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Poster Session V
Infections in immunocompromised patients

The risk of chronic Q fever in rheumatoid arthritis patients with and without anti-TNF-alpha therapy

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Objectives: During the Dutch Q-fever outbreak (2007-2010), many individuals became infected with the intracellular bacterium *Coxiella burnetii*. Initial infection is often asymptomatic. Chronic Q-fever, which develops in 1-5% of infected individuals, presents months to years after primary infection, mostly as endocarditis or vascular infection and has high mortality if left untreated. Immunosuppression, although not clearly defined, is a stated risk factor for chronic Q-fever. Anti-tumor necrosis factor α (anti-TNFα) therapy is associated with increased risk of intracellular infections. We examined whether rheumatoid arthritis **(**RA) patients with or without anti-TNFα therapy are at increased risk for chronic Q-fever.

Methods: RA patients living in Q-fever epidemic areas, were identified in rheumatology outpatient clinics in participating hospitals. A cohort of patients on anti-TNF therapy for at least 3 months during the epidemic was selected and a cohort TNF-naive patients who were using non-biological anti-rheumatic drugs during the same period.
Participants were screened for anti-*C. burnetii* antibodies, measuring IgG against *C. burnetii* phase I and II in serum. Patients with phase I and/or II IgG titers >=1:32 were defined as seropositives, indicating previous exposure to *C. burnetii*. All seropositive individuals were referred for follow-up. Chronic Q-fever was diagnosed by a team of Q-fever specialists based on high phase I IgG titre, positive *C. burnetii* PCR, imaging studies and clinical symptoms.

Results: From December 2011 to July 2012, 361 patients on anti-TNF therapy and 398 TNF-naive patients participated. Hundred-twelve patients (14.8%) were Q-fever seropositive: 57/361(15.8%) patients on anti-TNFα therapy, compared to 55/398(13.8%) anti-TNFα naive patients (*P*=0.47). A total of 10/112(8.9%) seropositive patients were diagnosed with chronic Q-fever: 7/57(12.3%) patients on anti-TNFα therapy and 3/55(5.5%) anti-TNFα naive patients (RR 2.25; 95% CI 0.61–8.27, *P*=0.32). Univariate analysis in all seropositive patients identified higher age, cardiac valvulopathy/prosthetic valve or aneurysm/vascular prosthesis and corticosteroid use as significant risk factors for chronic Q-fever.

Conclusion: This is the first systematic study on the risk on chronic Q-fever in a clearly defined immunocompromised patient group living in an epidemic area. We found a prevalence of Q-fever seropositivity in RA patients of 14.8%, approximately the same as the 12.2% previously reported for the general population living in the same area. Despite this, the overall prevalence of chronic Q-fever among seropositive RA patients was 8.9%, which is higher than what is described in an unselected population (1-5%). These figures suggest that RA patients may not be more susceptible for initial Q-fever infection, but may be more prone for the development of chronic Q-fever. However, in RA patients on anti-TNFα therapy, no significant additional risk for chronic Q-fever was found (RR 2.25; 95%CI 0.61–8.27, *P*=0.32). In this respect, we were unable to show that anti-TNFα therapy increases the risk for progression to chronic Q-fever.