Figure 3: Comparative analysis the changes of PsA signs and symptoms between pre- and post-treatment (n=22). Data were presented as median(range) and calculated and compared with Mann-Whitney *U* test. (A-I) show that low-dose IL-2 treatment provided a significant decrease in 28 tender joint count, DAS28-ESR index, VAS index, PHGA index, DLQI index and HAQ index. **P* < 0.05, ****P* < 0.001.

Results: Notably, the absolute numbers of lymphocyte subpopulations in peripheral blood such as NK, CD4⁺T, Th17 and Tregs in PsA patients were lower than those of healthy controls (*P* < 0.05). The absolute number of Treg was significantly and negatively correlated with the levels of disease indicators, including DAS28, the number of tender joints, visual analogue scale, physician's global assessment, dermatology life quality index, and health assessment questionnaire (*P* < 0.05). After low-dose IL-2 treatment, compared with the baselines, there was a significant increase in the absolute numbers of NK (*P* < 0.05), CD4⁺T (*P* < 0.01), Th17 (*P* < 0.01) and Treg cells (*P* < 0.001). Interestingly, IL-2 markedly raised the level of Tregs in PsA patients even higher than that of healthy donors (*P* < 0.001), leading to re-balance of Th17 and Tregs. Further, low-dose IL-2 treatment rapid reduced the disease activity such as DAS28, the number of tender joints, visual analogue scale, physician's global assessment, dermatology life quality index, and health assessment questionnaire (*P* < 0.05).

Conclusion: Patients with PsA had an imbalance between pro- and anti-inflammatory cells, particularly the reduction of the absolute number of Tregs. Low-dose IL-2 combination treatments restored the decreased number of Treg cells and lowered disease activity indicators of these patients without over-treatment and evaluable side effect. Further studies are needed to evaluate the long-term immunoregulatory ability of IL-2 treatment.

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SAT0371

CARDIOVASCULAR IMPACT OF HYPERURICEMIA IN PATIENTS WITH PSORIATIC ARTHRITIS

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Background: Inflammatory joint diseases (IJD) such as psoriatic arthritis (PsA) have an increased risk of cardiovascular disease (CVD) since inflammation plays a pivotal role in the pathogenesis of coronary artery disease (CAD), heart failure (HF) and atrial fibrillation (Afib)¹. Ischemic heart disease and HF are the main causes of the increased and premature mortality among patients with IJD². Additionally, patients with PsA have a prevalence of hyperuricemia (HUC) of 32%, 3 times greater as compared with the general population, which may be related to increased cell turnover as well as the release of pro-inflammatory cytokines and tumor necrosis factor³. Prolonged exposure to high levels of uric acid (UA) has been shown to result in oxidative stress causing endothelial dysfunction, ionic channel changes, atrial and ventricular remodeling⁴. There is experimental evidence indicating that uric acid stimulates renin-angiotensin-aldosterone system (RAAS), and it is associated with an increase in cardiac tissue xanthine oxidase activity, all of which induce cardiomyocyte hypertrophy, myocardial oxidative stress, interstitial fibrosis and impaired diastolic relaxation⁵.

Objectives: The aim of this study is to assess the correlation of HUC and the clinical expression of CVD in patients with PsA.

Methods: This is a retrospective cohort study using the 2016 National Inpatient Sample (NIS) of adults diagnosed with PsA based on ICD-10 codes, to detect the prevalence of cardiovascular (CV) conditions such as CAD, Afib, and HF with preserved ejection fraction (HFpEF) in patients with concomitant HUC or gout versus age matched controls. Chi square was used for point prevalence and multivariate linear regression adjusted for age, gender, race, CAD, diabetes mellitus, HTN, hyperlipidemia (HLD), smoking, chronic kidney disease (CKD) and Charlson comorbidity index for prevalence odds ratio (POR). We used STATA-15 for statistical analysis.

Results: We identified 37,315 patients with PsA, of whom 2,165 had concomitant HUC or gout (5.80%). Mean age was 61 years, 57% were females. Our results showed that PsA with concomitant HUC or gout compared to PsA without HUC or gout was associated with a higher rate of Afib (17.8% vs 6.1%, *p* < 0.001), CAD (35.1% vs 19.4%, *p* < 0.001) and HFpEF (7.2% vs 3.1%, *p* < 0.001). Furthermore, patients with PsA and HUC/gout appeared to have more risk of developing Afib (POR 1.79; 95%-CI 1.31-2.45; *p* < 0.001) and HFpEF (POR 1.56; 95%-CI 1.08-2.26; *p* = 0.018), compared to patients with normal uric acid after multivariate-adjustment for risk factors. No statistical difference in CAD was identified between the two groups (POR 1.21; 95%-CI 0.94-1.55; *p* = 0.131) after multivariate linear regression adjustment for confounders.

Conclusion: This study showed that HUC is independently associated with CVD, mainly with Afib and HFpEF in patients with PsA. It remains to be seen if a treat to target approach with normalization of UA in patients with PsA will result in improved CV outcomes. We believe that our findings merit further investigation and that this study adds weight to the hypothesis of UA as a potential risk factor for CVD. Prospective studies are needed to establish the role of serum uric acid level as a biomarker or predictor for CVD, including CAD, Afib and HFpEF in patients with PsA.

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