Endometriosis and Oxidative Stress

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Endometriosis: an overview

Endometriosis is a chronic pelvic inflammatory disease characterized by the presence of endometrial tissue outside the uterine cavity such as pelvic peritoneum, ovaries and the recto-vaginal septum and rarely in pericardium, pleura, and brain. The major symptoms of the disease are pelvic pain and infertility. In order to describe the pathophysiology of endometriosis, several theories have been suggested that none of them can explain the pathophysiology of disease in all its aspects but the most commonly accepted theory is Sampson's theory that introduced retrograde menstruation as origin of endometriosis.

Oxidative stress: definition

Oxidative stress occurs as a consequence of an imbalance between oxidant and antioxidant agents. Reactive oxygen species (ROS) are molecules that have unpaired electrons and can react with different molecules like lipids, nucleic acids and proteins. Antioxidants are a defense mechanism against ROS for redox status establishment in physiologic range. So, ROS overproduction or reduction in antioxidant capacity result is OS and its deleterious effects.

Endometriosis and oxidative stress

In serum and peritoneal fluid of women with endometriosis, markers of oxidative stress have been reported elevated. 8-hydroxy 1-deoxyguanosine an OS marker, were seen to be higher in patients with endometriosis than in patients with tubal, male factor, or idiopathic infertility. An increase in 8-hydroxy 1- deoxyguanosine and lipid peroxide levels was demonstrated in ovarian endometriomas. Women with endometriosis showed increased systemic OS expressed by higher levels of heat shock protein 70bo, which indicated that OS is not limit to peritoneal cavity. In peritoneal fluid of women with endometriosis has been reported elevated ROS generation by activated peritoneal macrophages. Many macrophages are present in endometriotic lesions and elevated macrophages number and inflammatory activity (release of pro-inflammatory cytokines) are major causes of increased OS and this oxidative environment can promotes growth of ectopic endometrium.

Ngo et al found that endometriotic cells have an altered phenotype of ROS production leading to an increase in the proliferative capabilities of the cells that favors the spreading of the disease. They can demonstrate that increased production of endogenous ROS is associated with an increase in the proliferation rate of endometriotic cells as in tumor cells.