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Disclosure of Interest None declared.

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COMPLEX ASSESSMENT OF BONE MINERAL DENSITY, FRACTURE RISK, VITAMIN D STATUS AND BONE METABOLISM IN HUNGARIAN SYSTEMIC SCLEROSIS PATIENTS

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10.1136/annrheumdis-2018-EWRR2019.155

Career situation of first and presenting author Instructor.

Introduction Osteoporosis has been associated with systemic sclerosis (SSc).

Objectives We wished to determine bone alterations in SSc patients by conventional densitometry (DXA), peripheral quantitative computed tomography (pQCT) and bone biomarkers.

Methods We included 44 SSc patients and 33 age-matched healthy controls. Lumbar spine and femoral neck bone mineral density (BMD) was assessed by DXA. Volumetric BMD was measured by pQCT at the radius. FRAX, 25-hydroxyvitamin-D₃ (25-OH-D₃), parathyroid hormone, osteocalcin, C-terminal collagen telopeptide and procollagen type I amino-terminal propeptide were also assessed.

Results SSc patients had lower L2–4 BMD (0.880 ± 0.108 vs. 0.996 ± 0.181 g/cm²; $p=0.019$) and femoral neck (FN) BMD (0.786 ± 0.134 vs. 0.910 ± 0.090 g/cm²; $p=0.007$) by DXA. In SSc vs controls, pQCT indicated lower mean cortical (328.03 ± 103.32 vs 487.06 ± 42.45 mg/cm³; $p<0.001$) and trabecular density (150.93 ± 61.91 vs 184.76 ± 33.03 mg/cm³; $p=0.037$). Vitamin D₃ deficiency was more common in SSc vs controls (60.0% vs 39.3%; $p=0.003$). L2–4 ($p=0.002$) and FN BMD ($p=0.015$) positively correlated with BMI. pQCT assessments confirmed an inverse correlation between pulmonary manifestation and total ($p=0.024$), trabecular ($p=0.035$) and cortical density ($p=0.015$). Anti-Scl70 positivity inversely correlated with pQCT total density ($p=0.015$) and the presence of digital ulcers with cortical density ($p=0.001$). We also found that vertebral and FN BMD as determined by DXA significantly correlated with pQCT total, trabecular and cortical density ($p<0.05$).

Conclusions The results of our study suggest that bone loss in SSc patients may be associated with lower BMI, anti Scl70 positivity, and the presence of pulmonary manifestations and digital ulcers. Both DXA and pQCT are appropriate tools to evaluate the bone alterations in SSc patients.

Acknowledgements This research was supported by Hungarian National Scientific Research Fund (OTKA) grant No. K 105073 (H.P.B. and Z.S.); by the European Union and the State of Hungary and co-financed by the European Social Fund in the framework of TAMOP-4.2.4.A/2-11/1-2012-0001 'National Excellence Program' (Z.S.); by the European Union grants GINOP-2.3.2-15-2016-00015 and GINOP-2.3.2-15-2016-00050 (Z.S.).

Disclosure of Interest None declared.

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TRANSMEMBRANE TNF TRANSGENIC MICE EXHIBIT ENLARGED LYMPH NODES AND ELEVATED NUMBERS OF HIGH ENDOTHELIAL CELLS ASSOCIATED WITH INCREASED LYMPHOCYTE RECRUITMENT

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10.1136/annrheumdis-2018-EWRR2019.156

Career situation of first and presenting author Post-doctoral fellow.

Introduction NF- κ B signaling plays a crucial role in chronic inflammatory diseases and can be activated via two pathways. Noncanonical NF- κ B signaling can be induced by different signals, including transmembrane (tm)TNF ligation to TNF receptor 2 (TNFR2), which is restricted to specific cell types including endothelial cells (EC). Activation of the noncanonical NF- κ B pathway in EC is involved in differentiation into high endothelial cells (HEC), which make up specialized high endothelial vessels (HEV) that recruit lymphocytes into lymph nodes (LN). tmTNF transgenic (tg) mice overexpressing tmTNF develop chronic inflammatory features, including arthritis and axial inflammation.

Objectives To study LN size and composition, including stromal cell and lymphocyte subsets in tmTNF tg mice. We hypothesized that overexpression of tmTNF results in increased noncanonical NF- κ B signaling in HEC leading enhanced lymphocyte recruitment.

Methods Peripheral (PLN) and mesenteric LN (MLN) of 7 week old tmTNF tg mice and age-matched wild types (WT) were collected, dissected and analyzed using flow cytometry. Cellular markers were used to distinguish between different EC subsets, and B and T lymphocytes.

Results Lymph nodes of tmTNF tg mice had more than two-fold increased total cell numbers compared to WT (PLN: $23.7 \times 10^6 \pm 7.9 \times 10^6$; MLN: $24.9 \times 10^6 \pm 5.3 \times 10^6$. WT PLN: $5.2 \times 10^6 \pm 1.0 \times 10^6$; MLN: $10.4 \times 10^6 \pm 4.6 \times 10^6$). Total numbers of HEC in tmTNF tg mice LN were increased (PLN: $2.3 \times 10^3 \pm 0.50 \times 10^3$; MLN: $1.9 \times 10^3 \pm 0.64 \times 10^2$. WT PLN: 714 ± 393 ; MLN: 1.199 ± 506), as were capillary EC (CEC) numbers (PLN: 2067 ± 42 ; MLN: 2425 ± 71 . WT PLN: 1097 ± 609 ; MLN: 1327 ± 230). Notably, HEC:CEC ratio was increased in tmTNF tg mice (PLN: 1.10 ± 0.241 . WT PLN: 0.66 ± 0.006). In addition, total B and T lymphocytes in tmTNF tg mice LN were strongly increased (B lymphocytes: PLN: $10.2 \times 10^6 \pm 3.5 \times 10^6$; MLN: $10.9 \times 10^6 \pm 1.8 \times 10^6$. WT PLN: $2.8 \times 10^6 \pm 5.8 \times 10^5$; MLN: $5.8 \times 10^6 \pm 2.4 \times 10^6$. T lymphocytes: PLN: $12.8 \times 10^6 \pm 4.5 \times 10^6$; MLN: $14.2 \times 10^6 \pm 2.8 \times 10^6$. WT PLN: $2.5 \times 10^6 \pm 3.8 \times 10^5$; MLN: $4.7 \times 10^6 \pm 2.1 \times 10^6$).

Conclusions Overexpression of tmTNF in mice leads to an increase in total HEC, CEC and lymphocyte numbers and an increased HEC:CEC ratio in PLN and MLN. The increase in HEC may be the result of enhanced noncanonical NF- κ B signaling, enhancing lymphocyte recruitment into the LN. Consequently, interfering with noncanonical NF- κ B signaling in EC may be a promising treatment strategy to dampen inflammation in immune-mediated inflammatory diseases, which is the subject of current studies.

Disclosure of Interest None declared.