than 30 minutes, erythrocyte sedimentation rate less than 45mm/hour and imaging studies with changes related to osteoarthritis (radiography, magnetic resonance imaging or bone scintigraphy). Patients with autoimmune diseases such as rheumatoid arthritis, lupus or Sjögren syndrome were excluded.

The clinical records of patients diagnosed with EDPOA and treated between January 2015 and June 2019 at the Valle del Lili foundation Hospital were reviewed. The patients treated with deflazacort GC were included. Pain was assessed by the treating rheumatologist using the visual analog scale (VAS, possible score 0-10). Tender joints were those with VAS> 5. The count of compromised joints was compared with inflammatory findings on bone scintigraphy (Figure 1).

Conclusion: In this case series a media dose of deflazacort of 21mg per week (3mg/day) was useful to significantly reduce the number of tender joints in patients with EDPOA.

References:

Disclosure of Interests: None declared

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FR0420 ASSOCIATION BETWEEN RED BLOOD CELLS DISTRIBUTION WIDTH AND CARDIOVASCULAR RISK IN OSTEOARTHRITIS

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Background: Osteoarthritis (OA) is the most common joint disease that results in patient’s morbidity and disabilities. There is strong evidence that OA is a significant risk factor for cardiovascular disease (CVD). Red cell distribution width (RDW) test is a measure of the variation in red blood cell volume and size. Elevated RDW has recently been found to correlate with CVD risk in patients with and without heart disease and autoimmune diseases. RDW may be a marker for factors driving CVS risk.

Objectives: To investigate whether RDW can serve as a potential parameter for indicating cardiovascular risk in OA patients.

Methods: A subsample of 819 OA patients was extracted from 2003-2006 National Health & Nutrition Examination Survey in a cross-sectional study. 63.7% of them were females. Their mean age was 66.4 ± 14.1 yrs. Demographic, medical data, inflammatory markers & lipid panel were obtained. Only patients with Haemoglobin>12mg/dl were included. Functional limitations were assessed using a physical function questionnaire.

Results: Elevated levels of RDW were associated with CVD risk factors in OA patients. 532 (65.8%) OA patients had functional limitations, while 78 (9.5%) and 63 (7.6%) known to have heart attacks or stroke ever. Mean RDW was 12.9 ± 1.1fL. There was a positive significant correlation between RDW & CVD risk factors including body mass index (β=0.26, p<0.001), C-reactive protein (β=0.29, p<0.001), serum uric acid (β=0.12, p<0.001), and functional limitation (β=0.16, p<0.001). No significant association between RDW & lipid panel was found. In multiple regression analysis controlling for age, sex as covariates, body mass index (β =0.02, 95%CI: 0.01, 0.03, p=0.002), C-reactive protein (β =0.35, 95%CI: 0.26, 0.45, p<0.001), and functional limitation (β =0.18, 95%CI: 0.13, 0.35, p=0.03).

Conclusion: In addition to known CVD risk in OA patients, elevated RDW levels should prompt physicians to aggressively screen and treat their patients for modifiable CVS risk factors, in addition to OA.

Disclosure of Interests: None declared

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FR0421 RATES OF PROGRESSION DIFFER BETWEEN STRUCTURAL PHENOTYPES OF KNEE OSTEOARTHRITIS: A SECONDARY ANALYSIS FROM THE FNHI COHORT

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Background: Imaging plays an important role in determining structural disease severity and potential suitability of patients recruited to disease-modifying osteoarthritis drug (DMOAD) trials. It has been suggested that there may be three main structural phenotypes in OA, i.e., inflammation, meniscus/cartilage and subchondral bone. These may progress differently and may represent distinct tissue targets for DMOAD approaches.

Objectives: To stratify the Foundation for National Institutes of Health Osteoarthritis Biomarkers Consortium (FNHI) cohort, a well-defined subsample of the larger Osteoarthritis Initiative (OAI) study, into distinct structural phenotypes
based on semiquantitative MRI assessment and to determine their risk for progression over 48 months.

**Methods:** The FNIH was designed as a case-control study with knees showing either 1) radiographic and pain progression (i.e., “composite” cases), 2) radiographic progression only (“JSL”), 3) pain progression only, and 4) neither radiographic nor pain progression. MRI of both knees was performed on 3 T systems at the four OAI clinical sites. Two musculoskeletal radiologists read the baseline MRIs according to the MOAKS scoring system. Knees were stratified into subchondral bone, meniscus/cartilage and inflammatory phenotypes. A secondary, less stringent definition for inflammatory and meniscus/cartilage phenotype was used for sensitivity analyses. The relation of each phenotype to risk of being in the JSL or composite case group compared to those not having that phenotype was determined using conditional logistic regression. Only KL2 and 3 and those without root tears were included.

**Results:** 485 knees were included. 362 (75%) did not have any phenotype, while 95 (20%) had the bone phenotype, 22 (5%) the cartilage/meniscus phenotype and 19 (4%) the inflammatory phenotype. The bone phenotype was associated with a higher risk of the JSL and composite outcome (OR 1.81 [95% CI 1.14, 2.85] and 1.65; 95% CI [1.04, 2.61]) while the inflammatory phenotype was associated with increased risk of experiencing the composite outcome (OR 0.96 [95% CI 0.38, 2.42] and 1.25; 95% CI [0.48, 3.25]) and the meniscus/cartilage phenotypes were not (OR 1.30 95% CI [0.55, 3.07] and 0.99; 95% CI [0.40, 2.49]).

In sensitivity analyses, the bone phenotype and having two phenotypes (vs. none) were both associated with increased risk of experiencing the composite outcome (bone: OR 1.65; 95% CI 1.04, 2.61; 2 phenotypes: OR 1.87; 95% CI 1.11, 3.16).

**Conclusion:** The bone phenotype was associated with increased risk of having both radiographic and pain progression together, or radiographic progression alone, whereas the inflammatory phenotype or meniscus/cartilage phenotype each individually were not associated with either outcome. Phenotypic stratification appears to provide insights into risk for structural or composite structure plus pain progression, and therefore may be useful to consider when selecting patients for inclusion in clinical trials.

**References:**

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**FR0422 HOW MANY OA PATIENTS WILL BE OVERTREATED IF TREATED WITH DMOADS? ANALYSIS OF OSTEOARTHRITIS INITIATIVE DATA**

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**Background:** Significant research effort is currently put on discovering drugs able to slow structural progression of osteoarthritis (OA). In many patients OA shows little or no increase in pain or function deterioration over time. Thus, giving disease-modifying OA drugs (DMOAD) for long periods to this subset of patients can be considered as overtreatment. There is a lack of studies directly evaluating how many patients with diagnosed OA experience no impact of the disease during long-term follow up and thus probably do not need any disease modification.

**Objectives:** To assess proportions of patients with diagnosed symptomatic knee OA or frequent knee pain that does not result in sustained pain and limitation of function during long-term follow up.

**Methods:** For the current study we used 8-year longitudinal data obtained from the Osteoarthritis Initiative (OAI) progression (>3000) and incidence (n = 3284) subcohorts, which are publically available at https://oai.nih.gov . For the analyses we included knees having frequent knee symptoms in the past 12 months before the baseline for at least one month. Thus the analyzed group comprised patients fulfilling the definition of symptomatic knee OA (frequent symptoms + Kellgren-Lawrence (KL) grade ≥ II) and people who might have early OA (frequent symptoms + KL grade < II) in accordance with the proposed draft classification criteria [1].

The proportion of knees experiencing no impact of OA at the 8 years follow-up was assessed.