



Endometriosis Pathophysiology and Peritoneal Function

Brown Hirne*

Department of Histology, University of Edinburgh, Edinburgh, UK

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Description

Endometrial tissue can be seen outside of the uterus in endometriosis, a persistent condition. In the general female population, it affects 6% to 10% of women and is linked to persistent pelvic discomfort, dysmenorrhea, dyspareunia, and/or infertility. While endometriosis-related discomfort in women can be relieved with surgery, symptoms can return in up to 75% of instances within two years. Ovarian suppression is the cornerstone of medical care and involves administering the combination oral contraceptive pill, progestogens, or gonadotrophin-releasing hormone agonists to patients. Endometriosis may now be classified into three main types: ovarian endometriomas, deep infiltrating endometriosis, and peritoneal endometriosis. This review is focused on peritoneal endometriosis.

The peritoneal mesothelium covers the surface of the peritoneal membrane and resembles an epithelial monolayer. The peritoneum is lined with a simple monolayer of mesothelial cells, which are thought to have several functions in maintaining the peritoneal cavity's physiological homeostasis. They aid in host defence, carry fluids and solutes to blood arteries, and lessen friction between tissues. The tight junctions that bind cells together at the mesothelial surface and the intricate arrangement of membrane-bound proteins linked to the actin cytoskeleton form a continuous surface.

The Extra Cellular Matrix (ECM) of the peritoneal membrane is composed of connective tissue and the basement membrane. The type I and IV collagen, laminin, and fibronectin that make up the basement membrane between mesothelial cells and connective tissue are all generated by mesothelial cells and are crucial for the cells' adherence to the peritoneum. Macrophages can be detected in tissue near to the peritoneal basement membrane and vascular areas or on the surface of peritone-

al mesothelial cells. When the peritoneum is injured, cytokines are generated that play a role in controlling inflammation. The main source of these cytokines is macrophages. Also capable of secreting inflammatory cytokines, membrane-dwelling fibroblasts can take part in host defence.

Peritoneal fluid

The fluid that fills the peritoneal cavity is made of ovarian exudate and plasma transudate, and its volume varies throughout the female menstrual cycle. Large amounts of chemicals can be passively dialyzed between peritoneal fluid and blood plasma due to the peritoneal cavity's vast surface area. It comprises a range of immune cell types, including Natural Killer Cells (NKC) and macrophages, and it aids to lessen friction between peritoneal surfaces.

Peritoneal endometriosis

Endometrial implants most frequently develop in the pelvic peritoneum, where endometriosis affects the Pouch of Douglas in more than 80% of patients. Endometriosis, however, can develop anywhere throughout the pelvic peritoneum, and it is incredibly uncommon to see it in other extra abdominal mesothelial regions like the pleura and pericardium. Traditional criteria for diagnosing endometriosis include the presence of stroma and ectopic endometrial glands inside the lesion; however, smooth muscle cells can also be present. Studies using animal models have shown that the peritoneum and the ectopic endometrial tissue interact closely on a cellular level. These studies used a mouse model where human tissue engrafts were introduced into nude (immunosuppressed) mice to examine the endometrial-peritoneal interactions during lesion development.

A multipurpose organ, the peritoneum is essential for both the host's defence against microbes and the dialy-

sis of solutes. A special environment within the body is provided by the peritoneal membrane and fluid, which are abundant in immune cells and cytokines. Differential expression of these cell surface factors may help to explain why some women develop illness while others do not by providing the ectopic endometrial cells with a focus for attachment. Cell surface adhesion factors are expressed on the peritoneal mesothelial cells.

It is believed that a compromised scavenger response in the peritoneal membrane is responsible for ectopic endometrial cells' capacity to avoid immune surveillance within the peritoneum. It is thought that this intricate inflammatory cascade is crucial to the beginning, establishment, and development of endometriosis. Endometriosis appears to be developed and maintained in large part by the pelvic peritoneum.