Mannose-binding lectin levels in endometriosis

The serum concentrations of mannose-binding lectin in patients with or without endometriosis do not differ. Mannose-binding lectin could be involved in the modulation of inflammatory responses, but it does not seem to take part in endometriosis pathogenesis. (Fertil Steril® 2010;94:775–6. ©2010 by American Society for Reproductive Medicine.)

Endometriosis is one of the most common gynecologic diseases in women of reproductive age and is frequently associated with infertility and pelvic pain. The disease is characterized by an abnormal development of endometrial tissue outside the uterus and is associated with an aberrant immunoinflammatory process that takes place in ectopic sites where endometrial tissue abnormally implants.

Mannose-binding lectin (MBL) is an important constituent of innate immunity. It was first observed as an opsonic defect in serum from children with frequent, unexplained infections. The cause of the opsonic defect was discovered 20 years later to be MBL deficiency (1). Subsequently, a decrease in MBL levels has been suggested to be associated with susceptibility to autoimmune disease. One theory of the pathogenesis of endometriosis is that it is an autoimmune disorder, and decreased levels of MBL might be associated with endometriosis, considering the apoptosis of the endometriotic cells in the peritoneal cavity. The aim of the present study was to determine the serum concentration of MBL in women with and without endometriosis. We hypothesized that patients with endometriosis have decreased levels of MBL.

The women recruited in this study provided informed consent for a research protocol approved by the Ethics Board for Human Research of Uludağ University. The study group (n = 32) comprised women with endometriosis identified during laparoscopy. The control group (n = 32) included normal women who were fertile, requesting tubal ligation, and exhibiting no visible evidence of endometriosis upon laparoscopy. Serum samples were collected from the patients during the operation. Enzyme-linked immunosorbent assay was used to quantify MBL concentrations in serum by means of an ELISA for human MBL (Cell Sciences Canton, MA). The minimum and maximum measurable concentrations by the lectin assay were 29.44 ng/mL and 7,218.41 ng/mL, respectively. No statistically difference was found between the groups (women with endometriosis: 2,975.78 ± 2,050.78 ng/mL; controls: 2,928.67 ± 1,988.73 ng/mL; $P>.05$). The serum concentrations of both groups are seen in Table 1.

TABLE 1

MBL concentrations in women with endometriosis and controls, according to menstrual phase.

	MBL concentration (ng/mL)		
Menstrual phase	Endometriosis	Control	*P* value
Proliferative phase	3,987.95 ± 2,136.27 (18)	2,923.20 ± 1,922.81 (24)	NS
Secretory phase	2,465.52 ± 1,867.27 (14)	3,556.54 ± 2,311.34 (8)	NS
Overall	2,975.78 ± 2,050.78 (32)	2,928.67 ± 1,988.73 (32)	NS

Note: Values are means ± SD. Numbers in parentheses are the total number of cases. NS = not significant.

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Recently published articles emphasize the importance of MBL levels and suggest MBL to be associated with susceptibility to autoimmune disease (2). This is the second study in the literature about the relationship between MBL levels and endometriosis. The first study assessed the concentration of MBL in peritoneal fluid of women with endometriosis and did not find any relationship (3). We have also found no statistical difference between endometriosis and control groups in terms of serum MBL levels. This may be attributable to systemic inflammation due to different kinds of additive factors like drugs, infectious diseases, or systemic autoimmune disorders.

This study can be improved by strict selection criteria with more patients. We believe that MBL is involved in the modulation of inflammatory response, but it does not seem to take part in the pathogenesis of endometriosis.

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