ASYMPTOMATIC HYPERURICEMIA IN INFLAMMATORY RHEUMATIC DISEASES

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Background: Uric acid (UA) is well-known biomarker of cardiovascular risk and inflammation. However, the data about interrelations between asymptomatic hyperuricemia (AHU) and rheumatic diseases (RD) are limited and contradictory [1].

Objectives: to identify the occurrence of AHU in pts with different RD and to evaluate the interrelations between the AHU and clinical features of the RD.

Methods: The study included data from 822 pts with AHU and RD involved in the Saint-Petersburg Register of Pts with AHU in period from the 01Jan2000 to the 01Apr2020. The AHU was defined as the serum level of uric acid (UA) that exceeded 360 μmol/l without signs of gouty arthritis. Pts with the secondary reasons of AHU (an oncologic diseases, late stages of chronic kidney disease, etc.), and inflammatory diseases another than RD were excluded from the study.

Objective: to compare AHU occurrence in patients with different RD.

Results: 34.5% of pts with RD had AHU compared with 26.6% of healthy controls. The AHU prevalence higher in pts with PsA (31.9%) compared with RA (26.9%), SSc (29.0%), SD (30.9%), RA, n=329 64.2±12.1 3.74 504.8±107.5 # 99 (30.09) ## 61.0±12.4 22.81 456.2±99.5 20 (30.09) # 16.28 442.4±107.5 16 (27.21) RA – rheumatic disease; RA — rheumatoid arthritis; PsA — psoriatic arthritis; SpA — spondyloarthritis; SLE — systemic lupus erythematosus; SSc — systemic sclerosis; SD — Sjogren’s disease; * –* p<0.01 for the differences with RD, RA, PsA, SSc, SD; ** –* p<0.01 for all intergroup differences; # — p<0.01 for the differences with RD, RA, SSc, SD. The prevalence of AHU is higher in pts with RD as compared with RA, SLA, SSc and SD pts.

References:

Gout is the most common inflammatory arthritis with both the prevalence and incidence showed significant rise in the UK in recent years [1]. The most frequent reasons for referral from primary care were diagnostic uncertainty 54%, failure to respond to primary care management 28%, and complex comorbidity 25% [2]. From primary care perspective, increased urate level (p=0.001), young age (p=0.009), fewer comorbidities (p=0.039) constituted the most common risk for gout. General Practice consultations in addition to poor compliance to urate lowering treatment ULT (p=0.004) and lower CVS risk scores (p=0.038) these all factors comprised the independent risk factors for Gout flares [3].

Objectives: To compare the management of gout in the rheumatology services in Wales against the 2017 British Society for Rheumatology (BSR) Guidelines.

Methods: A descriptive study over an 8-week period from January to February 2019. Simple Statistical Analysis in calculating the frequency (%).

Results: The audit cohort comprised 62% of chronic gout patients and 38% of acute admissions.

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

- RHEUMATOLOGY UNITS

The Saint-Petersburg Register of Pts with AHU in period from the 01Jan2000 to the 01Apr2020. The AHU was defined as the serum level of uric acid (UA) that exceeded 360 μmol/l without signs of gouty arthritis. Pts with the secondary reasons of AHU (an oncologic diseases, late stages of chronic kidney disease, etc.), and inflammatory diseases another than RD were excluded from the study.

Table 1. Results of the analysis of AHU in pts with different RD.

<table>
<thead>
<tr>
<th>RD</th>
<th>RA</th>
<th>PsA</th>
<th>SSc</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>N</td>
<td>219 (26.6)</td>
<td>149 (18.0)</td>
<td>107 (13.4)</td>
<td>90 (11.1)</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>64.2±12.1</td>
<td>56.6±12.9</td>
<td>56.1±15.1</td>
<td>50.8±14.1</td>
</tr>
<tr>
<td>Median</td>
<td>64</td>
<td>56.6</td>
<td>56.1</td>
<td>50.8</td>
</tr>
<tr>
<td>Age (Mean)</td>
<td>64.2±12.1</td>
<td>56.6±12.9</td>
<td>56.1±15.1</td>
<td>50.8±14.1</td>
</tr>
<tr>
<td>Male, %*</td>
<td>74.9%</td>
<td>72.8%</td>
<td>73.6%</td>
<td>77.8%</td>
</tr>
<tr>
<td>Serum UA, μmol/l (Mean±SD)</td>
<td>504±107</td>
<td>531±94.9</td>
<td>520±86.5</td>
<td>563±191</td>
</tr>
<tr>
<td>Normalization of UA during the follow-up, %</td>
<td>32 (21.44)</td>
<td>31 (21.4)</td>
<td>18 (16.82)</td>
<td>20 (22.22)</td>
</tr>
</tbody>
</table>

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 health centers. Inclusion criteria, patients with RA > 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) intercuticular 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 study 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 1.89mm (2.2 SD) (P = 0.040). 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:

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Objectives: To compare the management of gout in the rheumatology services in Wales against the 2017 British Society for Rheumatology (BSR) Guidelines.

Methods: A descriptive study over an 8-week period from January to February 2019. Simple Statistical Analysis in calculating the frequency (%).

Results: The audit cohort comprised 62% of chronic gout patients and 38% of acute admissions.
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:


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Disclosure of Interests: None declared.
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AB0639 EXOSOMES DERIVED FROM ENDOTHELIAL CELLS PREVENT OSTEOSTABLOBL APOTOPSIS IN STERIOD-INDUCED OSTEONECROSIS OF THE FEMORAL HEAD RAT MODEL VIA THE PISK/AKT/BD-2 SIGNAL PATHWAY

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Background: Osteonecrosis of the femoral head (ONFH) is a common disease caused by many trauma factors and ur-trauma factors. Among those ur-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 prevent the migration, viability and prevent apoptosis of osteoblasts under dexamethasone by activating the PI3K/AKT/BD-2 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/BD-2 signal pathway. EC-exos prevented the progression of SNFH of rat model.

REFERENCES:


Disclosure of Interests: None declared.
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AB0640 MORTALITY IN CALCIUM PYROPHOSPHATE CRYSTAL DEPOSITION DISEASE: PRELIMINARY DATA

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Disclosure of Interests: None declared.