Taenia Solium in a Patient with Systemic Lupus Erythematosus: Do Parasites Protect against Autoimmune Diseases

Lior Zeller MD[1,3,5], Leonid Barski MD[1,5], Elena Shleyfer MD[1,5], Uri Netz MD[4,5], Vered Stavi MD[1,5] and Mahmoud Abu-Shakra MD[2,3,5]

[1]Department of Medicine F, [2]Department of Medicine D, [3]Autoimmune Rheumatic Diseases Unit, and [4]Department of Surgery A, Soroka University Medical Center and [5]Faculty of Health Sciences, Ben-Gurion University of the Negev, Beer Sheva, Israel

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For Editorial see page 249

Systemic lupus erythematosus (SLE) is an autoimmune rheumatic disease of unclear etiology, characterized by the production of pathogenic autoantibody and immune mediated tissue damage [1]. Viral, bacterial or parasitic infection of subjects with a specific genetic background, immune abnormalities, or hormonal constellation may trigger autoimmunity that leads to SLE. Parasites may trigger the generation of autoantibodies and autoreactive T cells; however, the homology between self and parasitic antigens may enable parasites to protect themselves from the immune system and induce a state of immunosuppression. According to the "Hygiene Hypothesis," parasites can regulate the immune system of their host [2]. We describe here a patient with a new onset of SLE and concurrent infection with the platyhelminth *Taenia solium.*

PATIENT DESCRIPTION

A 35 year old agricultural worker of Thai origin was admitted due to an acute confusional state with psychotic features. Three weeks earlier he had been hospitalized due to a swollen cervical lymph node and low grade fever. Biopsy from the node was consistent with advanced immunosuppression (lymphoid depletion and isolated residual regressing germinal centers). Direct stain and culture from the lymph node ruled out tuberculosis.

On examination, the patient was confused and had visual hallucinations. There was temporal wasting and marked alopecia. His temperature was 36.1°C, heart rate 114 beats/minute and blood pressure 156/76 mmHg. Examination of the lung and heart was unremarkable. The abdomen was soft but diffusely tender. Hemoglobin concentration was 8.3 g/dl, leukocyte count 4300 mm^3, and absolute lymphocyte count 640 mm^3. Platelet count was 433,000 mm^3. Serum electrolyte, aspartate aminotransferase, alanine aminotransferase, lipase, amylase and alkaline phosphatase levels were all within normal range. The creatinine level was 0.76 mg/dl and the urea level 81 mg/dl. Protein to creatinine ratio in the urine was 1036 mg/g. Both a head computed tomography (CT) scan and lumber puncture examination were normal. Serology for human immunodeficiency virus, hepatitis B and C viruses, Epstein-Barr virus, and cytomegalovirus were negative. Antinuclear antibody was detected at a titer of 1:160 and with a spackled pattern. The complement C3 level was 22 mg/dl (normal range 90–180 mg/dl), C4 was 2 mg/dl (normal range 10–40 mg/dl). SSA, SSB, and ribosomal P antibodies were all positive. Anti-double strand DNA was not detected.

The patient was diagnosed with SLE according to the following SLICC criteria: positive antinuclear antibody, psychosis, alopecia, marked proteinuria, lymphopenia, and low complement levels. Steroid pulse therapy was instituted together with hydroxychloroquine 200 mg twice a day.

Five days after starting steroid therapy the patient complained of severe abdominal

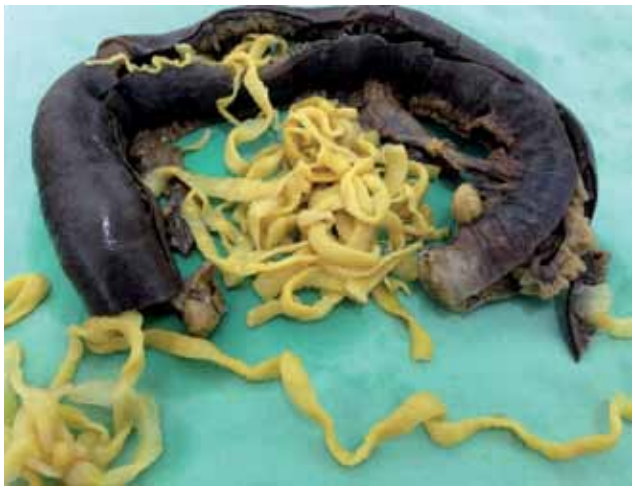

Figure 1. Resected small bowel with massive infestation by platyhelminthes

pain. An abdominal CT scan demonstrated small bowel obstruction. Surgical exploration showed a 70 cm section of necrotic tissue in the small bowel. Histological and microbiological studies demonstrated massive infestation by platyhelminths identified as *Taenia solium* [Figure 1]

COMMENT

Helminths are long-lived parasites that usually do not replicate in a human host. Therefore, the helminth survival strategy is based on immunomodulation. Immunomodulation by helminths is thought to be mutually beneficial for host and parasite since it protects the host from the severe consequences of inflammatory response, and prevents elimination of helminths. The helminths' immunomodulatory effects include interfering with maturation of dendritic cells, which affects the presentation of antigen and changes the direction of the immune response with preference to Th2 or Th3 pathways [3]. In addition, helminths affect T regulatory cells (Tregs) by induction of CD4+CD25+Foxp3+ Tregs, which results in suppression of autoimmunity [4]. This may explain the protective effects of helminthic infection against certain autoimmune diseases such as multiple sclerosis and inflammatory bowel disease [5].

Our patient represents the reverse of the "Hygiene Hypothesis." He presented with severe SLE associated with massive invasion of helminths to the small intestine. It is possible that the "Hygiene Hypothesis" does not apply to patients with SLE; however, it is plausible that the helminth invasion retarded the development of autoimmunity, and our patient would likely have presented with SLE at a younger age.

To the best of our knowledge this is the first report in the medical literature of a patient who presents simultaneously with new onset of SLE and a helminth infection causing bowel obstruction. Further research on the complex interaction between these two diseases and their impact on the immune system is needed.

Correspondence
Dr. L. Zeller
Dept. of Medicine F, Soroka University Hospital, P.O.B 151, Beer Sheva 84101, Israel
Phone: (972-80 640-3015
Fax: (972-8) 640-0097
email: zellerl@bgu.ac.il

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