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Endometriosis – revisited

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ABSTRACT

Although giant strides have been achieved in human reproductive sciences, with newer technologies and therapies, yet very little is known about many distressing problems like endometriosis. Endometriosis is still considered an enigma by many gynecologists and reproductive endocrinologists. Medical and surgical advances have emerged as possible solutions for this unrelenting malady in reproductive-aged women. We raised a debate if infertility is the cause or consequence of endometriosis. In this paper, we discuss the basic pathophysiology of endometriosis, while proposing a hypothesis that "Menstruation is a biomarker for failed physiology," where repeated cycles of ovulation and non-conception exposes a woman to more retrograde menstruation. We have also questioned the value of medical and surgical treatment in infertility associated with endometriosis and have presented literature-based evidences to substantiate the points for and against the different views expressed about endometriosis. We conclude by stating that "it is wise to treat infertility and not waste time, hope, and money in trying to treat endometriosis, especially in women with infertility."

Keywords: Endometrioma, Endometriosis, Hormonal therapy, Infertility, Laparoscopy

INTRODUCTION

Endometriosis is the presence and proliferation of endometrial tissue in sites outside of the endometrial cavity. These sites may be close to the uterus, as in ovaries and uterosacral ligaments but may also occur in remote areas such as the lungs, brain, and abdominal scars. Several theories have been proposed for its occurrence. Sampson's theory of retrograde menstruation as a cause seems more rational among the many theories, and would also explain the occurrence of endometriosis in most sites.[1]

Numerous hiatuses exist in our knowledge of this perplexing condition. Theodosius Dobzhansky wrote, "Nothing in biology makes sense except in the light of evolution." Evolution is a tree (and not a ladder) and we are just one twig in this huge tree. There is no superiority or inferiority among the species. It is just hubris that we named ourselves *Homo sapiens* (wise man). We are just another two-legged animal. All the basic principles of biology apply equally well to us as to other animals with variations. We fail to recognize that evolution is by default and not by design. Mutation, mostly is what drives evolution. We still do not know why women menstruate when most other mammals do not menstruate. When applied to basic facts in evolution, it is clearly understood that "association is not causation." This is especially relevant in the study of endometriosis.

ENDOMETRIOSIS – BASIC FACTS

We are still in the dark about the exact incidence of endometriosis as it is mostly asymptomatic. In classical studies, it has been observed that 25–50% of women with infertility have

endometriosis and 30–50% of women with endometriosis are infertile.[2]

The true prevalence of endometriosis can only be found by direct inspection with laparoscopy and histopathology. Missmer *et al*. quoted a vast range of 9–50% incidence at the time of laparoscopy for infertility evaluation.[2]

There are no clear data pointing to evidence of any increase in its incidence either. A recent paper cites at least 42 million Indian women have endometriosis.[3] Endometriomas are seen in 17–44% of women diagnosed with typical ground glass appearance on ultrasound.[4] They are bilateral in about 30%.[5]

PRE-REQUISITES FOR DEVELOPMENT OF ENDOMETRIOSIS

Two fundamental pre-requisites are menstruation and functioning ovaries or adequate circulating estrogens. When the uterus is under-developed as in Turners' syndrome or absent as in Müllerian agenesis, endometriosis is hardly ever seen. Very few cases have been reported in Turner's mosaic patients or in whom hormone replacement therapy has been used.[6]

There are a few case reports on the presence of endometriosis in Müllerian agenesis (Mayer-Rokitansky-Küster-Hauser syndrome) which is probably due to coelomic metaplasia.[7] Whereas, Konrad opines that endometriosis develops in these women only if there is a uterus or endometrial tissue.[8]

Similarly, cessation of menstruation temporarily during pregnancy and lactation ameliorates symptoms and signs of endometriosis. When menstruation totally ceases at menopause, complete resolution of endometriosis has been observed. All other hypoestrogenic states also bring down the symptoms and signs significantly.[9] Interestingly, there is scientific evidence that "menstruating non-human primates also develop endometriosis spontaneously."[10]

Evers humorously commented "Everyone knows endometriosis but no one knows what it is."[11]

We are aware that most mammals do not menstruate. Menstruation in women regularly occurs when ovulation does not result in a pregnancy. We therefore, surmised that menstruation is a biomarker of failed physiology.[12]

ENDOMETRIOSIS AND POLYCYSTIC OVARIAN SYNDROME

Schliep has noted coexistence of incidental endometriosis and symptoms suggestive of polycystic ovarian syndrome in nearly 1 in 20 women undergoing laparoscopy or laparotomy. The incidence was 1 in 50 in asymptomatic women.[13] This goes to prove that anovulation/oligoovulation is not a rare feature in documented cases of endometriosis.

IS ENDOMETRIOSIS AN AUTOIMMUNE DISORDER?

There is a hypothesis that endometriosis develops an autoimmune disorder, where the symptoms and signs of endometriosis such as pain, adhesions, and alteration in the tubo-ovarian architecture are secondary to the aberrant immunological response to endometriotic lesions in genetically prone women.[14]

All these various speculations on the occurrence of endometriosis bring us back to the fact that we know very little about this intriguing gynecological condition.

BACK TO BASICS

The natural history of the course of endometriosis is not clearly known. As early as 1990, studies observed that the condition remains stable or improves over time in most women. Spontaneous resolution occurs in 24% and worsening of symptoms in about 47% in his study.[11] Yet, neither the natural history nor the mechanism of pain or infertility in endometriosis is clearly understood.[15]

WHAT IS ENDOMETRIOSIS?

- Is it an inflammatory condition – If so, is it local or systemic?
- Is it a neoplastic condition? – since it spreads, infiltrates, and recurs.
- Is it an autoimmune condition?
- Is it a degenerative condition?
- Does endometriosis cause infertility? – Rawson and Gylfason *et al*. reported an incidence of 3–45% in women undergoing laparoscopic sterilization.[16,17] It is therefore impossible to find the exact incidence of endometriosis in the general population, and especially the fertile women.
- Is infertility the cause or consequence of endometriosis?
- Does anovulatory menstrual bleed also cause endometriosis?

The symptoms usually attributed to endometriosis such as dysmenorrhea, dyspareunia, infertility, and dyschezia can have other causes also. Most of the women with endometriosis have been noted to be asymptomatic.[17] Even the origin of pain in dysmenorrhea is debated as to whether it is inflammatory, congestive, spasmodic, neurogenic, or a combination of all.

ENDOMETRIOSIS AND INFERTILITY

This association is even more mind-boggling. This is a complex and controversial topic. The European Society for Human Reproduction and Embryology (ESHRE) has categorically stated that there is insufficient evidence to link endometriosis as the cause for infertility. It also added that the chance for spontaneous conception in endometriosis is no different from unexplained infertility.[18] The Practice Committee of American Society of Reproductive Medicine (ASRM) also echoes the controversies in associating endometriosis with infertility.[19] Surya and Pandiyan opined that endometriosis is the consequence of failure to conceive and not the cause of infertility, at least in its early stages.[1] He also made the interesting reverse causation theory, that is, – infertility leads to endometriosis and endometriosis worsens infertility. Adhesions impairing tubal function or patency may result in impaired fertility in some women with endometriosis.

DIAGNOSTIC EVALUATION AND ITS UNCERTAINTIES

Although laparoscopy is the gold standard for the diagnosis, the Practice Committee of ASRM does not recommend it routinely for all infertility patients except for those with pelvic pathology or severe dysmenorrhea.[20]

This raises questions as to how many have associated pelvic pathology and how many patients with endometriosis suffer from severe dysmenorrhea. Doyle *et al*. in his paper on adolescents identified endometriosis in 62–75% of patients with chronic pelvic pain and 70% had unabated pelvic pain after non-steroidal anti-inflammatory drugs and oral contraceptives.[21]

Even laparoscopic diagnosis needs histological confirmation of endometriosis but a negative histology does not rule out endometriosis. This paradoxical and complex picture makes endometriosis puzzling and controversial.

LAPAROSCOPIC SURGERY

Concurrent laparoscopic surgery to treat endometriosis is not recommended by ASRM in minimal or mild cases as around 40 laparoscopies are needed to gain one additional pregnancy in the so-called unexplained infertility group.[20] Adhesion formation necessitating relook laparoscopy within 12 weeks is quite significant in any extent of the condition.[22]

ESHRE in their recent guidelines suggest surgery for revised-ASRM stage 1 and 2 endometriosis and certainly not in deep lesions. Similarly, guideline development group does not recommend surgery strongly for endometriomas and deep seated pathology as there are no control studies for spontaneous conceptions with or without surgery.[18]

MEDICAL MANAGEMENT OF ENDOMETRIOSIS

1. Progestins – to create a pseudopregnancy state
2. Combined estrogen-progestin therapy
3. Gonadotropin-releasing hormone agonists and antagonists
4. Danazol
5. Aromatase inhibitors.

While the medical management helps in managing the pain, there is no evidence that it improves fertility, and moreover, there is a delay in fertility as most of the drugs used suppress ovulation.[18,19] ESHRE guideline strongly recommends against ovarian suppression therapy in women with infertility and endometriosis.[18]

INDICATIONS FOR SURGERY

The accepted indications are as follows: (1) Bowel stenosis, (2) ureteral obstruction, and (3) mass of uncertain nature/suspected malignancy.[23] Pain is a relative indication, and infertility is NOT an indication to intervene surgically.

SURGERY FOR ENDOMETRIOMAS

The oft cited reasons for excision of an endometrioma are as follows: (1) Changes during ovarian stimulation, (2) difficulty during oocyte pick up, (3) improved pregnancy rates, (4) problems during pregnancy, (5) possibility of rupture, and (6) risk of malignancy.

Arguments against surgery for endometriomas in infertile patients quote equally good pregnancy rates as compared to all other causes of infertility.[24]

Support is gained for this statement in the following evidences – good follicular response,[25] no compromise in oocyte or embryo quality,[26,27] negligible technical issues during oocyte pick up[28] very low risk of infection,[29] no significant increase in cyst volume,[30] no greater pregnancy complications,[31] and no significantly increased risk of malignant change in endometriosis.[32]

Recent alert on associated systemic illnesses (cardiovascular, mental, and all-cause mortality) in women with endometriosis remains unproven.[33]

SURGICAL MANAGEMENT AND POSSIBLE COMPLICATIONS

1. Premature ovarian failure is a serious concern, especially after bilateral surgery for endometrioma.[34]
2. Reduced ovarian reserve – This is the reality in significant number of women already suffering from infertility.[35,36] Great difficulty in finding the plane of cleavage between the lesion and normal ovarian tissue

is the reason of this avoidable loss of ovarian function. The oocytes are also invariably very close to the vicinity of the cyst.[37] Severity of the damage is more when large sized endometriomas are excised.[38]
3. Recurrence – Endometriosis is often discovered even after surgery in 11–32% within 1–5 years.[39]

MANAGEMENT OF INFERTILITY

1. Anovulation – Rare in endometriosis. Managed with ovulation induction and timed intercourse/intrauterine insemination (IUI).
2. Ovulatory cycles – Superovulation with ovulogens and IUI. Pregnancy rates and cumulative birth rates after 4 cycles are similar in minimal/mild endometriosis and unexplained infertility.[40]
3. Assisted reproduction technology (ART) with *in vitro* fertilization/intracytoplasmic sperm injection is indicated in (a) bilateral tubal block, (b) older women with long duration of infertility, (c) male factor infertility, and (d) failed IUI cycles.

No difference is noted in pregnancy and live birth rates in women with and without endometriomas.[41,42] Disappointing outcomes were noted in ART cycles following endometrioma surgery in terms of longer duration of ovarian stimulation with higher doses of gonadotropins and a lesser number of oocytes but with similar pregnancies and live birth rates as in the non-operated group.[43] This raises the question of wisdom to operate on endometriomas before ART cycles.[42] Recent ESHRE guidelines have reinforced this point and advised the practitioners not to perform surgery for endometriomas which will not only have negative impact in many areas but also lowers the ovarian reserve.[18]

The practice committee of ASRM cautions about the delay in fertility therapy if combined medical and surgical treatments are offered as fertility enhancers.[19]

On the same note, it can be stated that various alternative treatment modalities have been claimed to improve fertility outcomes, there is no substantial proof of their success and, hence, would only delay the appropriate treatment. "Fertility is a race against time" – Pandiyan. The age of the female partner and the duration of infertility matter most in this context.

FERTILITY PRESERVATION

Young patients with endometriosis are particularly vulnerable for reduced ovarian reserve following surgical interventions. Hence, promoting the idea of fertility preservation before surgery is an emerging concept. One can use the Strawman fallacy as performing ovarian surgery, compromise the reserve, and suggest fertility preservation or maintaining the ovarian reserve by avoiding surgery and try for spontaneous/assisted conceptions.

PREVENTION OF ENDOMETRIOSIS PROGRESSION

1. Pregnancy – studies have shown a considerable decrease in the size of endometrioma during pregnancy in a significant number of women,[44]
2. Continuous combined oral contraceptive pill,
3. Dienogest – newer progesterone has been shown to reduce the size of endometrioma, and
4. Levonorgestrel releasing intra uterine device is effective in reducing the pain and bleeding, possibly endometriotic lesions.

The protective effect of medication rapidly vanishes when it is discontinued with higher rates of recurrence.

CONCLUSION

The major pathology behind endometriosis is repeated, uninterrupted cycles of menstruation, which also increases the risk of retrograde menstruation. The intensity of the symptoms and the stage of the disease do not correlate most of the times. Emerging research is centered on the identification and localization of endometrial cells through the use of polymer-based nanoparticles. Nevertheless, their validity and clinical applicability necessitate comprehensive evaluation. When it comes to infertility, diagnostic laparoscopy does not have a role in the evaluation of endometriosis. Medical or surgical treatment is indicated only to ameliorate pain. Removal of endometriomas reduces ovarian reserve and does not increase the chance of conception. Therefore, surgery should be offered as the first line only when there is unabated pain, bowel/ureteric obstruction, or when malignancy is suspected. The message to our minds is loud and clear – "treat infertility and not endometriosis."

Endometriosis is not an enigma until we intervene and complicate it.

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