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LETTER TO THE EDITOR

The Relationship Between Microorganisms That Cause Pelvic Inflammatory Disease and Serum Cortisol Level

Pelvic inflammatory disease (PID) is a wide spectrum of inflammatory diseases including endometritis, salpingitis, oophoritis, tubo-ovarian abscess, and pelvic peritonitis. The causative microorganisms spread from the vagina or cervix to the upper genital structures via an ascending route (1). It may be asymptomatic or may present with mild or serious clinical symptoms. Based on stress intervals, there is a connection between normal flora, pro-inflammatory processes, and cytokines in cortisol levels (2). It is a cause of many morbidities and mortality such as infertility, subfertility, abscess formation, and the risk of sepsis.

Neisseria gonorrhoeae and C. trachomatis are the most common causes, and other cervical microorganisms, including Mycoplasma genitalium, are also thought to contribute to the disease. In addition, pathogens responsible for bacterial vaginosis (Peptostreptococcus species, Bacteroides species), respiratory pathogens (Haemophilus influenzae, Streptococcus pneumoniae, Staphylococcus aureus), and enteric pathogens (Escherichia coli, Bacteroides fragilis, Streptococcus agalactiae) have been associated with acute PID (3).

The microorganisms that make up the vaginal flora vary from puberty to menopause, and there is a balance between these microorganisms and the vagina. While the vaginal environment controls the flora microorganisms, these microorganisms also regulate the vaginal environment. Many aerobic and anaerobic microorganisms, along with lactobacilli, are found in the vaginal flora without causing disease, and they are protective against sexually transmitted diseases, especially bacterial vaginosis, fungal infections, and urinary tract infections (4).

Rather than behavioral changes linked to stress, the impact of stress on the development of BV may be mediated by immune function dysregulation brought on by stress. The best possible immune response is necessary to stop the spread of anaerobes linked to BV. An inadequate reaction, possibly resulting from genetic variations, raises the danger of infection (5).

Stress enhances the progression of infection (including BV) and its pathophysiologic consequences (6).

Cortisol produced by stress binds to glucocorticoid receptors on a variety of immune cells and modifies NF-κB activities, which control the release of inflammatory mediators such as chemokines (IL-8, CCL5) and cytokines (IL-1β, IL-6, TNF-α, and IFNγ). Additionally, by preventing lymphocyte and leukocyte proliferation, migration, and cytotoxicity as well as the release of IL-2 and IFNγ, glucocorticoids aid in immunosuppression (7,8).

A stress-induced dysbiosis of the vaginal mucosa characterized by disturbed immune response-related and vaginal mucosal proteins (such as lactoferrin), decreased neutrophil bactericidal power, and decreased commensal abundance of Lactobacillus have been shown in a study using mice (9). There is a need for further studies.

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