Letter to the Editor *doi: 10.5455/medscience.2014.03.8211*

The Relationship of Systemic Lupus Erythematosus and Helicobacter Pylori

Tulay Yildirim[1], Oguzhan Yildirim[2]

[1] Department of Physical Medicine and Rehabilitation, Namik Kemal University Faculty of Medicine, Tekirdag, Turkey.
[2] Department of Gastroenterology, Namik Kemal University Faculty of Medicine, Tekirdag, Turkey.

Systemic lupus erythematosus (SLE) is an inflammatory disease of unknown etiology. In the pathogenesis of SLE, several infectious agents have been held responsible such as cytomegalovirus, parvovirus B19 and Epstein Barr virus (EBV). There is a variable relationship between SLE and helicobacter, which is different from that of lupus and other infections. We would like to emphasize the interrelation between these entities.

(Rec.Date: Nov 11, 2014 Accept Date: Nov 14, 2014)

Corresponding Author: Tulay Yildirim, Department of Physical Medicine and Rehabilitation, Namik Kemal University Faculty of Medicine, Tekirdag, Turkey.
E-mail: drtulayoner@hotmail.com

The Relationship of Systemic Lupus Erythematosus and Helicobacter Pylori

Systemic lupus erythematosus (SLE) is an inflammatory disease of unknown etiology. In the pathogenesis of SLE, several infectious agents have been held responsible such as cytomegalovirus, parvovirus B19 and Epstein Barr virus (EBV) [1,2]. There is a variable relationship between SLE and helicobacter, which is different from that of lupus and other infections.

The case is here reported of a 28-year old female patient with a 2-year history of SLE, who was being monitored with the disease under control with 10mg prednisolone. The patient presented with complaints of epigastric pain and abdominal swelling which had been ongoing for 1 month. It was learned that the patient used empirical proton pump inhibitors (PPI) but could not obtain a sufficient response. In the physical examination, apart from epigastric sensitivity, there were no pathological findings. In the peripheral blood count, the haemogram and biochemical laboratory test values were evaluated as within the normal ranges. No pathological finding was observed on abdominal ultrasonography. In the upper gastrointestinal endoscopy, antral gastritis was observed including scattered mucosal erosions. In the histopathological examination of the biopsy taken from the antrum and corpus mucosa, chronic active gastritis was found, consistent with *Helicobacter Pylori* (H. Pylori) negative. The steroid treatment was halted, treatment with PPI and sucralfate was started and the complaints were seen to diminish. As a result, H. Pylori infection was not encountered in the SLE patient and no relationship was determined with autoimmunity.

A defined relationship between infection and autoimmunity has been increasingly reported in the last 20 years. Autoimmune diseases are characterized by impairments in the immune system when tolerance against their own antigens is lost. Although the etiology of these diseases is not fully known, it is thought that agents associated with location and environmental factors may play a role. Environmental agents of several pathogens have been observed to trigger autoimmunity in sensitive people [3].

When microbial agents commonly found in the environment are considered and their relationship with the immune system, they can be thought to trigger autoimmunity. The molecular mimicking of micro-organisms causing the loss of self-tolerance is present in several mechanisms. Sharing the amino acid chains of the host cell with microbial antigens

starts the immune response to host proteins and microbial antigens [4]. Other mechanisms thought to trigger autoimmunity are polyclonal activation, epitope spread, bystander activation and super antigens [3]. It has been suggested that various bacteria and viruses trigger autoimmunity [5]. It has been suggested that H. Pylori, which is one of the most commonly encountered infectious agents, triggers autoimmunity. This has been put forward due to the long life of H. Pylori in the host, global widespread prevalence and a complex relationship with the host immune system [6].

H. Pylori and SLE

Published reports show variations in the prevalence of H. Pylori in SLE patients. Anti-H. Pylori antibodies have been found to have the highest prevalence in SLE and undifferentiated connective tissue diseases [7]. Another study has shown that giant cell arteritis, SSc and PBC increased the prevalence of anti-H. Pylori antibodies [8]. In an early study, a negative relationship was determined between H. Pylori seropositivity and the development of SLE in Afro-American females [9]. In another study anti-H. Pylori antibodies were found at low titers in SLE patients compared to other autoimmune diseases [10]. In the most recent study, it was concluded that there is generally a negative relationship of various infectious agents, including H. Pylori, in the development of SLE [11]. In an animal study, H. Pylori exposed to urease enzyme was found to lead to the formation of anti-ssDNA antibodies [12]. Generally, the existing evidence does not support that H. Pylori has a role in the development of SLE [13]. All these studies show a negative relationship between SLE and H. Pylori.

References

1. Pordeus V, Szyper-Kravitz M, Levy RA, Vaz NM, Shoenfeld Y. Infections and autoimmunity: a panorama. Clin Rev Allergy Immunol. 2008;34(3):283-99.
2. Poole BD, Templeton AK, Guthridge JM, Brown EJ, Harley JB, James JA. Aberrant Epstein-Barr viral infection in systemic lupus erythematosus. Autoimmun Rev. 2009;8(4):337-42.
3. Amital H, Govoni M, Maya R, Meroni PL, Ori B, Shoenfeld Y, Tincani A, Trotta F, Sarzi-Puttini P, Atzeni F. Role of infectious agents in systemic rheumatic diseases. Clin Exp Rheumatol. 2008;26(1 Suppl 48):S27-32.
4. Cooke A, Ferraccioli GF, Herrmann M, Romani L, Schulze C, Zampieri S, Doria A. Induction and protection of autoimmune rheumatic diseases. The role of infections Clin Exp Rheumatol. 2008;26(1 Suppl 48):S1-7.

5. Chervonsky AV. Influence of microbial environment on autoimmunity. Nat Immunol. 2010;11(1):28-35.
6. Cover TL, Blaser MJ. Helicobacter Pylori in health and disease. Gastroenterology. 2009;136(6):1863-73.
7. Kalabay L, Fekete B, Czirják L, Horváth L, Daha MR, Veres A, Fónyad G, Horváth A, Viczián A, Singh M, Hoffer I, Füst G, Romics L, Prohászka Z. Helicobacter pylori infection in connective tissue disorders is associated with high levels of antibodies to mycobacterial hsp65 but not to human hsp60. Helicobacter. 2002;7(4):250-6.
8. Shapira Y, Agmon-Levin N, Renaudineau Y, Porat-Katz BS, Barzilai O, Ram M, Youinou P, Shoenfeld Y. Serum markers of infections in patients with primary biliary cirrhosis: evidence of infection burden. Exp Mol Pathol. 2012;93(3):386-90.
9. Sawalha AH, Schmid WR, Binder SR, Bacino DK, Harley JB. Association between systemic lupus erythematosus and Helicobacter pylori seronegativity. J Rheumatol. 2004;31(8):1546-50.
10. Showji Y, Nozawa R, Sato K, Suzuki H. Seroprevalence of Helicobacter pylori infection in patients with connective tissue diseases. Microbiol Immunol. 1996;40(7):499-503.
11. Francis L, Perl A. Infection in systemic lupus erythematosus: friend or foe? Int J Clin Rheumtol. 2010;5(1):59-74.
12. Yamanishi S, Iizumi T, Watanabe E, Shimizu M, Kamiya S, Nagata K, Kumagai Y, Fukunaga Y, Takahashi H. Implications for induction of autoimmunity via activation of B-1 cells by Helicobacter pylori urease. Infect Immun. 2006;74(1):248-56.
13. Matsukawa Y. Association between systemic lupus erythematosus and Helicobacter pylori. J Rheumatol. 2005;32(5):965.