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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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.

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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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AB0640

MORTALITY IN CALCIUM PYROPHOSPHATE CRYSTAL DEPOSITION DISEASE: PRELIMINARY DATA

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Background: Calcium pyrophosphate crystal deposition disease (CPPD) is associated with a high frequency of comorbidities and vascular calcification [1], but it is unknown whether these factors affect cardiovascular mortality.

Objectives: To study the structure of mortality in patients with CPPD and compare with the structure of mortality in the Russian Federation.

Methods: 217 patients with crystal-verified diagnosis of CPPD (McCarty criteria) were included in the prospective study, aged ≥ 18 years, mean age 59.2 ± 12.6 years, 82 (38%) men and 134 (62%) women. The median follow-up was 3.9 [1.9; 7.2] years, patients were followed up for at least a year. The exclusion criteria are the presence of other rheumatic diseases with arthritis symptoms. The examination of patients included the history taking, assessment of anthropometric parameters, the presence of comorbidity and the following laboratory tests: determination of serum creatinine level (with calculation of glomerular filtration rate (eGFR) using the MDRD formula), total cholesterol (TC), C-reactive protein (CRP). Statistica 12.0 package was used for statistical data processing.

Results: A total of 217 patients were included. Arterial hypertension was detected in 115 (53%) patients, coronary heart disease in 51 (24%) patients, diabetes mellitus in 26 (12%) patients, chronic renal failure in 17 (8%) patients, hyperparathyroidism in 18 (8%) patients, chronic heart failure in 22 (10%) patients. 65 (30%) patients had a family history for CVD, 31 (14%) patients were smokers. 122 (56%) patients had an increased level of total cholesterol > 5.0 mmol/L and 54 (25%) patients – the level of CRP > 5 mg/L.

23 (11%) patients, 12 (52%) men and 11 (48%) women, died, the average age of the deceased being 62.7 ± 9.2 years. In 15 (65%) cases out of 23, death occurred due to cardiovascular complications, which is higher than the cardiovascular mortality rate in the Russian Federation (53%). Among CVD, the distribution was as follows: acute myocardial infarction - 6 (40%) patients, apopleptic attack - 5 (33%) patients, thrombosis - 2 (13%) patients, rhythm disturbances - 1 (7%) patient and decompensation of chronic heart failure - 1 (7%) patient.

Conclusion: CVD is the main cause of death in patients with CPPD and the total frequency of mortality from CVD exceeds the population one. Further research is needed, including studies of the risk factors for overall and cardiovascular mortality in patients with CPPD.

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AB0641

EFFECT OF AGE ON PARATHYROID HORMONE RESPONSE TO VITAMIN D INSUFFICIENCY IN PRIMARY HYPERPARATHYROIDISM

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Background: Primary hyperparathyroidism (PHPT) is a common endocrine condition, commonly seen with increasing age. In vast majority, it is diagnosed incidentally and causes no particular symptoms. Symptoms are usually related to acute hypercalcaemia or the complications of chronically elevated serum calcium level. Vitamin D deficiency is common among general population and in patients with PHPT. Studies in secondary hyperparathyroidism (SHPT) have shown that parathyroid hormone (PTH) response is affected by age, with those over 80 showing greater rise in PTH levels. We wanted to see if age has a similar impact on PTH response to vitamin D in those with PHPT.

Objectives: To evaluate the impact of age on PTH response to vitamin D insufficiency in those with PHPT.

Methods: Patients with primary hyperparathyroidism (PHPT), attending general endocrine clinic of a district general hospital, were divided into two groups based on age; less than 70 (n=73) and 70 and above (n=61). Each group was subdivided into vitamin D insufficient (VDI) and vitamin D sufficient (VDS) subgroups. We compared calcium and parathyroid hormone levels and forearm BMD (presented as T score) in VDI and VDS subgroups in the two age groups, at the time of diagnosis. Data were analyzed using unpaired t-test and presented as mean $\pm$ SEM, using Graphpad Prism 9.0.1.

Results: There was significant difference in Vitamin D levels in VDI and VDS subgroups, in both age groups (<70 ; mean vitamin D 27.98 vs. 68.44, $p < 0.0001$; ≥ 70 ; mean vitamin D 34.44 vs. 75.74, $p < 0.0001$). The two groups were significantly different in terms of age (mean age 58 vs. 76, $p < 0.0001$). Although there was no difference in calcium and forearm BMD in VDI and VDS, in both age groups, those under 70 showed a greater PTH response to vitamin D insufficiency (mean PTH 19.29 vs. 12.91 respectively, $p < 0.001$).

Conclusion: While in SHPT, those with increasing age show greater rise in PTH levels, our data show that in PHPT, younger patients show a greater PTH rise

in response to vitamin D insufficiency. Further work is needed to elucidate the underlying mechanisms.

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AB0642

CLINICAL CHARACTERISTICS AND FACTORS ASSOCIATED WITH BONE EROSION IN GOUT PATIENTS WITH TOPHI

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Background: Bone erosion is a frequent complication of gout patients with tophi and can lead to joint damage, deformity and musculoskeletal disability. Few studies have focused on clinical characteristics and factors associated with bone erosion in gout patients with tophi.

Objectives: The aim of this study was to describe clinical characteristics of bone erosion in gout patients with tophi.

Methods: Bone erosion was detected by X-ray. Gout patients with tophi were divided into bone erosion group and non bone erosion group. The clinical characteristics were recorded. Comparison of clinical characteristics and risk factors for bone erosion were analyzed between two groups. Multivariate logistic regression analysis was conducted.

Results: A total of 171 gout patients with tophi were enrolled, 121 patients with bone erosion and 50 patients without bone erosion. Bone erosion group were older, with prolonged duration with gout and tophi, higher levels of serum creatinine, lower levels of glomerular filtration rate (GFR), C-reactive protein and BMI. In univariate regression analysis, age, gout duration, tophi duration, GFR were associated with bone erosion. In multivariable logistic regression analysis, tophi duration was independently associated with bone erosion.

Conclusion: Gout patients with bone erosion present different clinical characteristics compared with those without bone erosion. Tophi duration was strongly associated with bone erosion in patients with gout.

Table 1. Comparison of clinical characteristics between bone erosion patients and non bone erosion patients.

	Non Bone erosion	Bone erosion	P Value
N(male)	50(47)	121(118)	0.255
Age(year)	45.82 $\pm$ 14.15	53.74 $\pm$ 14.88	0.002
BMI (kg/m ²)	26.01 $\pm$ 4.58	24.18 $\pm$ 4.72	0.027
WBC(10 ⁹ /mL)	9.73 $\pm$ 3.40	11.37 $\pm$ 13.26	0.404
PLT(10 ⁹ /mL)	329.86 $\pm$ 96.22	328.31 $\pm$ 124.02	0.938
HGB(g/L)	86.58 $\pm$ 63.78	102.75 $\pm$ 51.16	0.201
ALT(U/L)	37.74 $\pm$ 26.56	34.26 $\pm$ 35.26	0.561
sUA(umol/L)	540.16 $\pm$ 121.79	539.00 $\pm$ 121.46	0.962
sCr(umol/L)	111.47 $\pm$ 25.26	135.77 $\pm$ 52.43	<0.001
GFR	74.01 $\pm$ 27.94	56.68 $\pm$ 22.84	0.003
(mL/min/1.73m ²)			
ESR(mm/h)	61.78 $\pm$ 37.32	53.08 $\pm$ 36.70	0.181
CRP(mg/L)	60.00 $\pm$ 58.26	36.45 $\pm$ 42.62	0.014
Gout duration (year)	9.22 $\pm$ 5.46	12.63 $\pm$ 7.59	0.001
Tophi duration(year)	3.77 $\pm$ 3.22	6.64 $\pm$ 4.81	0.001
Hypertension, n	17	52	0.277
Diabetes, n	9	12	0.143
Smoking history, n	20	55	0.513
Drinking history, n	14	37	0.737
Ulceration, n	10	35	0.228

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AB0643

IL-1 BLOCKAGE WITH ANAKINRA IN GOUT PATIENTS. SCOPING REVIEW OF THE PUBLISHED LITERATURE.

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