HOA cohort showed a positive association between soft tissue swelling, tenderness upon pressure and pain with radiographic progression.³

Objectives: To identify new prognostic factors and to confirm existing evidence on already identified prognostic factors for radiographic progression in HOA.

Methods: All 270 patients from this cohort were contacted to participate in a second follow-up visit after ten years at the Ghent University Hospital. A total of 106 patients consented to this follow-up visit. Presence of tender and swollen joints was assessed. Grip strength was measured. Functional Index for HOA (FIHOA) and Australian/Canadian OA Index questionnaires (AUSCAN) were completed by the patients. Pain was scored on a visual analogue scale from 0-100mm (VAS pain). Radiographs of hands were taken and scored using the anatomical phase scoring system for HOA by Verbruggen and Veys⁴. Patients were defined as radiographic progressor when two or more joints progressed to another phase within the scoring system, except for N-phase to S-phase. Logistic regression was performed on the clinical data and outcomes such as presence of tenderness (yes or no), soft tissue swelling (yes or no), VAS pain, disease duration, FIHOA and AUSCAN. VAS pain was dichotomized into two groups: VAS ≤33mm and VAS >33mm. Disease duration was dichotomized in ≤5 years and >5 years: p-values and odds ratios (OR) with 95% confidence intervals (95% CI) were calculated.

Results: After a mean follow-up of 9.7 years, 73.3% of the patients showed radiographic progression. The following clinical factors were associated with radiographic progression on patient level (OR [95%CI]): presence of tenderness (4.19 [1.52-11.61]), presence of soft tissue swelling (2.73 [1.08-6.92]) and disease duration > five years (4.00 [1.50-10.66]). Other outcomes were retained as prognostic factors for radiographic progression (OR [95%CI]): VAS pain (1.02 [1.00-1.04]), total FIHOA (1.12 [1.03-1.23]), total AUSCAN (1.02 [1.01-1.04]), AUSCAN pain (1.07 [1.02-1.12]), AUSCAN function (1.03 [1.01-1.06]) and VAS pain > 33mm (2.87 [1.14-7.24]). The mean number of E and R joints was 1.2 and 1.4 at baseline, and 0.2 and 5.2 after 10 years (p<0.001). The total number of joints that progressed to the E-phase and R-phase over a period of ten years was respectively 20 (1.05%) and 399 (21%).

Conclusion: Clinical presence of pain and soft tissue swelling at baseline remain strong predictors of radiographic progression in HOA after ten years. Tenderness upon pressure, moderate VAS pain and moderate impaired function at baseline are identified as new predictors. After ten years, more remodeling and less erosive joints are seen.

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THU0417 THE SEVERITY OF STRUCTURAL AND INFLAMMATORY FEATURES OF HAND OSTEOARTHRITIS ASSOCIATE WITH PERIPHERAL PAIN SENSITIZATION

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Background: Increased sensitivity to pain in the area around joints with osteoarthritis (OA), i.e., peripheral sensitization, is associated with higher pain severity. The mechanisms underlying peripheral sensitization in hand OA have not been substantially explored in previous studies.

Objectives: We aimed to explore whether the severity of structural or inflammatory OA features in the distal and proximal interphalangeal (DIP/PIP) joints were associated with lower pressure pain thresholds (PPT) at the same joint in a large study sample of persons with hand OA.

Methods: In these cross-sectional analyses we included 277 persons with hand OA from the Nor-Hand cohort. All participants underwent radiography and ultrasound examination (GE S8, ML 6-15MHz probe) of both hands. Pressure pain threshold (PPT) was tested with a hand-held algometer (FPIIX25, 1cm² flat rubber probe) in perpendicular position at the dorsal side of the DIP/PIP joint reported to be the most painful. Information about demographics and psychosocial factors was collected with questionnaires. We examined whether increasing radiographic severity (Kellgren-Lawrence 0-4 score) and ultrasound-detected inflammation (grey-scale synovitis 0-3 score and presence of power-Doppler activity) were associated with PPT at the same joint. We conducted separate analyses of linear regression with adjustments for age, sex, BMI, education level, use of analgesics, sleep disturbance, the Pain Catastrophizing Scale and the Hospital Anxiety and Depression Scale.

Results: The majority of the study population were women (89%), median age was 61 (IQR 57-66) years, mean BMI was 26.3 (SD 4.6) kg/m² and median duration of hand OA symptoms was 6 (IQR 3-13) years. The DIP/PIP joints demonstrated a wide range of PPT (mean (SD) 3.9 (1.9), range 0.6-11.4 kg/cm²) and OA severity (Table). We found that both increasing radiographic severity and inflammation were associated with lower PPTs (Table). When including grey-scale synovitis and power Doppler as confounders in analysis of radiographic severity, the results became weaker but remained significant (Table).

Abstract THU0417 – Table 1. Associations of structural and inflammatory OA features of DIP/PIP joints to pressure pain thresholds at the same joint, n=277.

	PPT (SD), kg/ cm ²	Adjusted beta (95% CI)	
Radiographic severity (Kellgren-Lawrence)			
Grade 0 (n=63)	4.5 (1.9)	0.0 (ref.)	0.0 (ref.)
Grade 1 (n=39)	4.3 (1.8)	-0.4 (-1.1, 0.3)	-0.4 (-1.1, 0.4) *
Grade 2 (n=61)	4.6 (2.2)	-0.1 (-0.7, 0.6)	0.1 (-0.6, 0.7) *
Grade 3 (n=54)	3.2 (1.6)	-1.5 (-2.2, -0.8)	-1.3 (-2.0, -0.6) *
Grade 4 (n=60)	2.9 (1.2)	-2.0 (-2.6, -1.3)	-1.6 (-2.3, -0.8) *
p for trend		<0.001	<0.001 *
Grey-scale synovitis			
Grade 0 (n=155)	4.3 (2.0)	0.0 (ref.)	
Grade 1 (n=56)	3.8 (2.0)	-0.6 (-1.2, -0.1)	
Grade 2 (n=42)	3.3 (1.2)	-1.1 (-1.8, -0.4)	
Grade 3 (n=24)	2.6 (1.5)	-1.7 (-2.5, -0.9)	
p for trend		<0.001	
Power Doppler			
Grade 0 (n=266)	4.3 (2.1)	0.0 (ref.)	
Grade 1-3 (n=14)	3.0 (1.3)	-1.3 (-1.8, -0.9)	

Adjusted for age, sex, BMI, analgesics, education, sleep disturbance, the Pain Catastrophizing Scale and the Hospital Anxiety and Depression Scale. *Additional adjustments for grey-scale synovitis and power Doppler.

Conclusion: Both structural and inflammatory hand OA features in DIP/PIP joints were associated with lower pressure pain thresholds, i.e. pain sensitization, at the same joint. Our results suggest that peripheral sensitization might be driven by structural and inflammatory features, and that targeting the disease early might prevent peripheral sensitization and reduce pain.

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