Conclusion: •Gout management by rheumatologists across Wales concurs well with the recent BSR guidelines for most audit standards and showed an improvement in percentage of patients who achieved a target serum uric acid level <300 and <360 $\mu\text{mol/L}$ according to the BSR and EULAR guidelines, respectively.

•Areas for improvement include documentation of Patient Education, improvement of audit tool (Age, Alcohol, current ULT).

•To spread the message to primary care setting, where gout is predominantly managed, to ensure that ULT is optimized to achieve target serum uric acid level to benefit patients.

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Acknowledgements: Dr Martin Bevan for supervising the work and Rheumatology Units across Wales for collecting data and the British Society for Rheumatology in formulating the audit tool.

Disclosure of Interests: None declared.

DOI: 10.1136/annrheumdis-2021-eular.2985

AB0638

CYTOKINE STATUS IN METABOLIC SYNDROME IN PATIENTS WITH GOUTY ARTHRITIS

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Background: Gout is an inflammatory and metabolic disease. Hyperuricemia can contribute to inflammation, hypertension and cardiovascular disease, adipogenesis and lipogenesis, impaired insulin and glucose metabolism, and the development of liver disease. In turn, non-alcoholic fatty liver disease is the most common chronic liver disease worldwide; is closely related to obesity, type 2 diabetes mellitus, dyslipidemia and other metabolic risk factors included in metabolic syndrome (MS). Interest in the problem of MS has not faded for many years, which is associated with its extremely rapid spread in the world.

Objectives: To assess the cytokine status in patients with gouty arthritis (GA) in combination with metabolic syndrome (MS).

Methods: There were 60 patients with reliable GA under observation. Among the surveyed men and women accounted for 60% and 40%, respectively, with an average age of 54 years, an average duration of the disease of 8 years. A family history of gout was present in 42% of patients. The onset of gouty arthritis was observed at 35.6 ± 10 years. Hypouricemic therapy was prescribed in 70% of patients. Thirty-six patients were diagnosed with the tofus form, 24 had no tofus. Patients were included in the study during arthritis remission. Blood samples were taken for general clinical and biochemical analyzes (ESR according to Westergren and uric acid levels were estimated), as well as for determination of serum concentrations of TNF-alpha and IL-6 by enzyme immunoassay.

Results: The patients were divided into two groups: group 1 - 40 people with GA without signs of MS, group 2 - 20 patients with GA and MS. Patient groups were matched by sex, age, form and severity of the disease. In the 1st group, the body mass index ranged from 28.00 to 34.25 kg / m², in the 2nd group - from 29.05 to 49.39 kg / m². In patients with isolated gout, the waist in men averaged 96 cm, in women - 86.5 cm; in the 2nd group: in men - 98 cm, in women - 88 cm. Among the criteria for MS, in addition to abdominal obesity, in the 2nd group, arterial hypertension (in 64%), dyslipidemia (mainly types IIa and IIb) were significantly more frequent, violation of carbohydrate metabolism (fasting glycemic level 8.0 ± 2.0 mmol / l), a higher level of uricemia (from 397.8 to 660.5 mmol / l) compared with the 1st group. The average level of IL-6 in the serum of patients in group 1 was 1.46 pg / ml, in group 2 - 14.03 pg / ml, the average level of TNF-alpha in group 1 was 0.51 pg / ml, in group 2 - 1.28 pg / ml.

Conclusion: In GA patients with signs of MS, there is a significant increase in the production of key proinflammatory cytokines, namely IL-6, TNF-alpha. It was found that with a combination of MS and GA, the concentrations of IL-6, on average, 9.6 times, and TNF-alpha - 2.5 times, exceed the parameters of patients without signs of MS. A direct relationship was established between the expression of IL-6 and TNF-alpha with body mass index, as well as with insulin resistance and fluctuations in blood pressure. Thus, the cytokine imbalance is

associated with increased risks of both cardiometabolic complications and the progressive course of GA.

Disclosure of Interests: None declared.

DOI: 10.1136/annrheumdis-2021-eular.3016

AB0639

EXOSOMES DERIVED FROM ENDOTHELIAL CELLS PREVENT OSTEOBLAST APOPTOSIS IN STEROID-INDUCED OSTONECROSIS OF THE FEMORAL HEAD RAT MODEL VIA THE PI3K/AKT/Bcl-2 SIGNAL PATHWAY

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Background: Osteonecrosis of the femoral head (ONFH) is a common disease caused by many trauma factors and un-trauma factors. Among those un-trauma factors, steroid-induced osteonecrosis of the femoral head (SNFH) accounted for a large proportion and mainly concentrated in young people. SNFH has been reported as an irreversible disease and associated with the damage of blood vessels and the loss balance of bone homeostasis. Circulating endothelial progenitor cells (CEPCs), one part of circulating endothelial cells (CECs), are immature precursor cells with proliferative potential. The damage of vascular endothelial cells in SNFH has been confirmed by many studies, but the changes of CECs and CEPCs in the peripheral blood of patients with SNFH have not been studied yet.

Objectives: The objective of the study is to explore the number of CECs and CEPCs in SNFH patients and normal people and then investigate whether EC-secreted exosomes (EC-exos) could prevent the progression of SNFH in rat model and its mechanism of action.

Methods: We collect peripheral blood of 3 SNFH patients and 3 healthy people and detected the levels of CECs and CEPCs by Flow cytometer. TEM, NTA and western blot was used to characterize the isolated EC-exos. Annexin V-FITC/PI double staining with flow cytometric analysis and western blot were used to evaluate MC3T3-E1 cells apoptosis. CCK-8, scratching experiment and transwell were used to evaluate MC3T3-E1 cells viability and migration ability. Micro-CT and morphological staining were used to evaluate the progress of SNFH in rat model.

Results: Firstly, we found that the number of CECs and CEPCs in the peripheral blood was decreased in SNFH patients than normal people. Then our results indicated that EC-exos could improve the migration, viability and prevent apoptosis of osteoblasts under dexamethasone by activating the PI3K/AKT/Bcl-2 signal pathway in vitro. Finally, our Micro-CT and morphological staining results in SNFH rat model revealed that EC-exos prevented the progression of SNFH.

Conclusion: EC-exos could enhance the cell viability and migration ability of osteoblasts under dexamethasone and play an anti-apoptosis role against steroids-induced osteoblast apoptosis by activating the PI3K/AKT/Bcl-2 signal pathway. EC-exos prevented the progression of SNFH in rat model.

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Disclosure of Interests: None declared.

DOI: 10.1136/annrheumdis-2021-eular.3088

AB0640

MORTALITY IN CALCIUM PYROPHOSPHATE CRYSTAL DEPOSITION DISEASE: PRELIMINARY DATA

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