Table 1. Demographic, Clinical and Laboratory Features of Patients (n=49)

Female gender, n (%)	35 (71.4)
Mean age at diagnosis, years (mean±SD)	12.5±3.3
Mean age at the time of inclusion, years (mean±SD)	17.7±2.6
Duration of illness, years (median/IQR)	5.7 (3.0-7.0) years
Active system involvement at study, n (%)	
Renal	14 (28.5)
Skin	11 (22.4)
Musculoskeletal	9 (18.3)
Hematologic	9 (18.3)
Neurologic	1 (2.0)
Serositis	1 (2.0)
Laboratory findings (median/minimum-maximum)	
Hemoglobin, gr/dl	12.7 (7.3-18.3)
WBC, /mm ³	6.900 (2.600-26.200)
Platelet, /mm ³	252.000 (39.000-529.000)
Erythrocyte sedimentation rate, mm/hour	10 (1-71)
C-reactive protein, mg/dl	0.1 (0-21.1)
Complement 3, mg/dl	87.6 (25.4-186.0)
Complement 4, mg/dl	15.3 (0-45.8)
Anti-ds DNA, IU/ml	12.8 (0-741.7)
SLEDAI score (mean±SD)	4.69±6.69

Conclusion: Our data supports that Galectin 9 and IL-2R α are good markers for disease activity in childhood SLE. We need larger series to evaluate differences between disease clusters in SLE. We failed to show a significant correlation with checkpoint proteins and SLEDAI except for PDL1.

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The aftermath of metabolic imbalance in OA

OP0196

CIRCULATING LEVEL OF IL-6 IS ASSOCIATED WITH 10.7-YEAR KNEE CARTILAGE VOLUME LOSS AND WORSE PAIN TRAJECTORY

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Background: There is growing evidence that inflammation plays a critical role in osteoarthritis (OA) progression and its symptoms evolution. OA pain is heterogeneous and there are distinct subgroups within OA pain patients. Recently, we identified three homogeneous subgroups following distinct pain trajectories in which metabolic mechanism may be involved. Whether circulating inflammatory markers are associated with long-term knee structural changes on MRI, and whether the association between inflammatory markers and the trajectories we identified differs remain to be clarified.

Objectives: To examine whether inflammatory markers are associated with 10.7-year knee structural changes including knee cartilage volume (CV) and bone

marrow lesions (BMLs), and to assess the associations between inflammatory markers and different pain trajectories.

Methods: This study was conducted as part of a population-based older adult (mean age 63 years, 51% of females) cohort study with 1,099, 875, 768 and 563 participants attending at baseline, and 2.6-, 5.1- and 10.7-year follow-ups. Circulating levels of interleukin (IL)-6, tumour necrosis factor alpha (TNF- α) and high sensitivity C-reactive protein (CRP) were measured at baseline in 193 randomly selected participants. T1-weighted or T2-weighted MRI of the right knee was performed to measure CV and BMLs at baseline and 10.7-year. X-ray was performed to assess radiographic knee osteoarthritis (ROA). The Western Ontario and McMaster Universities Osteoarthritis Index (WOMAC) pain questionnaire was used to measure knee pain at all four visits. Data on demographic, psychological, lifestyle and comorbidities were also collected. Pain trajectories was previously identified using the group-based trajectory modelling. Linear, log-binomial and multi-nominal logistic regression modellings were used for the analyses.

Results: IL-6 was associated with both medial and lateral tibial CV loss (Medial: β =-0.51% per log pg/ml, 95%CI -0.88 to -0.15; Lateral: β =-0.34% per log pg/ml, 95%CI -0.64 to -0.04) after adjusting for age, sex, body mass index, physical activity, comorbidities, and ROA. TNF- α was not associated with either medial or lateral CV loss, but CRP was positively associated with medial tibial CV loss (Medial: β =0.27% per log mg/L, 95%CI 0.04 to 0.49), not lateral CV loss. No inflammatory markers were found to associate with medial and lateral BML size increase. Of 169 participants who had complete data at baseline, 54%, 35% and 11% of participants fell into 'Minimal pain', 'Mild pain' and 'Moderate pain' trajectory group, respectively. In multivariable analysis, IL-6 was associated with an increased risk of being a 'Moderate pain' trajectory (relative risk ratio [RRR]: 4.03, 95%CI 1.34 to 12.13) in comparison with 'Minimal pain' trajectory group. There was no significant association of TNF- α and CRP with trajectory groups.

Conclusion: IL-6 was associated with both medial and lateral tibial CV loss (Medial: β =-0.51% per log ml/pg, 95%CI -0.88 to -0.15; Lateral: β =-0.34% per log ml/pg, 95%CI -0.64 to -0.04) after adjusting for age, sex, body mass index, physical activity, comorbidities, and ROA. TNF- α was not associated with either medial or lateral CV loss, but CRP was positively associated with medial tibial CV loss (Medial: β =0.27% per log ml/pg, 95%CI 0.04 to 0.49), not lateral CV loss. No inflammatory markers were found to associate with medial and lateral BML size increase. Of 169 participants who had complete data at baseline, 54%, 35% and 11% of participants fell into 'Minimal pain', 'Mild pain' and 'Moderate pain' trajectory group, respectively. In multivariable analysis, IL-6 was associated with an increased risk of being a 'Moderate pain' trajectory (relative risk ratio [RRR]: 4.03, 95%CI 1.34 to 12.13) in comparison with 'Minimal pain' trajectory group. There was no significant association of TNF- α and CRP with trajectory groups.

Disclosure of Interests: None declared

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OP0197

EVALUATION OF THE RELATIONSHIP OF LEPTIN WITH THE METABOLIC PHENOTYPE OF KNEE OSTEOARTHRITIS IN THE CONTEXT OF OBESITY THERAPY

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Background: Obesity and components of the metabolic syndrome (MS) play a key role in the pathogenesis of the metabolic phenotype of osteoarthritis (OA). Obesity is a process meta inflammation due to the synthesis of proinflammatory mediators (adipokines and adipocytokines). The severity of OA depends on the presence of obesity, adipokine activity, comorbidity, and the presence of MS. The problem of treatment of metabolic OA and obesity is very relevant, in connection with the obesity pandemic.

Objectives: To evaluate the relationship of leptin with the clinical manifestations of OA and various components of MS in the context of obesity therapy.

Methods: The study included 50 female patients aged 45-65 y.o. with Kellgren-Lawrence stage II-III knee OA and obesity (body mass index (BMI)>30kg/m²). The average age of the patients was 56.5 ± 5.86 years, the average duration of the disease was 7.43 ± 3.93 years. 62% of patients were diagnosed MS. Patients were randomly divided into 2 groups for the treatment of obesity.

Patients from group 1 (n=25) took orlistat 120mg 3 times a day in combination with a hypocaloric diet and exercise for 6 months. Patients from group 2 (n=25) were on a hypocaloric diet in combination with exercise for 6 months. The groups were comparable in clinical parameters. The clinical course of OA was determined by the WOMAC and VAS. Anthropometric data (height, weight) were determined. All patients underwent a laboratory examination at 2 points (initially and after 6 months): a biochemical blood test, leptin was determined by PCR in peripheral blood.

Results: 8% of patients had 1 MS component, 34% - 2 MS components, 34% - 3 MS components, 22% - 4 MS components, and 2% - 5 MS components at the beginning of the study. High leptin levels ($p=0.001$) were determined in patients with more than 3 MS components. The correlation analysis showed direct correlations of high leptin levels with the severity of knee WOMAC pain ($r=0.36$, $p=0.02$) and VAS pain ($r=0.51$, $p=0.01$). A positive correlation was determined between high leptin levels and body weight ($r=0.56$, $p<0.01$) and waist circumference ($r=0.38$, $p<0.01$). Patients from group 1 had a significant decrease of body weight by 10.07% ($p<0.05$), the indicators of the WOMAC index improved: pain decreased by 52.5% ($p<0.05$), stiffness by 47.98% ($p<0.05$), joint functional failure by 51.55% ($p<0.05$) after 6 months of drug therapy for obesity. Patients of group 2 had a not significant decrease of body weight by 0.84% ($p>0.05$), and they were worse indicators of clinical manifestations of OA according to WOMAC compared to group 1 ($p<0.05$). 24% of patients from group 1 showed a decrease in the number of MS components. 12% of patients from group 2 had a decrease in the number of MS components and 12% patients increase in MS components. Patients from 1 group with a significant decrease in body weight, there was a decrease in the level of leptin ($p = 0.05$) (graphic 1), in contrast to patients without weight loss on the background of non-drug therapy ($p = 0.64$). We found direct correlations between a decrease in leptin levels and a decrease in the WOMAC index (pain, stiffness, joint functional failure, total WOMAC) ($r=0.5$, $p=0.01$; $r=0.4$, $p=0.04$; $r=0.4$, $p=0.03$; $r=0.5$, $p=0.01$, respectively). Patients with a decrease in the number of MS components had a significantly lower leptin levels ($p=0.01$).

Graphic 1. Dynamic of leptin.

Conclusion: Leptin is a predictor of a worse course of the metabolic phenotype of OA associated with MS and obesity. High levels of leptin were observed with more MS components, and were associated with the severity of knee pain and body weight. We observed a decrease in the level of leptin, a decrease in the number of MS components, and an improvement in the clinical manifestations of OA against the background of a significant decrease in body weight. Thus, the treatment of obesity in patients with the metabolic phenotype of OA and other interventions aimed at reducing the level of leptin may contribute to reducing the progression of knee OA.

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Monitoring and restoring health: Should we have a core set for multidisciplinary care in rheumatology_

OP0198-HPR

A STRUCTURED MODEL FOR OA CARE IN PRIMARY HEALTHCARE IS A COST-EFFECTIVE ALTERNATIVE COMPARED TO USUAL CARE FOR PEOPLE WITH HIP AND KNEE OA

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Background: To improve quality of care for patients with hip and knee osteoarthritis (OA), a structured model for integrated OA care was developed based on international treatment recommendations. A previous analysis of a cluster RCT (cRCT) showed that compared to usual care, the intervention group reported higher quality of care and greater satisfaction with care. Also, more patients were treated according to international guidelines and fulfilled recommendations for physical activity at the 6-month follow-up.

Objectives: To assess the cost-utility of a structured model for hip or knee OA care.

Methods: A cRCT with stepped-wedge cohort design was conducted in 6 Norwegian municipalities (clusters) in 2015-17. The OA care model was implemented in one cluster at the time by switching from "usual care" to the structured model. The implementation of the model was facilitated by interactive workshops for general practitioners (GPs) and physiotherapists (PTs) with an update on OA treatment recommendations. The GPs explained the OA diagnosis and treatment alternatives, provided pharmacological treatment when appropriate, and suggested referral to physiotherapy. The PT-led patient OA education programme was group-based and lasted 3 hours followed by an 8-12-week individually tailored resistance exercise programme with twice weekly 1-hour supervised group sessions (5-10 patients per PT). An optional 10-hours Healthy Eating Program was available. Participants were ≥ 45 years with symptomatic hip or knee OA. Costs were measured from the healthcare perspective and collected from several sources. Patients self-reported visits in primary healthcare at 3, 6, 9 and 12 months. Secondary healthcare visits and joint surgery data were extracted from the Norwegian Patient Register. The health outcome, quality-adjusted life-year (QALY), was estimated based on the EQ-5D-5L scores at baseline, 3, 6, 9 and 12 months. The result of the cost-utility analysis was reported using the incremental cost-effectiveness ratio (ICER), defined as the incremental costs relative to incremental QALYs (QALYs gained). Based on Norwegian guidelines, the threshold is €27500. Sensitivity analyses were performed using bootstrapping to assess the robustness of reported results and presented in a cost-effectiveness plane (Figure 1).

Results: The 393 patients' mean age was 63 years (SD 9.6) and 74% were women. 109 patients were recruited during control periods (control group), and 284 patients were recruited during interventions periods (intervention group). Only the intervention group had a significant increase in EQ-5D-5L utility scores from baseline to 12 months follow-up (mean change 0.03; 95% CI 0.01, 0.05) with QALYs gained: 0.02 (95% CI -0.08, 0.12). The structured OA model cost approx. €301 p.p. with an additional €50 for the Healthy Eating Program. Total 12 months healthcare cost p.p. was €1281 in the intervention and €3147 in the control group, resulting in an incremental cost of -€1866 (95% CI -3147, -584) p.p. Costs related to surgical procedures had the largest impact on total healthcare costs in both groups. During the 12-months follow-up period, 5% (n=14) in the intervention compared to 12% (n=13) in the control group underwent joint surgery; resulting in a mean surgical procedure cost of €553 p.p. in the intervention as compared to €1624 p.p. in the control group. The ICER was -€93300, indicating that the OA care model resulted in QALYs gained and cost-savings. At a threshold of €27500, it is 99% likely that the OA care model is a cost-effective alternative.