Journal of Clinical and Cellular Immunology

OPEN ACCESS Freely available online

Commentary

A Commentary on *Helicobacter pylori* Infection in SLE, RA, and AS Patients

Asmaa E Hamed*

Department of Rheumatology, Al-Azhar University (Assiut), Al-Azhar University Square, King Faysal, Assiut, Egypt

DESCRIPTION

The etiology of most autoimmune diseases remains elusive. Prevailing evidence suggests an environmental trigger in a genetically susceptible individual. *Helicobacter pylori* (*H. pylori*) have managed to survive in a hostile environment in its host for long period and have evaded eradication by immune system. Its chronic interaction with the immune system and its presence worldwide makes *H. pylori* ideal bacteria to study as a trigger of autoimmune phenomena.

Helicobacter pylori is a widespread, spiral shaped, flagellated Gram-negative bacterium which usually infects the gastric mucosa [1]. Its seropositivity increases with age as it affects about eighty percent of the middle-aged adults in the developing countries and about twenty-five to fifty percent in the western populations [2]. The relation between infections and autoimmune disease has been previously investigated and many Gram-negative bacteria as *Salmonella*, *Shigella* and *Chlamydia* were confirmed to be associated with the development of reactive arthritis [3].These bacteria have been found to stimulate the host immune response due to the presence of lipopolysaccharides and other antigenic molecules that can cause inflammation. It has been previously hypothesized that *H. pylori* can induce such immune host response through many mechanisms as the molecular mimicry and antigenic similarity, the disruption of the tolerogenic immune response, the activation of the polyclonal lymphocytes and the imbalance between T regulatory/Th17 cells in addition to the induction of autoantibody production. The association between *H. Pylori* and auto immune diseases has long been reported including the idiopathic thrombocytopenic purpura chronic idiopathic urticaria auto-immune thyroiditis, autoimmune atrophic gastritis and some other rheumatic autoimmune disorders as rheumatoid arthritis, systemic lupus erythematosus, Sjogren syndrome and ankylosing spondylitis [4].

On the other hand, other studies reported a protective role of *H.Pylori* in some inflammatory conditions as multiple sclerosis, inflammatory bowel disease and allergic conditions as pediatric asthma in the same context, a low seropositivity of *H. pylori* has been reported among Korean patients with HLA-B27 positive acute anterior uveitis [5].

Rheumatoid arthritis, systemic lupus erythematosus and ankylosing spondylitis are the most encountered rheumatic autoimmune diseases sharing the presence of chronic inflammation, auto antibody production and breaking of the self-immune tolerance. However, they are completely different in the clinical and laboratory profiles [6]. Although the precise mechanism of the pathogenesis is still unknown, the genetic and epigenetic events of the host with the environmental and infectious factors resemble the most important triggering elements of the autoimmunity in the three rheumatic diseases. However, there is still a controversy about the pathogenetic role of *H. Pylori* in these rheumatic autoimmune conditions and further studies from different areas are needed to support these hypotheses. In this study, we aimed to evaluate the impact of the presence and activity of H. pylori on the disease activity in RA, SLE and AS. Patients were divided into three groups:

Group A: (RA group)

Twenty RA patients diagnosed according to the American college of rheumatology (ACR)-EULAR RA classification criteria 2010.

Group B: (SLE group)

Twenty SLE patients diagnosed according to the Systemic Lupus International Collaborating Clinics (SLICC) classification criteria.

Group C: (AS group)

Twenty AS patients diagnosed according to modified New York criteria.

Fifty healthy adults' age and sex matching were selected as a control group. Any patient or control known to have treatment for *H.pylori* or taking antibiotic treatment for the previous three months, patients receiving biologic therapy and those known to have diabetes mellitus, autoimmune thyroid disease or autoimmune hepatitis or any other autoimmune disorders were

Correspondence to: Asmaa E. Hamed, Department of Rheumatology, Al-Azhar University (Assiut) 71524, Al-Azhar University Square, King Faysal, Assiut, Egypt, E-mail: Drasmaahamam495@gmail.com

Received: August 16, 2021; **Accepted**: August 30, 2021; **Published**: September 06, 2021

Citation: Hamed AE (2021) A Commentary on *Helicobacter pylori* Infection in SLE, RA, and AS Patients. J Clin Cell Immunol. 12:628.

excluded from the study as well as those subjected to gastrointestinal surgery or endoscopy through the previous three months and those with pregnancy. Complete history taking and thorough clinical examination was done for each patient.

REFERENCES

1. Pestka S, Krause CD, Walter MR. Interferons, interferon-like cytokines, and their receptors. Immunol Rev. 2004;202:8–32.
2. Schindler C, Levy DE, Decker T. JAK-STAT signaling: From interferons to cytokines. J Biol Chem. 2007;282:20059–20063.
3. Donnelly RP, Kotenko SV. Interferon-lambda: A new addition to an old family. J Interferon Cytokine Res. 2010;30:555–564.
4. Bluyssen HA, Blaszczyk K, Olejnik A. STAT2/IRF9 directs a prolonged ISGF3-like transcriptional response and antiviral activity in the absence of STAT1. Am J Physiol Cell Physiol. 2015;299:C354–C362.
5. Liu M, Guo S, Hibbert JM, Jain V, Singh N. Stiles CXCL10/IP-10 in infectious diseases pathogenesis and potential therapeutic implications. Cytokine Growth Factor Rev. 2011;22(3):121–130.
6. Li H, Chen LY. Characteristics, diagnosis and prognosis of acute-on-chronic liver failure in cirrhosis associated to hepatitis B. Sci Rep. 2016;6:25487-25487.