عنوان مقاله:

Macrophage apoptosis in Endometriosis: A role for Nitric oxide and endoplasmic reticulum stress

محل انتشار:

سومین کنگره بین‌المللی تولیدمثل (سال: 1396)

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خلاصه مقاله:

Background: Endometriosis, a common gynecologic disorder associated with infertility and pelvic pain, is characterized by the presence of endometrial tissue outside the uterus. A proinflammatory and prooxidant environment has been implicated in the etiopathogenesis of endometriosis. We, herein, offer a hypothesis that ER stress-induced macrophage apoptosis in the setting of excessive NO may contribute to an increased resistance of endometrial cell to macrophage-mediated cytolysis in the peritoneal cavity. Methods: This systematic review assessed related and newest articles from 2000 to 2017.Result: Iron overload in macrophages induces oxidative stress and exaggerates chronic inflammation, implying its causative role in the development and progression of endometriosis. NO regulates a variety of physiologic processes; however, excessive production of NO and NO-derived reactive nitrogen species result in protein misfolding within the endoplasmic reticulum (ER) and increased leakage of sarcoplasmic reticulum Ca2+, eliciting ER stress. Excessive and/or prolonged ER stress triggers apoptosis induced by persistent oxidative stress and protein misfolding. Accumulating evidence demonstrates that ER stress-induced apoptosis is involved in the pathogenesis of neurodegenerative diseases, vascular diseases including atherosclerosis, ischemia/reperfusion, heart failure, and diabetes mellitus. Of note, a deregulated NO production and ER stress has been implicated in the pathophysiology of both preeclampsia and intrauterine growth restriction (IUGR).Conclusion: Important roles for ER-initiated apoptosis pathways have been recognized for several diseases. Therefore, the growing recognition of an association of ER stress with human disease and better understanding of the fundamental mechanisms regulating ER stress may emerge novel therapeutic strategies designed to boost inflammation resolution .in productive disorders

کلمات کلیدی:

Apoptosis, Endoplasmic Reticulum Stress, Iron Overload, Macrophage, Nitric Oxide, Endometriosis