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).

Figure 1. Comparison between number of joints with inflammatory findings on bone scintigraphy and number of swollen joints in physical evaluation

The number of tender joints was recorded at the start of treatment, which was a dose of 6mg/day of deflazacort for two months. Subsequently, the dose was reduced depending on the improvement of pain (items: intensity of pain and number of tender joints) until achieving a stabilization along the time with an improvement of 75% of the items evaluated. The number of painful joints was recorded again two months after the stabilization on pain control was achieved.

Quantitative variables were described with medians and interquartile ranges because the absence of normal distribution of the sample size. To assess the presence of a significant decrease on the number of tender joints the Wilcoxon range test was used, a value of $p < 0.001$ was considered statistically significant. The data were analyzed with Stata v.15.

Results: Twenty-eight patients with EDPOA were included, with a median of age of 50 years (IQR 44-51), 56 years (IQR 52-66) and 61 years (IQR 54-69) at the time of menopause, onset of symptoms and the diagnosis of EDPOA respectively. A median of 18 tender joints (IQR 10-27) was obtained from the physical examination of the records reviewed. The dose of deflazacort that achieved stabilization on the improvement of the pain along the time was 21mg/week (IQR 12-21); after 8 weeks of treatment the number of tender joints was 2 (IQR 1-4), which implies a reduction of 14 (IQR 8-20; $p < 0.0001$) on the tender joint count (Figure 2).

Figure 2. Number of tender joints before and after eight weeks of treatment achieving with a stable pain control in patients with EDPOA treated with deflazacort with a media dose of 3mgr/day.

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.

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FRI0420

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) blood 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 > 12 mg/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 ± 1.1 fL. There was a positive significant correlation between RDW & CVD risk factors including body mass index ($r = 0.17$, $p < 0.001$), C-reactive protein ($r = 0.29$, $p < 0.001$), serum uric acid ($r = 0.12$, $p < 0.001$), and functional limitation ($r = 0.16$, $p < 0.001$). No significant association between RDW & lipid panel was found. In multiple regression analysis controlling for age, sex as covariates, body mass index ($\beta = 0.02$, 95%CI: 0.01, 0.03, $p = 0.002$), C-reactive protein ($\beta = 0.35$, 95%CI: 0.26, 0.45, $p < 0.001$), and functional limitation ($\beta = 0.18$, 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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FRI0421

RATES OF PROGRESSION DIFFER BETWEEN STRUCTURAL PHENOTYPES OF KNEE OSTEOARTHRITIS: A SECONDARY ANALYSIS FROM THE FNIH 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 (FNIH) 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 phenotypes¹. 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 (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.

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FRI0422 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 (n= 1390) 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.

No impact of OA was defined if a knee fulfilled all the following criteria: 1. WOMAC pain score within normative values 2. WOMAC disability score within normative values 3. Absence of joint replacement.

We used previously reported reference population age and gender adjusted values for WOMAC knee pain and WOMAC knee function [2]. It is unlikely that any OA treatment is capable to improve WOMAC pain and function measures above these reference ranges in a given person [2].

Results: We included 3092 knees from 2147 participants in the analysis. The mean age of participants was 61.16 years, the mean BMI was 28.59. Almost half of symptomatic knees with baseline KL grades 0-I were not impacted by the disease at 8 year follow up. Every fifth knee with symptomatic knee OA (KL grades ≥ II) had no impact of the disease at the end of follow up. Every third knee with symptomatic KL grade II OA did not develop pain or disability outside the reference range. The percentage of symptom-free knees at year 8 declined progressively with higher KL grades (Table).

Table. Percentage of knees with no impact of OA on 8 year follow up depending on baseline KL grade.

Baseline KL grade	Total n of knees	Percentage of knees with no impact of OA	
0	894	47 %	45.78%
I	469	43.1%	
II	923	31.31%	19%
III	609	17.7%	
IV	197	7.1%	

Conclusion: If given DMOADs, a substantial proportion of OA patients would be overtreated, especially those with early OA.

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FRI0423 CLINICAL BURDEN OF TREATING COMMERCIALY-INSURED OSTEOARTHRITIS PATIENTS WITH PRESCRIPTION NSAIDS

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Background: Prescription NSAIDs/Cox-2s ("NSAIDs") are commonly prescribed by physicians to treat patients with chronic pain, and much is known about the potential negative outcomes associated with their use¹. Such negative outcomes include gastrointestinal ("GI") issues and hepatorenal toxicity.¹ In addition, CV risk of Cox-2s has not been completely clarified.^{1,2} However, less is known about the extent to which these outcomes are pervasive and problematic in specific patient populations such as those diagnosed with osteoarthritis ("OA").

Objectives: The goal of this research is to assess the clinical burden of commercially-insured patients diagnosed with OA of the hip and/or knee before and after treatment with prescription NSAIDs, in a large, national database in recent years.

Methods: The Optum Healthcare Solutions, Inc. data (1/2012-3/2017) were used to identify patients ≥18 years old with ≥2 diagnoses of hip and/or knee OA, and ≥90 days supply of NSAIDs during the three-year period from first prescription (index date) after their first OA diagnosis. Patients were required to be continuously-enrolled during the six months before (baseline period) and 36 months after (follow-up period) the index date. Selected clinical outcomes such as GI issues, CV events, and renal toxicity were compared between the baseline and follow-up periods. Costs and resource use were normalized to account for differential duration in analytic time periods.

Results: Data for 22,435 patients (60.8% as female, with an average age of 62) with hip and/or knee OA were analyzed. On average, patients were prescribed NSAIDs for 489 days during the follow-up period. From the baseline period to follow-up period, negative clinical outcomes associated with GI issues increased by 393% (1.5% v 7.5%), driven by a 667% (0.3% v 2.3%) increase in acute GI hemorrhages. Additionally, negative clinical outcomes