Conclusion: Our findings show substantial decline in risk of dementia in patients with RA onset in the 2000s as compared to 1980s, including when compared to the general population comparators. This decline coincides with the advent of new biologic treatments for RA. Further studies should investigate this association using manual verification rather than billing codes for dementia, and should also elucidate the role of inflammation, autoimmunity, and anti-rheumatic treatments in risk of dementia.

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Conclusion:

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Acknowledgements:

Mortality using manual verification rather than billing codes for dementia, and should can be acknowledged when applying the EULAR definition of arthralgia suspicion. Furthermore, it is unknown whether this association differs pertinently when investigated with self-reported pain, or with pain in the form of tenderness at physical examination.

Objectives: The prevalence of subclinical inflammation with pain in MCP-joints specifically is not clear. Subsequently, it is unknown whether this association develops as the inflammatory process progresses to clinically apparent IA (determined at physical examination). In this nationwide 12-year assessment, the mortality risk among PsA cases/controls was significantly increased for all ages except <40 years, with the numerically highest point-estimates for ages 40-49 years and 50-59 years. Cause of death frequencies were numerically similar for the PsA cases/controls. Furthermore, causes of death from the Cause of Death Registry were described for PsA cases and controls.

Methods: Between April 2012- February 2019, 602 patients were consecutively included in the Leiden clinically suspect arthralgia (CSA)-cohort. Follow-up ended when patients developed clinically apparent IA (determined at physical examination), or else after 2 years (median follow-up time 25 months). MCP-joints were assessed for self-reported joint pain by the patient using a mannequin and self-reported joint tenderness by physical examination. Baseline ultrasonographic (US) MRI of the MCP (2-5)-joints were scored by two readers, blinded for clinical data, on subclinical inflammation (synovitis, tenosynovitis, osteitis). Associations between MCP-pain or MCP-joint tenderness and MRI-detected subclinical inflammation were studied at patient level by logistic regression analyses, entering the mentioned MRI-detected features separately (univariable) and together (multivariable).

Results: 33% of 227 patients with self-reported MCP-pain had MRI-detected subclinical inflammation and 38% of 226 patients with MCP-joint tenderness had MRI-detected subclinical inflammation. Self-reported MCP-pain joint was univariable associated with subclinical inflammation and synovitis in particular (OR 2.00, 95% CI: 1.21-3.30, OR 2.87, 95% CI: 1.29-6.39). In multivariable analysis this MCP-pain was associated with synovitis (OR 2.54, 95% CI: 1.12-5.77). MCP-joint tenderness was univariable associated with subclinical inflammation, and synovitis and tenosynovitis in particular (OR 1.84, 95% CI: 1.29-2.63, OR 1.76, 95% CI: 1.10-2.81, OR 1.69, 95% CI: 1.12-2.55, respectively). In multivariable analysis, tenosynovitis remained significant (OR 154, 95% CI: 100-2.36). Of all patients with self-reported MCP-pain joint who developed IA, 50% had MRI-detected subclinical inflammation. For MCP-joint tenderness this was 61%. Patients with MCP-joint tenderness without subclinical inflammation who developed IA, developed clinical arthritis at a joint that was not scanned (85%), hence they may have had subclinical inflammation that was not imaged. The other 15% did develop arthritis in an MCP-joint, suggesting that subclinical inflammation developed after CSA-onset.

Conclusion: Arthralgia in the MCP-joints is associated with subclinical inflammation in particular with synovitis and tenosynovitis. The prevalence of subclinical inflammation is highest for tender joints at physical examination; this can be acknowledged when applying the EULAR definition of arthralgia suspicious for progression to RA.

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Unrevealing the impact of PsA and comorbidity prevention

OP0218 MORTALITY IN PATIENTS WITH PSORIATIC ARTHRITIS IN SWEDEN


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Background: In contrast to the increased mortality reported in other inflammatory diseases such as rheumatoid arthritis and psoriasis, prior mortality studies in psoriatic arthritis (PsA) have shown inconsistent results.

Objectives: To compare all-cause mortality between PsA patients in Sweden and matched general population controls, and to describe cause of death distributions in the two groups.

Methods: All individuals in Sweden with ≥1 main diagnosis of PsA (ICD-10: L40.5/ M07.0/07.03) from outpatient visits to rheumatology or internal medicine clinics at age ≥18 years (y) 2001-2017 were identified from the Swedish National Patient Register. Each case was matched to 5 general population controls based on sex, county and age in the year of the first registered arthritis diagnosis for the case. Cases and controls were followed from 1 Jan, 2007, or from first PsA diagnosis thereafter for index cases, until first occurrence of death (data from the Swedish Cause of Death Register), emigration or 31 Dec, 2018. Mortality was assessed overall, as well as stratified by sex (45% males) and disease duration (PsA diagnosis prior to 2007 [38% of cases] vs. 2007-2017), using matched Cox proportional hazard regression, or – in case the Cox assumption regarding proportionality did not hold – matched Breslow test. To account for potential PsA misclassification (in a previous validation study, 86% of 400 cases fulfilled PsA classification criteria), a sensitivity analysis was performed by randomly removing 20% of cases with one of their own controls. Moreover, incidence rate ratios (IRR) of death were calculated overall and stratified by sex, disease duration and age. Finally, causes of death (from the Cause of Death Register) were described for PsA cases and controls.

Results: Over the 12y follow-up, 3 121 deaths occurred among 33 036 PsA cases (268 402 person-years at risk) and 12 864 deaths among 161 144 controls (1 302 250 person-years), resulting in an increased mortality among the PsA cases (HR 1.11 [95% CI 1.07-1.16], p < 0.001, Figure and Table; sensitivity analysis HR 1.09 [1.05-1.14]). The increased mortality was seen mainly among female PsA cases and among cases with longer disease duration (Figure; Table). IRRs of death were significantly increased for all ages except <40y, with the numerically highest point-estimates for ages 40-49y and 50-59y (Table). Cause of death frequencies among the PsA cases/controls were determined by the Cause of Death Register; emigration or 31 Dec, 2018. Mortality was assessed overall, as well as stratified by sex (45% males) and disease duration (PsA diagnosis prior to 2007 [38% of cases] vs. 2007-2017), using matched Cox proportional hazard regression, or – in case the Cox assumption regarding proportionality did not hold – matched Breslow test. To account for potential PsA misclassification (in a previous validation study, 86% of 400 cases fulfilled PsA classification criteria), a sensitivity analysis was performed by randomly removing 20% of cases with one of their own controls. Moreover, incidence rate ratios (IRR) of death were calculated overall and stratified by sex, disease duration and age. Finally, causes of death (from the Cause of Death Register) were described for PsA cases and controls.

Conclusion: In this nationwide 12-year assessment, the mortality risk among PsA patients in Sweden was increased by around 10% as compared to the general population, mainly driven by increased risks among females and patients with longer disease duration. Cause of death distributions were numerically similar between PsA cases and controls.

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