Chin Med Sci J
September 2015

Vol. 30, No. 3
P. 199-202

CHINESE
MEDICAL SCIENCES
JOURNAL

SHORT COMMUNICATION

Serum Myeloperoxidase Level in Systemic Lupus Erythematosus

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Key words: myeloperoxidase; systemic lupus erythematosus

Chin Med Sci J 2015; 30(3):199-202

SYSTEMIC lupus erythematosus (SLE) is a systemic autoimmune disease. Several mechanisms have been put forward as underlying the loss of self-tolerance and development of organ dysfunction, such as genetic, environmental, hormonal and immunoregulatory factors.[1] In recent years, oxidative stress is considered as an important factor in the atherogenesis of SLE.[2] Morgen *et al*[3] illustrated elevated levels of protein oxidation in serum from patients with SLE, which may play a role in the pathogenesis of chronic organ damage in SLE.

Myeloperoxidase (MPO), a member of the human peroxidase family, is a heme enzyme stored in and released by activated polymorphonuclear neutrophils (PMNs).[4,5] In the past, experiments of MPO mainly focused on its microbicidal capacity;[6] however, in recent years, major investigations of MPO have been shifted to its non-microbicidal inflammatory processes. For instance, high level of serum MPO has been associated with atherosclerosis,[7] chronic kidney disease,[8] and activated ulcerative colitis.[9]

Received for publication December 9, 2014.

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A few studies have investigated the role of serum MPO level in autoimmune disease. For example, MPO is elevated in patients with rheumatoid arthritis.[10] However, serum MPO level in SLE is still in controversy. Decreased serum MPO level was noted in one study in patients with SLE compared with controls and a trend toward decreased MPO level with increasing disease activity.[3] In contrast, another study showed that patients with SLE presented increased serum MPO level compared with controls, but there was no correlation between plasma MPO level and disease activity,[11] suggesting that MPO may play a role in the inflammatory process of some SLE manifestations.

The purpose of the present study is to determine the serum MPO level in SLE patients, and the possible correlation between MPO level and disease activity of SLE.

PATIENTS AND METHODS

Participants

A total of 196 out-patients with SLE were consecutively enrolled in this study between August 2012 and March 2014 from the Department of Nephrology of Peking University First Hospital. All the patients fulfilled the American College of Rheumatology (ACR) revised criteria for the classification

of SLE.[12] Patients with other autoimmune diseases, infectious disease, diabetes mellitus, leukemia, and malignancy were excluded. Meanwhile, 121 healthy controls were recruited from the Regular Physical Examination Center of Peking University First Hospital during the same period, with the same exclusion criteria as the SLE group. All the participants signed informed consent, and the study was approved by the Ethic Committee of Peking University First Hospital.

Disease activity assessment

Disease activity was assessed based on the SLE Disease Activity Index (SLEDAI) score.[13] Neutrophils, complement 3 (C3), complement 4 (C4), anti-dsDNA antibody were all detected at the time of enrollment into the present study. All the SLE patients were allocated into two groups according to SLEDAI score, Group I (n=73) with inactive disease (SLEDAI=0-4) and Group II (n=123) with active disease (SLEDAI≥5). Group II were further divided into 3 subgroups: the mild active group (SLEDAI=5-9, n=66), the moderate active group (SLEDAI=10-14, n=34) and the severe active group (SLEDAI≥15, n=23). The SLEDAI score of all SLE patients was assessed by a rheumatologist of Peking University First Hospital.

Laboratory indicator measurement

The peripheral blood was collected into serum tube and centrifuged at 3000 rpm for 10 minutes. Serum was cryopreserved in refrigerator at –80°C. MPO was quantified with a commercial ELISA kit (American RB company) according to the manufacturer's instructions, the values were expressed as U/L. Anti-MPO antibody was tested with a commercial ELISA kit (EUROIMMUN Medical Laboratory Diagnostics Stock Company, Germany) according to the manufacturer's instructions, the values were expressed as RU/ml. Avalue≥20 RU/ml was considered as a positive result. Neutrophils were measured by LH-750 (Beckman-Coulter, America). C3 and C4 were detected by rate nephelometry (IMMAGE 800, Beckman-Coulter, America). Anti-dsDNA antibody was detected using a commercial ELISA kit (EUROIMMUN Medical Laboratory Diagnostics Stock Company).

Statistical analysis

Statistical analysis was performed with SPSS version 13.0. The Kolmogorov-Smirnov test showed that all data presented as non-normally distribution, all results were expressed as median and interquartile range (IR). Mann-Whitney U test was used for comparison of quantitative variables between SLE patients and healthy controls, and Kruskal-Wallis test was used for comparison of quantitative variables among the 3 active subgroups. Chi-square test was used for comparison of rates in different groups. Spearman rank correlation test was used to evaluate correlation between serum MPO level and SLEDAI score. Two-sided P values less than 0.05 were considered statistically significant.

RESULTS

The median age of SLE patients and healthy controls were 34 (27-45) and 32 (28-45) years, respectively, with no significant difference (P=0.828). The median course of disease in the SLE patients was 36 (12-108) months. The median serum MPO level in the SLE patients was 305.55 (138.33-591.85) U/L, significantly higher than that in the healthy controls [202.93 (148.04-298.78) U/L, P=0.001, Table 1].

The neutrophils and serum MPO between Group I and Group II were not significantly different; C3, C4 and anti-dsDNA antibody between the 2 groups were significantly different (Table 2). The serum MPO level in the three subgroups of Group II were 291.43 (153.40-583.52), 305.55 (146.84-614.98) and 438.37 (160.75-677.01), respectively, without significant difference (P=0.465).

There was no correlation between serum MPO level and SLEDAI score in SLE patients (r=0.130, P=0.069); serum C3, C4 and anti-dsDNA antibody levels were all correlated with SLEDAI score (r=–0.465, P=0.001; r=–0.356, P=0.001; r=0.329, P=0.001). There was significant correlation between serum MPO level and neutrophils (r=0.462, P=0.001).

Patients with neuropsychiatric involvement had higher serum MPO level than patients without (P=0.044); patients with arthritis involvement had lower serum MPO level than patients without (P=0.033). Patients with or without nephritis (P=0.189), mucocutaneous manifestations (P=0.072) and hematologic abnormalities involvements (P=0.331) had no significant difference in serum MPO levels.

DISCUSSION

Previous research findings regarding the MPO level in SLE patients are controversial, the inconsistency may be due to small sample size. The result of the present study showed higher serum MPO level in SLE patients compared with healthy controls.

MPO act on its substrate hydrogen peroxide and generates a number of reactive oxygen species with documented cytotoxic properties, such as hypochlorous acid