Conclusion: Our results indicate an enhanced coagulation and fibrinolytic impairment in newly diagnosed RA patients. Effective antirheumatic treatments reduce this hemostatic imbalance, with significantly more pronounced effects of biologic drugs compared to conventional (MTX+glucocorticoids) treatment.

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OP0060

EXPOSURE TO SPECIFIC TUMOR NECROSIS FACTOR INHIBITORS AND RISK OF DEMYELINATING AND INFLAMMATORY NEUROPATHY IN PATIENTS WITH INFLAMMATORY ARTHRITIS. A COLLABORATIVE OBSERVATIONAL STUDY ACROSS FIVE NORDIC RHEUMATOLOGY REGISTERS

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Background: Though rare, studies have reported increased risk of neurological events including demyelinating disease of CNS (DML), multiple sclerosis (MS), and inflammatory neuropathy (INP) in patients with inflammatory joint disease treated with tumor necrosis factor inhibitors (TNFi).[1,2] More in-depth investigations are required to elucidate the association between TNFi and neurological events in these patients, especially whether rates differ across type of TNFi mode of action.

Objectives: To estimate the incidence of neurological events in patients with rheumatoid arthritis (RA) and spondyloarthritis (SpA, including axial spondyloarthritis and psoriatic arthritis) starting treatment with TNFi across five Nordic countries. To compare the incidence of neurological events in etanercept (ETN)-treated patients to patients treated with other TNFi (oTNFi).

Methods: We defined treatment cohorts of patients initiating TNFi between 2001 through 2018 from clinical rheumatology registers in Denmark (DK), Finland (FI), Iceland (IS), Norway (NO), and Sweden (SE). One patient could contribute to more than one treatment episode. Demographic data (sex, age), co-medication (methotrexate) and clinical variables (CRP, disease duration (<1 year, 1 to 5 years, >5 years) were extracted and used as covariates. We estimated crude incidence rates (IR) for neurological events and subtypes (ICD-10 codes: MS: G35, DML: G35, G36.0, G36.8-9, G37.1, G37.3, G37.5, G37.8-9, H46, H48.1, G04.8-9, INP: G61.0, G61.8-9), all countries pooled. We compared risk of neurological events between patients treated with ETN and oTNFi using Cox regression with time since treatment start, adjusted for the above covariates, robust standard errors, and stratified by country.

Results: We included 52,682 treatment starts, in 33,885 RA patients (DK 8,259, FI 3,765, IS 723, NO 1353, SE 19,785; 75% women, mean age 56 years) and 46,549 treatment starts in 28,772 SpA patients (DK 7,000, FI 2,885, IS 962, NO 2,684, SE 15,241; 47% women, mean age 45 years).

Numbers of DML, MS, INP and all neurological events, person-years (pyrs), and IRs in RA and SpA patients, for the two treatment groups are displayed in Figure 1. IRs for these neurological events showed some variation by diagnosis (RA vs. SpA), with rates of DML (and MS) in SpA patients around two (and three, respectively) times higher than the corresponding rates in RA (p<0.01), but similar rates for INP in RA and SpA patients. Comparing oTNFi to ETN, all Cox regression hazard ratios (HR) were statistically non-significant and close to one, whatever the outcome and the group of patients (Figure 1), with the adjusted HR (95%CI) for developing any neurological event in oTNFi compared to ETN being 1.08 (0.91-1.28) in RA patients and 0.96 (0.78-1.19) in SpA patients.

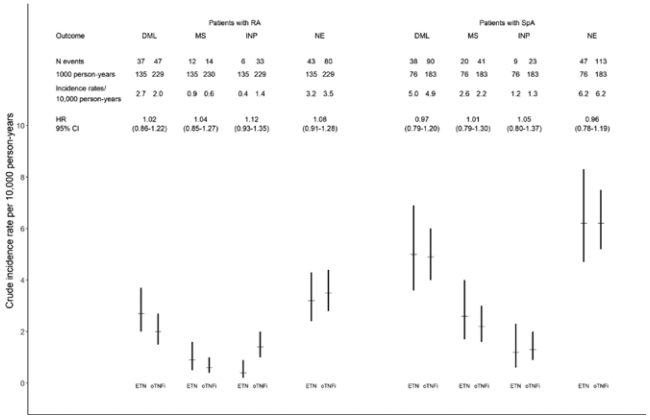

Figure 1. Number of events, pyrs and IRs of DML, MS, INP and all neurological events (NE) in RA and SpA patients, treated with ETN or oTNFi. HRs (95%CI) comparing oTNFi to ETN.

Conclusion: The incidences of DML and MS were lower in RA compared to SpA patients, while rates of INP were similar in both patients' groups. There was no evidence of differences in these rates between ETN and oTNFi. The findings are of importance from a safety perspective for patients starting TNFi.

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