disorders. Inhibition of TNFa has opened promising new treatment options for RA patients. Anti-TNF drugs such as infliximab, etanercept, and adalimumab have been shown to not only diminish signs and symptoms of the disease, but also prevent joint damage. However, in cardiac trials, TNFa inhibitors have shown no more positive effects on heart failure risk -- and sometimes less -- than placebo.

Does TNFa inhibition prevent heart failure in RA patients -- or promote it? That's the critical question Dr. Joachim Listing and a team of specialists with the German Rheumatism Research Centre in Berlin set out to answer. Featured in the March 2008 issue of *Arthritis & Rheumatism*, their study indicates that anti-TNF therapy does a patient's heart more good than harm, when it successfully reduces the inflammatory toll of RA.

To clearly assess the role of TNFa inhibitors in heart failure risk, the researchers analyzed a 3-year span of disease activity and cardiovascular incidents in 4,248 RA patients enrolled in an ongoing Germany-wide study of biologic therapy. At the time of enrollment, 2,757 of the subjects had started treatment with an anti-TNF drug -- infliximab, etanercept, or adalimumab -- and 1,491 had started a new disease-modifying antirheumatic drug (DMARD). Within the study period, several hundred of the patients were also treated with glucocorticoids, nonsteroidal anti-inflammatory drugs (NSAIDs), or COX-2 inhibitors. Over 78 percent of the patients were women. The mean age at baseline was 53.7 years for the anti-TNF group and 56 years for the DMARD controls.

Recorded at baseline and regular intervals through the 60-month follow-up, data on every patient included C-reactive protein level, duration of morning stiffness, and the number of tender and swollen joints, based on the 28-joint count Disease Activity Score (DAS). Cardiovascular events, whether acute or congestive, were also noted. Researchers used Cox proportional hazards models to investigate the impact of disease-related and treatment-specific risk factors on the development or worsening of heart failure.

At baseline, RA patients in the anti-TNF group had significantly more active disease, more physical limitations, and more heart problems than patients in the control group. Not surprisingly, the incidence rates of heart failure were significantly higher -- more than double -- for patients with a cardiovascular condition at the start of treatment than for those in good heart health. After adjusting for age, sex, body mass index, and prevalence of cardiovascular events, an increased risk of heart failure was found in patients with low functional capacity and high disease activity. Notably, a 2-point increase in the DAS28 score resulted in a 1.8-fold increase in heart failure risk.

When adjusting for functional capacity and disease activity at follow-up, along with the standard risk factors, the contribution of anti-TNF therapy to heart failure risk was insignificant. The small residual risk was balanced by the treatment's effectiveness in reducing inflammation, ultimately protecting the heart and other vital organs in addition to the joints. In contrast, COX-2 inhibitors and glucocorticoids, which tend to promote elevated blood pressure and insulin resistance, were associated with an increased risk of heart disease and heart attack.

Source: Wiley-Blackwell

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