LIFESTYLE MODIFICATION FOR THE PREVENTION OF CARDIOVASCULAR DISEASE IN PATIENTS WITH GOUT

Keywords: Lifestyles, Gout, Cardiovascular disease

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Background: High morbidity and mortality of cardiovascular (CV) disease in patients with gout draw attention to CV risk management in daily clinical practice.

Objectives: To evaluate the effect of a favorable lifestyle on the incident CV events in patients with gout.

Methods: We identified 9,110 patients with gout from UK Biobank Cohort based on the combination of self-report and/or hospital diagnostic codes. Lifestyle behaviors defined by smoking status, physical activity, obesity and diet were categorized into favorable (≥3 healthy factors), intermediate (2 healthy factors), and unfavorable patterns (0-1 healthy factor). CV risk was estimated in participants with and without gout according to serum uric acid (SUA) levels and lifestyle patterns.

Results: Incidence rate of CVD was significantly higher in patients with gout than in participants without gout (11.38 vs 5.49 per 1000 person-year). A positive correlation was observed between SUA level and CV risk in the general population, whereas CV risk was consistently high in gout population, regardless of SUA level. A favorable lifestyle was associated with lower CV risk in both patients with gout and participants without gout. In all categories of SUA levels (<6mg/dL, normal; 6.8-9.9mg/dL, high; ≥9mg/dL, very high), CV risk was significantly lower in gout patients with a favorable lifestyle than in those with an unfavorable lifestyle.

Conclusion: Patients with gout are at high risk of CVD even with SUA levels in the normal range. Lifestyle modification can be an effective and inexpensive therapeutic strategy to prevent CV events in patients with gout.

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QUALITY OF LIFE AND CLINICAL GOUT ASSESSMENT CHANGES IN UNCONTROLLED GOUT PATIENTS UNDERGOING PEGLOTICASE THERAPY AS PART OF THE MIRROR RANDOMIZED CONTROLLED TRIAL

Keywords: Quality of life, Gout, Randomized control trial

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Background: Refractory/uncontrolled gout can severely limit physical function and impact patient quality of life [1, 2]. Unfortunately, limited treatment options exist for patients intolerant of or refractory to oral urate-lowering therapies. Pegloticase can lower serum urate (SU) levels, but patient-reported outcomes (PROs) from this study have not been reported [3].

Methods: We report the prespecified PROs and gout-related clinical measures through Month 12 of pegloticase + MTX/PBO co-therapy in the MIRROR RCT trial participants.

Methods: We collected serum and synovial fluid from 18 pseudogout patients and 12 RA patients with acute flares. We also collected serum from five pseudogout patients after resolving arthritis. We performed a metabolomic analysis of the samples using gas chromatography-mass spectrometry (GC-MS). Metabolites contributing to the differentiation between pseudogout with acute arthritis, pseudogout with resolving arthritis, and RA were determined by analysis of the orthogonal partial least-squares discriminant analysis (OPLS-DA) weightings (variable importance in the projection (VIP)> 1, the absolute value of modeled correlation (|p|corr|) > 0.5 in the S-plot) and Wilcoxon rank sum test (p < 0.05).

Results: A total of 123 metabolites from synovial fluid and 101 metabolites from serum were identified by GC-MS. By using OPLS-DA (Figure 1) and Wilcoxon rank sum test, nine metabolites from synovial fluid and 19 metabolites from serum were selected as the metabolites that contributed to the differentiation between pseudogout and RA, and nine metabolites from serum were selected as the metabolites that contributed to the differentiation between pseudogout with acute arthritis and pseudogout with resolving arthritis. Among the selected metabolites, we identified five metabolites which characterized pseudogout compared with RA and two metabolites which elevated in pseudogout with acute arthritis compared with both RA and pseudogout with resolving arthritis.

Conclusion: By metabolomic analysis, we identified metabolites that differentiate pseudogout from RA and the potential serum diagnostic biomarkers for pseudogout.

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DIAGNOSTIC AND DIFFERENTIAL BIOMARKERS IDENTIFIED BY METABOLIC ANALYSIS IN PATIENTS WITH PSEUDOGOUT

Keywords: Biomarkers, Crystal arthritis, -Omics

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Background: Pseudogout is an acute arthritis induced by calcium pyrophosphate dihydrate crystal deposition (CPPD) [1]. Pseudogout most commonly involves one or several large joints, such as the knee or wrist, typically tender and swollen [2]. The pathogenic mechanism of CPPD is partially understood, and the etiology of attacks is unclear. There is no diagnostic serum biomarker for pseudogout. CPPD disease is best diagnosed by identifying positively birefringent, typically rhomboid-shaped crystals in the synovial fluid of affected joints. However, CPPD disease and rheumatoid arthritis (RA) often coexist, and the similarity of pseudogout with acute flares of RA might lead to misdiagnosis.

Objectives: To identify the metabolites which characterize pseudogout compared with RA and to identify serum biomarkers of pseudogout using metabolomic analysis of serum and synovial fluid.

Methods: We collected serum and synovial fluid from 18 pseudogout patients and 12 RA patients with acute flares. We also collected serum from five pseudogout patients after resolving arthritis. We performed a metabolomic analysis of the samples using gas chromatography-mass spectrometry (GC-MS). Metabolites contributing to the differentiation between pseudogout with acute arthritis, pseudogout with resolving arthritis, and RA were determined by analysis of the orthogonal partial least-squares discriminant analysis (OPLS-DA) weightings (variable importance in the projection (VIP)> 1, the absolute value of modeled correlation (|p|corr|) > 0.5 in the S-plot) and Wilcoxon rank sum test (p < 0.05).

Results: A total of 123 metabolites from synovial fluid and 101 metabolites from serum were identified by GC-MS. By using OPLS-DA (Figure 1) and Wilcoxon rank sum test, nine metabolites from synovial fluid and 19 metabolites from serum were selected as the metabolites that contributed to the differentiation between pseudogout and RA, and nine metabolites from serum were selected as the metabolites that contributed to the differentiation between pseudogout with acute arthritis and pseudogout with resolving arthritis. Among the selected metabolites, we identified five metabolites which characterized pseudogout compared with RA and two metabolites which elevated in pseudogout with acute arthritis compared with both RA and pseudogout with resolving arthritis.

Conclusion: By metabolomic analysis, we identified metabolites that differentiate pseudogout from RA and the potential serum diagnostic biomarkers for pseudogout.

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