Role of Peritoneal Fluids in Endometriosis

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Introduction:
Endometriosis is a disease in which endometrial glands and stroma implant and grow in areas outside the uterus. The most common place to find implants is in the peritoneal cavity, but they can be found in any other place. The location and inflammatory response to these lesions are believed to play a key role in symptoms and signs associated with endometriosis.

Main theories that explain the etiology of endometriosis: 1) Sampson's theory of transplantation and implantation, through their fallopian tubes into the peritoneum during menstruation (Sampson JA, 1927), 2) Meyer's theory, suggests that metaplasia of the coelomic epithelium is the origin of endometriosis (Meyer R, 1919), and 3) Halban's theory; suggests that distant lesions are established by the hematogenous or lymphogenous spread of endometrial cells.

Peritoneum and peritoneal fluids:
Is the most extensive serous membrane in the human body, and consists of two layers

1. Loose connective tissue; such as collagen.
2. Mesothelial layer: mainly squamous cells (origin of CA 125).

Peritoneal fluids arises from plasma and ovarian oxidates, with average volume of 5-20 ml. The peritoneal fluid (PF) is a dynamic environment that links the reproductive and immune systems. It appears likely that endometriotic tissue is influenced by this environment and in turn, the PF is altered by the presence of endometriosis. The result is a mixture rich in cells that can assist the growth and maintenance of endometrial implants as well as inhibition of fertility (Vinatier D, 1996).

Role of peritoneal fluid’s cells in endometriosis:
Many cells present in the peritoneal fluids and appear to play a role in endometriosis:

1. Lymphokines: may play a role in endometriosis-associated infertility. Lymphokines are substances whose function involves the regulation of proliferation and differentiation of lymphocytes, and are protein products of stimulated macrophages. Lymphokines stimulate monocytes from the bloodstream to enter the peritoneal cavity and differentiate into activated macrophages.

2. Macrophages: As peritoneal fluid washes the peritoneal and ovarian surfaces, on which most endometriotic lesions occur. The high numbers of peritoneal macrophages presumably result from an influx of blood monocytes into the peritoneum or from local proliferation of peritoneal macrophages. Once in the peritoneal cavity, monocytes differentiate into tissue macrophages. Mononuclear phagocyte proliferation and differentiation are influenced by different cytokines, including macrophage colony-stimulating factor (M-CSF)(Weinberg JB, 1991). Blood monocytes and peripheral macrophages constitute a phagocytic system that has several functions: removal of antigens by phagocytosis, processing of these antigens by the production of specific factors, and presentation of these antigens to specific lymphocytes for disposal. Macrophage activation in turn results in the release of thromboxane A2 (Leiva M, 1992). Moreover, monocytes from patients with endometriosis have been shown to produce significantly more cytokines such as tumor necrosis factor and interferon. Macrophages are involved in immune surveillance of the peritoneal cavity. They are also known to secrete a number of growth factors including TNF-, IL-1 (Interleukin), IL-6, IL-18 which is a strong pleiotropic cytokine known to be involved in various immune diseases( Oku H, 2004). Some uncertain reports about using IL-6 and PF TNF-a can be used as screening tools for nonsurgical diagnosis of endometriosis need more investigation.
Macrophages may also cause infertility by interfering with fertilization through gamete phagocytosis (Muscato JJ, 1982). also factors produced by macrophages may interfere with sperm motility, ovulation and corpus luteum formation may be influenced by macrophages.

3. **Interleukins and other growth factors:** are produced by macrophages and is the primary mediator of the inflammatory response. Embryos exposed to interleukins are less likely to progress to the eight-cell stage at 24 hours, and a lower percentage progress to the morula and blastocyst stage at 48 and 72 hours. The elevation of IL-18 in the peritoneal fluid of endometriosis patients and the induction of COX-II (cyclooxygenase) in peritoneal monocytes by IL-18 suggest that IL-18 plays a pathogenic role in endometriosis (Oku H, 2004). and INF-, which may regulate the actions of leukocytes in the peritoneal fluid or which could act directly on ectopic endometrium. The number, size, and activation of these cells are increased in endometriosis, and the presence of mRNA encoding VEGF (vascular endothelial growth factor) has been demonstrated in activated guinea pig peritoneal fluid macrophages. The elevated levels of angiogenic and other growth factors in the peritoneal fluid may contribute to the development of an adequate vasculature and the subsequent maintenance of endometriotic explants (Oosterlynck DJ, 1993).

4. **Prostaglandins:** have also been implicated as a cause of infertility in patients with endometriosis (Drake TS, 1981). The greatest amount of prostaglandin produced in the human reproductive tract is produced within the endometrium. Prostaglandin F2 alpha increases the tone and amplitude of the cervical and uterine musculature and narrows the cervical os. It may therefore increase the venous constriction of the uterus and the intensity of uterine contractions, therefore increasing the degree of dysmenorrhea present. Also interruption of prostaglandin synthesis in endometriosis may theoretically interfere with placentation or implantation (Drake TS, 1981). Peritoneal fluid levels of two of prostaglandin endoperoxides (thromboxane B2 and 6-keto-prostaglandin F1 alpha) in patients with and without endometriosis were measured, both compounds were significantly elevated in endometriosis (Drake TS, 1982). This suggests an increase in the peritoneal fluid levels of thromboxane A2 and prostacyclin, both of which could act on tubal smooth muscle and interfere with tubal function, which explains the phenomenon of endometriosis-induced infertility when there is no direct damage to the reproductive organs (Drake TS, 1982).

5. **Mesothelial cells:** These are the cells lining the peritoneum. They are also present in the retroperitoneal tissue; these "reserve" mesothelial cells are essential in reconstructing the peritoneal layer, also they produces CA 125. In the peritoneal fluid of women with endometriosis the monocyte chemotactic protein-1 (MCP-1) produced by peritoneal mesothelial cells and endometrial cells is expressed in these mesothelial cells which induced by oxidized LDL (low-density lipoprotein), that provides direct evidence of inflammatory action of peritoneal fluid of women with endometriosis (Rong R, 2002).

6. **Placental growth factor (PIGF):** which is glycoprotein that is expressed in endothelial and epithelial cells as well as in some tumors, the production of PIGF is sensitive to cyclical changes in ovarian steroid concentrations and could contribute to an important to the development of endometriosis, particularly in patients having red lesions. It seems possible that PIGF in peritoneal fluid promotes neovascularization (Suzumori, 2004).

7. **Vascular endothelial growth factor (VEGF):** The angiogenic activity of peritoneal fluid from women with endometriosis is elevated (Oosterlynck DJ, 1993), and elevated levels of VEGF are found in the peritoneal fluid of women with endometriosis. VEGF is also expressed by endometrium.

**Conclusion:**
All available data suggest that peritoneal fluid components changes may play an essential role in the interruption of the whole natural processes of ovulation, fertilization and implantation, more investigation must be done to clarify the role of each of these pathological findings and ways of their elimination.

**References:**
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**References:**
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