Commentary

Etiology of Endometriosis – Simplified

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Chettinad Health City Medical Journal 2017; 6(1): 2 - 3

Introduction

Endometriosis is an estrogen dependent condition commonly seen in women of reproductive age group and most often these women present with infertility and pelvic pain / dysmenorrhea. It is not clear whether endometriosis is the cause of infertility/ pelvic pain/ dysmenorrhea or it is just an associated condition. In the reproductive age group women, the incidence of endometriosis is 10 – 11%[1] whereas the incidence is more than 30%[1] in infertile population. The common sites of endometriosis are ovaries, fallopian tubes, posterior wall of the uterus, cul-de-sac, broad ligament, round ligament. Extra pelvic sites are intestines, urinary bladder, ureters, lungs, extremities, skin, and central nervous system. Ovaries are being the commonest site of endometriosis[2].

Hypothesis

Various theories have been proposed to explain the etiopathogenesis of endometriosis like retrograde menstruation, coelomic metaplasia, induction theory, lymphovascular, oxidative stress and inflammation, immune dysfunction, apoptosis suppression, genetic and stem cell theory. Do we really need these many theories, when one theory alone can explain the etiology of Endometriosis? Having too many theories may complicate the understanding of the pathophysiology and the management of the disease.

Our hypothesis

To say "Menstruation causes Endometriosis" may be stating the obvious. Retrograde menstruation theory alone can explain the etiology of endometriosis.

Retrograde menstruation theory is also called as implantation theory was proposed by John A. Sampson in 1927. It is retrograde flow of endometrial cells into the peritoneal cavity via the fallopian tubes. These endometrial cells adhere to the ovary or peritoneal cavity, implant, grow and bleed over the course of menstrual cycle. It has been documented by laparoscopy during perimenstrual phase that 76%-90%[3] of the women have retrograde menstruation. Probably all women with patent tubes may have retrograde menstruation. The most common location of endometriosis is on the posterior aspect of the uterus and towards left side of the pelvis. In congenital Mullerian anomaly like imperforate hymen and cervical stenosis, there are increased chances of retrograde menstruation and there by increased risk for developing endometriosis[4].The incidence of endometriosis is increased in women with an early menarche, frequent menstrual cycles or women with menorrhagia. There are increased chances of these women being exposed to retrograde menstruation. This retrograde menstrual blood in the peritoneal cavity gets absorbed by lymphatic system, it drains into the venous drainage, through which it spreads to the distant sites. This explains the etiology of extra pelvic endometriosis. There are only few articles on proven (Histopathological) premenarchal endometriosis without obstructive Mullerian anomaly[5]. Neonatal uterine bleeding in the immediate post natal period occurring due to withdrawal of maternal estrogen may be the source for endometrial cells in pre-pubertal endometriosis[6]. Endometriosis in post menopausal (without HRT) is rare and the incidence is only 2.2 %[7]. The author stated that all these patients were obese and signs of increased estrogen activity would have caused peripheral conversion of androgen into estrogen. These excessive estrogen has caused reactivation of the pre existing endometriotic lesions. Hence, the triggering factor in pre pubertal endometriosis may also be due to the conversion of androgen into estrogen secreted from the peripheral adipose tissue in obese children. The incidence of endometriosis in multiparous women is also relatively low i.e. 3.7 %[8]. In these women, Pregnancy ameliorates endometriosis.

Conclusion

When there is continuous periodic constant menstruation, there are increased chances of endometriosis. The frequent occurrence of menstruation, longer duration of menstruation and excessive menstruation may overwhelm the defense mechanism in some women. The failure of defense mechanisms, may lead to implantation of endometrial cells, further growth and bleed over the course of menstrual cycle leading to endometriosis.

Acknowledgements

We thank the faculty of the Department of Andrology & Reproductive Medicine for their support through important discussions and valuable comments on this topic.

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