Oxidative Damage and Mitochondrial DNA Mutations with Endometriosis

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摘要

Abstract

Endometriosis, a frequently encountered disease in gynecology, is a considerable threat to the physical, psychological, and social integrity of women. Moreover, up to 50% of infertile patients have this disease. The etiology and pathogenesis of this important disease are poorly understood; it is defined as an ectopic location for endometrium-like glandular epithelium and stroma outside of the uterine cavity. It still remains an open question as to what extent the peritoneal environment influences the establishment and/or progression of endometriosis. As a result of such stress, a sterile, inflammatory reaction with the secretion of growth factors, cytokines, and chemokines is generated, which is especially deleterious to successful reproduction. Significantly higher amounts of oxidative damage were detected in endometriotic lesions than in controlled normal endometrium, including mitochondrial DNA (mtDNA) rearrangement, 8-OH-deoxyguanosine (8-OH-dG), and lipoperoxide contents. There were approximately sixfold increases in 8-OH-dG and lipoperoxides in chocolate cysts compared with normal endometrial tissues. A novel 5,335-bp deletion of mtDNA was identified in endometriotic tissue. According to these results, we propose that oxidative stress and mtDNA mutations might be anticipated in the initiation or progression of endometriosis. Only by understanding the mechanisms involved in the pathogenesis of endometriosis can we develop a basis for new diagnostic and therapeutic approaches.