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