SATURDAY, 17 JUNE 2017

Spondyloarthritis - clinical aspects (other than treatment)

SAT0382 CHANGES IN VOLUMETRIC BONE MINERAL DENSITY AND BONE MICROARCHITECTURE IN PATIENTS WITH ANKYLOSING SPONDYLITIS. A FIVE-YEAR PROSPECTIVE STUDY USING HRPQCT

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Background: Studies have demonstrated increased prevalence of osteoporosis in patients with ankylosing spondylitis (AS) in the hip and lumbar spine assessed by conventional DXA but the peripheral skeleton is less studied. The peripheral skeleton can be studied in detail by high-resolution peripheral quantitative computed tomography (HRpQCT) demonstrating data of the volumetric bone mineral density (vBMD) and bone microarchitecture. We have previously shown that patients with AS from Western Sweden had lower vBMD measured by HRpQCT in radius and tibia compared with healthy controls [1]. No prospective study in this matter has been published in AS.
Objectives: To investigate changes over 5 years in the peripheral vBMD and microarchitecture in patients with AS.
Methods: HRpQCT of ultra-distal radius and tibia was performed in male AS-patients (NY criteria) at baseline and at the five-year follow-up. The patients were also assessed with blood samples and questionnaires.
Results: Of the 69 patients included at baseline 57 (83%) patients were re-examined at the five-year follow-up. Baseline characteristics of the 57 patients [median (IQR)]: age 48 (35 to 61) years, symptom duration 21 (11 to 34) years, ESR 10 (5 to 17) mm/h, CRP 3 (1 to 7), $ASDAS_{CRP}$ 1.8 (1.3 to 2.8) and BASDAI 2.3 (1.2 to 4.2). 23% used TNF-inhibitors, 75% used NSAIDs and 2% bisphosphonates. All measurements at tibia had good quality and matched images had common regions $\geq$80%. The images of radius of 12 patients had to be excluded due to insufficient quality. At tibia, the total, cortical and trabecular vBMD decreased significantly. In the microarchitecture an increase in the trabecular separation was seen (Table). Changes in vBMD were negatively and significantly correlated; Spearman's correlation coefficient between -0.3 and -0.4, to Δ-values (difference between follow-up and baseline) for ESR, CRP (cortical vBMD), $ASDAS_{CRP}$ (total vBMD and cortical vBMD) and BASDAI (total vBMD). At radius, no significant change in vBMD was observed; however, less power for analyses of radius. An increase was seen in the cortical thickness and the trabecular number while the trabecular thickness decreased (Table). Changes in cortical vBMD was negatively and significantly correlated, r $\approx$ -0.3, to Δ-CRP and Δ-$ASDAS_{CRP}$.

Table.

Site		Baseline mean ± SD	Five-year follow-up mean ± SD	Difference mean% ± SD	p-value
Tibia	**Total vBMD, mg/cm³**	297.9 ± 55.5	292.8 ± 55.8	**-1.7 ± 3.9**	**0.002**
	Cortical vBMD, mg/cm³	842.7 ± 54.8	834.1 ± 62.5	**-1.1 ± 1.9**	**0.001**
	Trabecular vBMD, mg/cm³	184.8 ± 34.6	179.5 ±31.9	**-2.6 ± 4.8**	**0.001**
	Cortical thickness, mm	1.22 ± 0.32	1.21 ± 0.32	-1.0 ± 5.2	0.35
	Trabecular thickness, mm	0.08 ± 0.01	0.07 ± 0.01	-1.5 ± 7.3	0.26
	Trabecular number in 1/mm	2.06 ± 0.30	2.04 ± 0.32	-0.7 ± 6.7	0.19
	Trabecular separation, mm	0.42 ± 0.08	0.43 ± 0.08	**1.8 ± 6.9**	**0.027**
Radius	Total vBMD, mg/cm³	319.6 ± 64.9	320.7 ± 69.9	0.1 ±4.7	0.47
	Cortical vBMD, mg/cm³	854.4 ± 54.9	854.1 ± 59.5	-0.1 ±1.9	0.98
	Trabecular vBMD, mg/cm³	177.5 ± 40.5	173.6 ± 41.9	-2.3 ± 6.2	0.082
	Cortical thickness, mm	0.83 ± 0.21	0.85 ± 0.21	**2.5 ± 5.4**	**0.004**
	Trabecular thickness, mm	0.08 ± 0.02	0.07 ± 0.01	**-5.4 ± 10.5**	**0.001**
	Trabecular number in 1/mm	1.96 ± 0.24	2.01 ± 0.24	**4.2 ± 10.8**	**0.041**
	Trabecular separation, mm	0.44 ± 0.07	0.43 ± 0.06	-2.7 ± 10.1	0.080

Conclusions: Over five years, this group of male patients with AS decreased in the vBMD of tibia, both trabecular and cortical. Even though there were alterations in the microarchitecture, no significant change in vBMD of radius was seen. Increases in inflammatory markers and disease activity had a negative impact on the cortical vBMD. The differences in the development of vBMD and microarchitecture in loaded and unloaded skeleton as well as factors associated with the changes needs to be further investigated.
References:
[1] Klingberg, E., et al., Bone microarchitecture in ankylosing spondylitis and the association with bone mineral density, fractures, and syndesmophytes. Arthritis Res Ther, 2013. 15: p. R179.
Disclosure of Interest: None declared
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SAT0383 BIOMARKERS OF ECM DEGRADATION REFLECT DISEASE ACTIVITY IN RADIOGRAPHIC AND NON-RADIOGRAPHIC SPONDYLARTHRITIS

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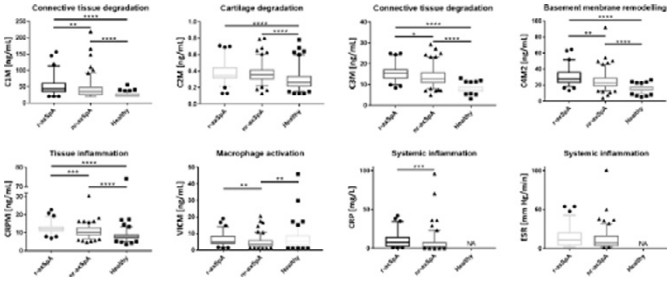
Background: Axial spondyloarthritis (axSpA) comprises two groups - radiographic (r-axSpA) and non-radiographic (nr-axSpA) with varying disease activity, spine involvement and response to biological therapy. An objective biomarker of disease activity may be able to select patients, which will benefit from a given biological treatment.
Objectives: We investigated the association of extracellular matrix (ECM) degradation biomarkers in axSpA patients (r-axSpA and nr-axSpA) with disease activity.
Methods: AxSpA patients (n=193; 72 r-axSpA and 121 nr-axSpA) and 100 healthy controls were included in the study. Biomarkers of type I, II, III and IV degradation (C1M, C2M, C3M, C4M2), MMP-degraded CRP (CRPM) and MMP-degraded and citrullinated vimentin (VICM) were detected by ELISA in serum. Mann-Whitney t-test tested the difference in the biomarker levels between groups and multiple regression analysis investigated the association between biomarkers and clinical manifestations with adjustment for age, gender, BMI, disease duration and CRP. ROC AUC tested the biomarkers capacity to differentiate the patient groups.
Results: Patients with r-axSpA compared to nr-axSpA patients had higher radiographic status and longer disease duration ($p<0.001$), whereas nr-axSpA had more swollen joints (p=0.0093). They were alike in age, BMI and disease activity (ASDAS-CRP, BASDAI and HAQ). All tested biomarkers except VICM were elevated in the axSpA patients compared to healthy subjects (all $p<0.001$). VICM was lower in the axSpA group, particularly in nr-axSpA compared to healthy (p=0.036 and p=0.002 respectively). R-axSpA compared to nr-axSpA patients had higher level of C1M, C3M, C4M2, CRPM and VICM ($p<0.001$, =0.001, <0.001, <0.001 and =0.003), but not C2M (p=0.92). C1M correlated to ASDAS-CRP in both r-axSpA and nr-axSpA (r-partiel: 0.46 and 0.44) with adjustment for age, gender, BMI and disease duration, but the correlation was lost by adjustment for CRP. Also C3M and C4M2 correlated to ASDAS-CRP in both axSpA groups. C2M was in r-axSpA patients moderately correlated to ASDAS-CRP, but minimal after adjustment of CRP (r-partiel: 0.32 unadjusted and 0.18 adjusted). CRPM correlated to ASDAS-CRP with adjustment to CRP in r-axSpA (r-partiel: 0.27 unadjusted, 0.26 adjusted), but not in nr-axSpA (r-partiel: 0.19 unadjusted, 0.03 adjusted).
Especially, C3M and C4M2 could differentiate between healthy and the axSpA patients (AUC 0.95 and 0.89), but also C1M, C2M, CRPM had AUC of $\geq$0.72. VICM was the only biomarker to differ between r-axSpA (AUC 0.92 and nr-axSpA (AUC 0.51).



Conclusions: Biomarkers of ECM turnover, C3M and C4M2 in particular, were associated with r-axSpA and nr-axSpA and could define disease. The biomarkers of ECM degradation, especially C1M, may reflect the pathogenetic background of axSpA.
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SAT0384 RISK OF MYOCARDIAL INFARCTION AND CEREBROVASCULAR ACCIDENT IN ANKYLOSING SPONDYLITIS: A GENERAL POPULATION-BASED STUDY

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Background: There is conflicting data on the risk of myocardial infarction (MI) and cerebrovascular accidents (CVA) in patients with Ankylosing Spondylitis (AS).