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FRI0101 RA IN MALE CARPENTERS: OCCUPATIONAL WOOD DUST EXPOSURE INCREASES ACPA AND RF SEROPOSITIVITY AND SIGNIFICANTLY RAISES RF TITRES

K. Bellis¹, M. Whallett², D. Murphy^{1,2}, D. Hutchinson^{1,2}. ¹Rheumatology, Royal Cornwall Hospital; ²University of Exeter Medical School, Cornwall Campus, Truro, Cornwall, United Kingdom

Background: Wood dust has been hypothesised as a trigger in RA. Exposure may activate peptidyl arginine deaminase through exposure to silica and carbon nanoparticles,¹ stimulating RF autoantibody production.² Though non-silica dusts have shown increased RA risk in construction workers, wood dust exposed individuals were excluded from analysis in a recent large registry study.³
Objectives: To analyse the autoantibody status of male RA wood workers in Cornwall, UK, compared to matched RA controls with no occupational dust or fume exposure.
Methods: All male RA patients were sent an occupational questionnaire, detailing current occupation, last occupation (if retired) and other occupations for >1 year. Telephone follow up was completed to minimise missing data. 41/720 (6%) patients died during the study period (22 months). 39/720 (5%) were excluded due to incomplete data. 147/640 (23%) had exposure to wood dust including other dust co-exposures, with 44/147 (30%) identifying wood dust as their primary occupational exposure as carpenters, approximately seven times higher than expected given UK employment census data. This cohort was matched to 102 RA controls with no dust or fume exposure for age +/- 5 years, sex and index of multiple deprivation (IMD) +/- 1 decile. RF titres were compared via 2 tailed Mann Whitney U test, and ACPA seropositivity was compared using a Z test of 2 proportions.
Results: No significant differences were seen in median age between woodworker cases (median 59 years IQR 50–63), and controls (median 58 years IQR 48–65). Woodworker never smokers demonstrated significantly higher seropositivity rates for both RF and ACPA than respective controls, and RF titres were significantly higher amongst woodworkers irrespective of smoking (Table 1). Median smoking rates were not significantly different between cohorts: woodworkers median pack years 23.5 (IQR 14.75–36), control median pack years 20.5 (IQR 10.25–32.75).

Table 1. Seropositivity rates and RF levels for woodworkers vs. controls

	Median RF (IQR)	ACPA+ (%)
Control never smokers (n=40)	16 (IQR 6.7–47.2)*	19/40 (48%)***
Control ever smokers (n=62)	68.9 (IQR 20.65–167.58)**	46/62 (74%)****
Wood worker never smokers (n=8)	86.4 (IQR 19.5–230.3)*	7/8 (88%)***
Wood worker ever smokers (n=36)	156.1 (IQR 54–321.1)**	29/36 (81%)****

*p<0.04; **p<0.01; ***p<0.04; ****p= not sig.

Conclusions: Wood dust exposed RA patients are more likely to be seropositive. Sequential environmental insults of smoking and wood dust exposure have an additive effect on rheumatoid factor levels, conferring increased disease severity. Further studies are needed to determine if occupational wood dust exposure causes RA.

References:

- [1] Murphy D, Sinha A, Hutchinson D. Wood Dust: A Trigger for Rheumatoid Arthritis? Am J Med 2015;128:e35.
- [2] Hubbard R, Lewis S, Richards K, Johnston I, Britton J. Occupational exposure to metal or wood dust and aetiology of cryptogenic fibrosing alveolitis. Lancet 1996;347:284–9.
- [3] Blanc PD, Jarvholm B, Toren K. Prospective risk of rheumatologic disease associated with occupational exposure in a cohort of male construction workers. Am J Med 2015;128:1094–101.

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FRI0102 ULTRASOUND AND MRI DURING FLARES OF PALINDROMIC RHEUMATISM REVEAL A DISTINCT PHENOTYPE EVEN IN IMMINENT RA

K. Mankia¹, M.-A. D'Agostino¹, R. Wakefield¹, G. Eugenio¹, L. Horton¹, J. Nam¹, A. Grainger², P. Emery¹. ¹Leeds Institute of Rheumatic and Musculoskeletal Medicine and Nihl Leeds Musculoskeletal Biomedical Research Unit; ²Radiology, Leeds Teaching Hospitals NHS Trust, Leeds, United Kingdom

Background: Palindromic rheumatism (PR) is a recurrent, self-abortive arthritis and/or peri-arthritis which progresses to RA in up to 50% of patients, especially

those that are anti-CCP positive (1). Whether PR is truly a prodrome of RA or a distinct syndrome is unclear; the pathological phenotype of PR flare, and whether this changes when RA is imminent, remains unknown

Objectives: To describe the clinical and imaging (US and MRI) phenotype during PR flares and to determine whether this changes in PR patients with imminent RA. We hypothesised PR patients with imminent RA would have a RA phenotype during flare

Methods: Patients were recruited from a prospective PR cohort. PR flares were defined as ≥2 of pain, swelling, erythema in or around ≥1 joint, that later normalised. Clinical details were recorded during flares. Blinded US assessment (wrists, MCPs, PIPs, elbows, knees, MTPs, ECUs and 2nd-5th finger flexor tendons) was performed during and between flares. Synovitis, tenosynovitis, subcutaneous oedema, peri-articular inflammation and peri-tendinous oedema were reported at each joint. Where possible, MRI was also performed on the most symptomatic region during flare. Patients were followed for progression to RA

Results: US was performed in 22 patients during flare. 19 patients also had non-flare US. Mean age was 49 yrs. 16/22 (73%) were anti-CCP+ and 6/22 (27%) anti-CCP-. Six (27%) patients developed RA (mean 23 weeks post flare); these patients had higher frequency of flares and absence of flare trigger (figure) but no difference in distribution of flaring joints. The flare US showed grey scale (GS) synovitis in 13/22 (59%) patients, power Doppler (PD) synovitis in 4/22 (18%) and no erosions. 12/22 (55%) patients had peri-articular inflammation and/or subcutaneous oedema; in 6 patients this was without synovitis/tenosynovitis. Tenosynovitis and/or peri-tendinous oedema were present in 6/22 (27%) patients. Non-flare US demonstrated fewer abnormalities with improvement post flare. In 5 patients multiple flares were imaged with variable US abnormalities identified. The US flare phenotype did not differ (from non-progressors) in patients who progressed to RA, with PD synovitis present in only 17% (table 1) contrasting with our early RA cohort where PD synovitis occurred in 73% of patients (2). MRI was performed on 8 patients (2 flares imaged in 1 patient) and detected more pathology than US. Bone marrow oedema (BME) was found in only 1 patient. No erosions were seen.

Table 1

US abnormality	Progressed to RA (n=6)	Not progressed to RA (n=16)
PD synovitis	1 (17%)	3 (19%)
GS synovitis	2 (33%)	10 (63%)
Tenosynovitis	1 (17%)	4 (25%)
Peri-tendinous oedema	0 (0%)	2 (13%)
Peri-articular inflammation	1 (17%)	7 (44%)
Subcutaneous oedema	3 (50%)	6 (38%)

	Progressed to RA (n=6)	Not progressed to RA (n=16)
Anti-CCP positive	5 (83%)	11 (69%)
Disease duration (yrs), median (IQR)	2.6 (1.8-9.2)	3.1 (1.1-7.4)
Frequency of flares (per yr), median (IQR)	25 (12-50)	9 (5-23)
Flare Trigger identifiable?	0 (0%)	7 (44%)
	Mechanical	0 (0%)
	Emotional Stress	0 (0%)
Flare symptoms		
	Joint pain	6 (100%)
	Joint swelling	5 (83%)
	Skin colour change	6 (100%)
Number of joints involved in the flare, median (IQR)	2 (1-5)	2 (1-4)
CRP (mg/L), median (IQR)	10.8 (6.0-31)	9.0 (2.2-41.3)

Conclusions: PR flares have a distinct imaging phenotype characterised by peri-articular inflammation and subcutaneous oedema, often without synovitis. The low prevalence of PD synovitis, BME and erosions distinguishes PR from RA. PR patients with imminent RA have no triggers and more frequent flares, but retain the distinct PR phenotype. This suggests distinct pathological mechanisms in PR and should be of value for potential therapeutic interventions.

References:

- [1] Russell AS et al. J Rheumatol 2006;33(7):1240–2.
- [2] Horton SC et al. Rheumatol 2016;55:1177–87.

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FRI0103 CONTRIBUTION OF SUBJECTIVE DISEASE ACTIVITY SCORE 28 (DAS28) COMPONENTS TO THE RESPONSE TO TREATMENT OF RHEUMATOID ARTHRITIS

K.M. Son¹, S.Y. Lee², Y.I. Seo², J.-E. Choi³, H.A. Kim². ¹Hallym University Dongtan Sacred Heart Hospital; ²Hallym University Sacred Heart Hospital, Gyeonggi-Do; ³Ewha womans University, Seoul, Korea, Republic Of

Background: Some patients with rheumatoid arthritis (RA) do not respond adequately to treatment.

Objectives: We investigated the contributions made by the subjective components of the disease activity score 28 (DAS28) to the treatment response of RA. In addition, factors associated with poor response to treatment at 6 months, despite normalization of objective measures, were examined.