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Mini Review

A Brief Review of Pathogenesis of Endometriosis

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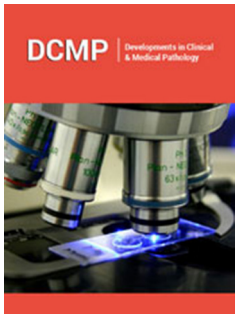

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Introduction

Endometriosis is an estrogen-dependent benign inflammatory disease, which is characterized by presence of endometrial-type mucosa in extra-uterine sites and causes chronic inflammatory at the site of lesion [1,2]. This disease affects their quality of life, work efficiency and sexual life [3] and cause of 25-70% of severe pelvic pain, dysmenorrhea, dyspareunia and infertility [4]. the proposed pathogenic theories are including retrograde menstruation, coelomic metaplasia, induction theory [5]. According to the most covincing modle, the retrograde menstruation phenomenon is accepted theory and states that viable endometrial fragments reaching the peritoneal cavity. they can implant, grow and invade onto the peritoneum and abdominal organ, proliferate and cause chronic inflammation with formation of adhesions [6]. However, this theory also does not fully explain all cases of the disease. Therefore, there are other factors that cause the progress of disease. These factors can include environmental, genetic, hormonal, or immunological factors. Various evidence suggests that endometriosis causes numerous quantitative and qualitative changes in immune cells and their products, such as cytokines [7]. Therefore, this disease is considered as a hormonal-immunological disease. Recent studies have suggested that endometrial stem/progenitor cells may be involved in the pathogenesis of endometriosis [8]. In the current studies have shown that menstrual blood plays a role in endometriosis [9]. Because it is a rich source of stem cells that can proliferate and differentiate into different classes. These stem cells proliferate easily and express multipotent markers such as Oct-4, SSEA-4, and c-kit at the cellular and molecular level [10].

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