The anti-Ro52 prevalence in the Sjögren’s syndrome picture: a single center cross-sectional study

Larissa Valor, Hannah Schenker, Melanie Hagen, Johannes Knitza, Jürgen Rech, Georg Schett, Friedrich-Alexander-University Erlangen-Nürnberg, Department of Internal Medicine 3 – Rheumatology and Immunology, Erlangen, Germany

Background: Sjögren syndrome (SJS) is an autoimmune disorder characterized by inflammation and destruction of exocrine glands. The presence of autoantibodies (AA) against the Ro52/TRIM21, an RNP complex binding to the stem-loop structure of human cytoplasmic RNA, might be relevant in the SJS pathogenicity. It has been suggested that distinguishing between antibody reactivity against Ro60 and Ro52/TRIM21 could be helpful in terms of evaluating clinical course, features and even pre-symptomatic stages of the disease (1).

Objectives: To evaluate the prevalence of anti-Ro52/TRIM21 antibodies in a cohort of patients diagnosed with primary SJS.

Methods: In this cross-sectional study we evaluated 179 patients with primary SJS according to the ACR classification criteria who had been admitted between December 2008 and December 2018 to our clinic. All patients had an ANA titer higher than 1:320 (2) (in at least two positive determinations for any pattern). ANA, anti-Ro52/TRIM21, anti-Ro60, anti-La and rheumatoid factor (RF) were tested by immunoblot (Euroimmonun, Lübeck, Germany).

Results: In our cohort the median age at diagnosis was 57 years (range: 20-85 years); (6) with a clear dominance of females (n=160, 89%), the most frequently reported ANA patterns were speckled (93%), while only few patients had a homogeneous (6%) pattern. 177/179 were positive for anti-Ro52/TRIM21 (98%), 159/179 (88%) for anti-Ro60, 127/179 for anti-La (79%) and 94/179 (52%) showed RF reactivity. 76/179 (42%) patients showed all four reactivities (anti-Ro52/TRIM21, anti-Ro60, anti-La and RF). Out of these 76 patients, 11 (6%) patients exhibited Raynaud’s syndrome, 25 (13%) exhibited arthritis/arthralgia, 31 (17%) had hypergammoglobulinemia, 13 (7%) had hypocomplementemia and 26% had elevated free kappa/lambda chains, as typical clinical and laboratory features described in SJS.

Conclusion: Our results showed that anti-Ro52/TRIM21 but not anti-Ro60 is present in virtually all patients with SJS and has the most prevalent antibody reactivity. This finding needs to be considered in the current classification criteria of SJS (2), which include the presence of anti-Ro60, rather than anti-Ro52/TRIM21. Also, including the anti-Ro52/TRIM21 measurement in larger cohorts and longitudinal studies would also help us in improving the knowledge of its pathogenic role and to define of more focused diagnostic/therapeutic strategies.

REFERENCES


Disclosure of Interests: Larissa Valor: None declared, Hannah Schenker: None declared, Melanie Hagen: None declared, Johannes Knitza: None declared, Jürgen Rech: Grant/research support from: Bristol-Myers Squibb and Celgene (greater than $10,000). Consultant for: Bristol-Myers Squibb, Celgene, Chugai, GlaxoSmithKline, Janssen, Eli Lilly, Novartis, Roche, Sanofi Aventis, and UCB (in total more than $10,000), Speakers bureau: Bristol-Myers Squibb, Celgene, Chugai, GlaxoSmithKline, Janssen, Eli Lilly, Novartis, Roche, Sanofi Aventis, and UCB (in total more than $10,000), Georg Schett: None declared


SAT0211

Lymphopenia with systemic lupus erythematosus: lymphocyte subsets of pathophysiological features

Nino Yao1, Jie Liang1, Fangyuan Hu1, Chong Gao2, Li Xiaofeng1, Caihong Wang1

1. The Second Hospital of Shanxi Medical University, Taiyuan, China; 2. Brigham and Women’s Hospital, Boston, United States of America

Background: Systemic lupus erythematosus (SLE) is an autoimmune disease involving multiple systems. It is mainly characterized by abnormal activation of T and B lymphocytes and the production of a large number of autoantibodies. Blood system involvement is a common clinical manifestation. The occurrence of lymphopenia is very common, easy to merge infection and primary immunodeficiency. The diagnosis was consistent with the 1997 revised American College of Rheumatology (ACR) SLE classification criteria, and no glucocorticoids and immunosuppressive agents were used. According to the absolute value lymphocytes with SLE, 40 patients were divided into lymphopenia group (LY<10×109/L), and 26 patients were normal lymphocytes group (LN). 30 female healthy controls. The absolute numbers of lymphocyte cell subsets and T subsets (total T, CD4+T, CD8+T, T helper 1 (Th1), T helper 2 (Th2), T helper 17 (Th17) and CD4+CD25+Foxp3+ regulatory (Treg) cells) in peripheral blood were measured by Flow Cytometry (FCM).

Objectives: To study the distribution characteristics of peripheral blood lymphocyte cell subsets in patients with newly diagnosed SLE and to explore the possible mechanism of lymphocyte reduction.

Methods: Sixty-six female patients with initial SLE. The diagnosis was consistent with the 1997 revised American College of Rheumatology (ACR) SLE classification criteria, and no glucocorticoids and immunosuppressive agents were used. According to the absolute value lymphocytes with SLE, 40 patients were divided into lymphopenia group (LY<10×109/L), and 26 patients were normal lymphocytes group (LN). 30 female healthy controls. The absolute numbers of lymphocyte cell subsets and T subsets (total T, CD4+T, CD8+T, T helper 1 (Th1), T helper 2 (Th2), T helper 17 (Th17) and CD4+CD25+Foxp3+ regulatory (Treg) cells) in peripheral blood were measured by Flow Cytometry (FCM).

Results: In the lymphopenia group, the absolute counts of T cell subsets and NK cells were significantly reduced, but the percentage of B cells and Th1 cells and the ratio of Th1/Th2 cells were significantly increased (Figure 1). The absolute number of CD4+ T cells and CD8+ T cells was significantly positively correlated with C3 levels (Figure 2).

Conclusion: Decreased T cell subsets and NK cells may be key pathophysiological features, as risk factor for development of severe infections and primary immunodeficiency in patients lymphopenia with SLE. Meanwhile, decreased CD4+ T cells and CD8+ T cells and increased B cells may cause auto-antibodies production leading to lymphopenia with SLE. Further studies are needed to assess profound lymphopenia and require specific management.

REFERENCES


Disclosure of Interests: None declared