Methods: Prospective, observational, cross-sectional study. Carotid Doppler was performed on patients in the outpatient clinic with a diagnosis of gout from November 2019 to 2020 of the rheumatology service of the Hospital Docente Padre Billini and 360 health controls. Inclusion criteria, patients > 18 years, diagnosis of monosodium urate deposits arthritis according to the ACR/EULAR 2015 classification criteria, carotid Doppler, measurement of the cIMT. Controls without disease, matched by sex and age. The data was analyzed with SPSS V23 for Windows 10.

Results: Of 37 patients with a diagnosis of arthritis due to deposition of monosodium urate crystals, (34) met inclusion criteria, 100% male, 34 healthy controls. Average of 61.5 years. Average of the disease 8.2 years. Distribution 61 (21) interictical gout, 32 (11) chronic tophaceous gout, 0.5% (2) acute gouty arthritis. Comorbidities 67% (23) dyslipidemia, 35% (12) hyperglycemia. 26% (9) presented arterial hypertension. 20% (7) have diabetes mellitus. 58% (20) are alcohol drinkers, 11% (4) smokers. Mean uric acid 8.6 mg/dl, in mg/dl at Doppler, 52% (18) elevated serum creatinine. Carotid Doppler in patients with gout showed a 55% (19) increase in the cIMT > 0.9mm, with a mean of 2.03mm (1.95 SD). Carotid Doppler in healthy controls 17% (6) increased cIMT, mean of 1.9mm (2.2 SD) (P = 0.04). Patients with gout had 29% (10) atheromatous plaques, 17% (6) calcified plaques versus 14% (5) atheromatous plaques, 8% (3) calcified in healthy controls.

Conclusion: Our study showed that half of the patients with gout had increased cIMT compared to the third of the healthy controls. The presence of atheromatous and calcified plaques was mainly associated with dyslipidemia, so we can conclude that the evaluation of the intima-media thickness by carotid Doppler allows it to be a predictor of cardiovascular disease in patients with gout.

REFERENCES:

Disclosure of Interests: None declared. DOI: 10.1136/annrheumdis-2021-eular.27477

Table 1. Characteristics of the Patients with the Rheumatic diseases and asymptomatic hyperuricemia.

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age, years</th>
<th>Male, %</th>
<th>Serum UA, μmol/l</th>
<th>Normalization of UA during the follow-up, n, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>10.1136/annrheumdis-2021-eular.27477</td>
<td>56.3±1.5</td>
<td>40.27</td>
<td>493.3±98.5</td>
<td>242 (28.64)%</td>
</tr>
<tr>
<td>RA</td>
<td>n=32</td>
<td>64.6±12.1</td>
<td>3.74</td>
<td>504.8±107.5</td>
</tr>
<tr>
<td>SpA</td>
<td>n= 149</td>
<td>56.6±12.9</td>
<td>53.69</td>
<td>531.3±94.9</td>
</tr>
<tr>
<td>SpA</td>
<td>n= 107</td>
<td>45.6±15.1</td>
<td>33.43</td>
<td>520.6±86.5</td>
</tr>
<tr>
<td>ESR</td>
<td>n=137</td>
<td>50.3±14.1</td>
<td>20.44</td>
<td>453.6±31.4</td>
</tr>
<tr>
<td>SSc</td>
<td>n= 57</td>
<td>61.0±12.4</td>
<td>22.81</td>
<td>456.2±99.5</td>
</tr>
<tr>
<td>n=43</td>
<td>62.0±10.7</td>
<td>16.28</td>
<td>442.4±107.5</td>
<td>16 (37.21)%</td>
</tr>
</tbody>
</table>

RD – rheumatic disease; RA – rheumatoid arthritis; SpA – psoriatic arthritis; SpA – spondyloarthritis; SLE – systemic lupus erythematosus; SSc – systemic sclerosis; SD – Sjögren’s disease; * – < 0.001 for the differences with RD, RA, PsA, SSc, SD; ** – p < 0.01 for all intergroup differences. RA, PsA and SpA, RA and SpA, RA and PsA, RA and SSc, RA and SD, RA and SLE, RA and ESR, RA and SSc, RA and SD, RA and SLE, RA and ESR, RA and SSc, RA and SD, RA and SLE, RA and ESR, RA and SSc, RA and SD were revealed the interrelations between the level of UA and ESR and RA and CRP (Spearman’s R =0.1, p=0.01), and UA and CRP (Spearman’s R =0.12, p=0.001).

The level of UA in male pts was 5070 [361-940], in female pts 450.0 [361-1010] μmol /l (p<0.0001), in SLE pts with elevated anti-nuclear factor (ANF) UA was 429 [361-940] and with normal-494 [361-973] (p=0.0001). In pts with high and low RD activity UA was 490 [361-940] and 454 [363-1010] μmol/l respectively (p=0.0001). The higher UA level was found in any RA as compared with low in activity of the same RD (p<0.0001 for all the differences).

Normalization of UA was found in 243 (29.6 %) pts, lack of normalization of UA in 434 (52.8 %) of cases, n = 677 , Table 1. ULT received 219 (26.6 %) pts. Normalization of UA without ULT was registered in 16 (1.9 %) of the pts.

Conclusion: ULT is higher and normalize less often in patients with SpA and PsA as compared with RA, SLA, SSc and SD pts. In any of analyzed rheumatic diseases the level of UA is higher in male pts and in pts with high disease activity. REFERENCES:
Conclusion: Gout management by rheumatologists across Wales concords 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 umol/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.

REFERENCES:

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.

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AB0639 EXOSOMES DERIVED FROM ENDOTHELIAL CELLS PREVENT OSTEOSTAB APOPTOSIS IN STEROIDS-INDUCED OSTEONECROSIS 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 ECST3-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 signaling pathway. Finally, we found that EC-exos could improve the migration and viability of osteoblasts under dexamethasone and play an anti-apoptosis role against steroids-induced osteoblast apoptosis by activating the PI3K/AKT/Bcl-2 signal pathway in vitro. Finally, we found that EC-exos could prevent 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.

REFERENCES:

Disclosure of Interests: None declared.

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AB0640 MORTALITY IN CALCIUM PYROPHOSPHATE CRystal DEPOSITION DISEASE: PRELIMINARY DATA

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BACKGROUND: Calcium pyrophosphate dihydrate (CPPD) crystal deposition disease (CPPD), a biologically heterogeneous disease characterized by synovitis, tenosynovitis, osteoarthritis, and erosions of bone and cartilage, is one of the most common causes of late-onset arthropathy. Many patients with CPPD experience significant pain, disability, and reduced quality of life. Although CPPD is not commonly associated with increased risks of both cardiovascular complications and the progressive course of GA.

Disclosure of Interests: None declared.

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AB0641 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, and alongside 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, and alongside the development of liver disease.

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 is present in 42% of gouty arthritis. 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, 24 had no tofus. Patients were included in the study during arthrosis 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 49.39kg/ m2. In patients with isolated gout, the waist in men averaged 98 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 Ila and Ilib) 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 - 1.38 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 cardiovascular complications and the progressive course of GA.