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Review Article

Effectiveness of hysterectomy in endometriosis treatment

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ABSTRACT

A preliminary investigation into endometriosis risk factors, the majority of cases of endometriosis occur in reproductive-aged women. Subfertility, dysmenorrhea, and pelvic discomfort are the main symptoms. Ovarian cysts and other non-gynecologic causes can cause persistent pelvic pains. Age at menarche and cycle duration are two additional menstrual risk variables associated with endometriosis. The likelihood of endometriosis is greater in the majority of epidemiologic investigations that have examined the subject, which typically defines early menarche as occurring before or at the age of 11. A shorter cycle duration, commonly defined as 27 days or less, most of these research, along with others, have found that is linked to an increased risk. Period length and heavy flow are areas where there is less conclusive data. Given that endometriosis risk factors include early menarche and short cycles; endometriosis's pathophysiology can be better understood by delving into the underlying physiological reasons for these problems.

Keywords: Dysmenorrhea, Endometriosis, Menarche

INTRODUCTION

Endometriosis is a common gynecological disorder that primarily affects women who can bear children. Subfertility, dysmenorrhea, and pelvic discomfort are the main symptoms.[1] The majority of patients report either noncyclic or cyclic pelvic discomfort among these symptoms. As the most disabling symptom of the disease, it drastically reduces the standard of living for women who suffer from it.[2,3] Current medical therapies for endometriosis mostly aims to alleviate discomfort by modifying the menstrual cycle hormonally.[2] However, endometriosis is not the only cause of persistent pelvic pains (CPPs). Ovarian cysts, chronic pelvic inflammatory disease, pelvic adhesion, and other non-gynecologic causes can potentially cause or worsen CPP.[4]

Finding the root gynecologic problem that enhances The initial stage in alleviating pain is CPP, as various diagnoses typically need different therapies. Among the few disorders that are believed to produce cervical polyps, endometriosis satisfies the classic epidemiologic criteria for causation.[5]

EPIDEMIOLOGY

Less common are incidence statistics for the general public. A population-based study that used medical records from the 1970s estimated the prevalence of surgically or pathologically diagnosed disease in white females aged 15–49 to be 1.6 per 1000.[15] Endometriosis was the primary diagnosis in 1.3% of 1000 instances in a more recent research that included hospital discharges in women between the ages of 15 and 44.[16] It appears that endometriosis is a reproductive and postmenopausal disorder and that the estrogen milieu plays a role in its pathophysiology, according to demographic data. According to two clinical investigations, whereas whites may have a higher risk of illness, blacks may have a reduced risk.[6,10]

Researching a woman's reproductive history for endometriosis risk factors can be tricky because infertility is both a possible symptom of the illness and a component of the case definition in any given research. Despite our earlier remark on the study's overall lack of relevance, based on endometriosis detected during tubal ligation. An

association between increasing parity and a decreased probability of endometriosis detection was shown in research.[7]

The risk for endometriosis is high, and dysmenorrhea is often thought of as a sign of the illness.[8,18,19] Age at menarche and cycle duration are two additional menstrual risk variables associated with endometriosis. The likelihood of endometriosis is greater in the majority of epidemiologic investigations that have examined the subject, which typically defines early menarche as occurring before or at the age of 11.[7,18-22] A shorter cycle duration, commonly defined as 27 days or less. Most of these research, along with others, have found that is linked to an increased risk.[6,7,19,21-23] Period length and heavy flow are areas where there is less conclusive data. Given that endometriosis risk factors include early menarche and short cycles; endometriosis's pathophysiology can be better understood by delving into the underlying physiological reasons for these problems.

Table 1: A synopsis of endometriosis risk factors.

Variables	Association description	List of authors
Exercise	Exercising regularly might reduce the danger	7, 18, 2
Height	The danger grows as one's height rises.	7, 18
Caffeine	Using it might raise the danger	36, 37
Age at menarche	Having your menstrual period at a young age puts you at a high risk	7, 18–22
Weight, BMI	Inadequate inverse relationship	7, 17, 18
PCB and dioxin exposure	Evidence from primates is good, whereas evidence from women is variable.	40–42
Dysmenorrhea	Promising indicator, albeit often misdiagnosed as a sign of pre-existing conditions	7, 17, 18
Family history	Strong indications that a previous illness runs in families	26–30
Alcohol	Its use might increase the risk	18, 35
Cycle length	A shorter and more regular cycle is associated with a much higher risk	6, 7, 19, 21–23
Parity	Raising parity reduces risk	8
Smoking	If you smoke, you could lower your risk	7, 19, 21

PHYSIOPATHOLOGY

Endometriosis varieties

The three types of ectopic endometrial lesions, which consist of stroma and endometrial glands, are superficial peritoneal endometriosis (SPE), ovarian endometrioma (OMA), and deep infiltrating endometriosis (DIE), defined according to their location and physiopathology.[43]

Pathogenesis of peritoneal endometriosis

In between fifteen and fifty percent of endometriosis cases, peritoneal endometriosis is detected.[44] The peritoneal lesions were categorized by Nicolle et al as either red, black, or white. Here we may see the disease's progression through its evolutionary stages. The first stage of peritoneal endometriosis is marked by red, actively developing lesions that are rich in blood vessels.[43] The second stage, advanced endometriosis, is indicated by black lesions, while latent or quiescent endometriosis, or healed endometriosis, is represented by white lesions.[43]

What is the mechanism by which endometriotic cells enter the peritoneum? Among the pathogenic hypotheses, retrograde menstruation is most strongly backed by evidence. When endometrial pieces' implant in the peritoneal cavity after traveling down the fallopian tubes during uterine contractions.[45]

A lot of women have retrograde menstruation.[46] Endometrial cells likely develop endometriotic lesions when they cling to peritoneal surfaces for reasons other than the ones already mentioned. Concurrent processes may also involve the peritoneal mesothelium transforming into glandular endometrium, a condition known as celomic metaplasia.[46] Just because there are populations of progenitor cells and endometrial stem cells in eutopic endometrium.[47] These cells may be helped by retrograde menstruation, which makes us think they have a role in when peritoneal endometriosis starts.[48] Individuals suffering from endometriosis often have aberrant integrin profiles, which render their endometrial stromal cells inherently sticky.[49] This, characteristic of endometriosis, is exacerbated by an inflammatory milieu. Estradiol induces the endometriotic tissue to increase. When endometriotic lesions are present, aromatase, steroidogenic acute regulatory protein, and estrogen receptor β expression levels are elevated, and (17β-hydroxysteroid dehydrogenase 2) expression was reduced.[50,51] Epigenetic differential methylation of PR-B, HOX, and GATA family transcription-factor genes and alterations in progesterone signaling demonstrate a "progesterone resistance" feature. This is another feature of ectopic endometrial cells, disrupting the process of endometrial decidualization and preventing the formation of epithelial cells that rely on estrogen.[52,53] The overproduction of chemokines, cytokines, and prostaglandins contributes to a localized inflammatory response, which in turn supports and intensifies all of these processes.[53]

Ovarian endometrioma

Around 50% of women who undergo infertility treatment also have ovarian endometrioma, and 2-10% of reproductive-age women do as well.[45]

Pathogenesis of ovarian endometrioma

A cyst in the ovary that is surrounded by endometrial tissue and contains a thick, dark fluid is called an ovarian endometrioma (OMA).[43] Numerous pathogenetic hypotheses on the origin of OMA have been generated from histological examinations of the cyst wall and interior coating.

Ovarian cortex invagination and superficial implants

Adhesions formed by the ovary and peritoneum on the back of the wide ligament's leaf, endometriotic implants derived from retrograde menstruation may be more easily established, according to Brosens. After that, the ovarian cortex will invaginate, which will lead to the development of the distinctive "chocolate" liquid. This is because the active implant will leak menstrual debris into the surrounding ovarian cortex, creating a buildup.[54]

Implants on the surface and invasion of the corpus luteum

A corpus luteum that is unable to resorb owing to endometriotic lesions and adhesions on the ovarian cortex may have been the source of the trapped blood, contradicting the notion put out by Vercellini and colleagues.[55]

Intravaginal mesothelial inclusions metaplasia

Ectopic endometrial tissue and ovarian epithelial invaginations were reported by Donnez et al. The authors propose that celomic metaplasia is a possible cause: a disorder influencing the ovarian mesothelium that creates mesothelial inclusions after invaginating into the ovarian cortex.[56]

Deep infiltrating endometriosis

An estimated 2% of endometriosis patients will experience DIE, bringing the overall risk of occurrence to 20%.[57]

Pathogenesis of deep infiltrating endometriosis

Histological features of deep infiltrating endometriosis (DIE) include glandular cells, stromal cells, cells with mixed differentiation, pure undifferentiated glandular cells, and well-differentiated glandular cells.[58] Consequently, undifferentiated endometriotic lesions can develop when endometrial foci penetrate deeper into organs and tissues that can withstand the peritoneal fluid's suppressive effects.

Zanatta et al discovered increased ERa, PR-A, and PR-B mRNA expression in rectosigmoid DIE lesions, which differs from endometriosis in the ovaries and peritoneum.[59]

The endometrial stroma and epithelial cells need immunotolerance and adaptation skills to thrive in hostile environments like the vagina or the digestive tract. At least part of the blame for these changes lies with Sphingosine-1-phosphate (S1P) receptor 1, which controls an intricate inflammatory cascade that starts with tumor necrosis factor-a (TNF-a) and interleukin (IL) 1b.[60] This cascade generates prostaglandins and nitric oxide. Researchers in 2014 found that IL-1b and IL-1 receptor type II (IL-1sRII) levels were significantly higher in the blood of people with DIE, in particular those who suffer from the most severe bowel disorders.[42-45]

Endometriotic cells are known to have an overabundance of VEFG, therefore red vascular peritoneal endometriotic lesions exhibit this trait more prominently than older scarred lesions that are black or white. When it comes to this phrase, SPE, OMA, and DIE are different. When compared to endometriosis in the ovaries or bladder, deep endometriotic lesions in the rectum are more likely to have increased blood vessel density and greater expression of VEFG-A and VEGF receptor 2.[30,31]

Deep endometriosis lesions form as a result of pathogenic processes including inflammation, neovascularization, and undifferentiated cell formation. These procedures likely facilitate the adaptation and proliferation of new endometriotic tissue in otherwise unfriendly anatomical locations.

HISTORY

Endometriosis symptoms can range from mild to severe and be associated with other medical issues or pathological processes. To aid in diagnosis and therapy, thoroughly examining and assessing important is the patient's perception of pain.[59]

The following are some of how endometriosis pain can manifest: dysmenorrhea, painful menstruation, and dyspareunia, painful intercourse, dyschezia, or painful feces; dysuria, or painful micturition, discomfort in the pelvis, lower back, or abdomen that does not go away after six months.[60]

INVESTIGATIONS

When investigating a possible case of endometriosis, ultrasonography is the method of choice. It can identify uterine fibroids, ovarian cysts, and other pelvic diseases. Primary investigations into these instances should not rely on blood tests or other imaging techniques without good reason. Cancer antigen 125 (CA-125) levels in the blood should not be monitored in patients with moderate to severe endometriosis, regularly tested. Although it may be raised in these cases. With a specificity of 90% and an estimated sensitivity of 28%, A meta-analysis was carried out using 23 studies that looked at blood CA-125 levels in women who had endometriosis surgically identified.[65] Nevertheless, the SOGC recommendations should be

followed for the evaluation of any pelvic mass that has not yet been identified. The Risk of Malignancy Index includes the CA-125 level.[47]

When a more invasive form of endometriosis is detected, further imaging tests such as rectal ultrasonography, colonoscopy, cystoscopy, or magnetic resonance imaging (MRI) may be required. Such as invasion of the bowels or bladder. Histologic analysis and direct visualization during laparoscopy constitute the diagnostic gold standard. Endometriotic lesions, their location and form, and any organ involvement are the greatest indicators of a serious disease. For endometriosis staging during laparoscopy, an organization known as the American Society for Reproductive Medicine has proposed a taxonomy.[38] Since the patient's symptoms are unrelated to the ease stage, this categorization is useless for therapy purposes. The ASRM system describes the usual range of illness severity levels that are communicated to healthcare procedures: minimum, mild, moderate, and severe. Due to practical considerations, each medical facility will use its own unique set of criteria to arrive at a diagnosis and describe the patient's condition. Laparoscopy now can objectively record diseases through the use of video and picture capture equipment. It is not necessary to do a diagnostic laparoscopy before therapy for every patient who presents with pelvic discomfort. The dangers of surgery, such as vascular damage and intestinal bladder perforation, are still present with laparoscopy, despite its reputation as a less invasive operation. Laparoscopy carries an 8.9% overall risk of serious or mild complications.[29]

MECHANISM OF PAIN

Increased levels of prostaglandin E2, nerve growth factor, and other inflammatory cytokines and growth factors are hallmarks of endometriosis's chronic inflammatory state, which is associated with dyspareunia and other pain symptoms. Over time, these inflammatory substances can cause hyperalgesia, central sensitization, and myofascial pain, which are indications of peripheral sensitization.[56] To better understand chronic pain and find alternatives to unnecessary surgeries, the idea of central sensitization is fundamental. One theory is that central sensitization can occur as a result of chronic inflammation, nerve damage, recurrent and persistent noxious stimulation, or both. Preventing this illness requires prompt treatment of pain symptoms. Central sensitization can intensify after surgery, and individuals with this condition commonly claim that their symptoms were worse following the procedure. Endometriosis pain severity is strongly connected with the recently observed altered brain chemistry in women.[53,54]

SURGICAL DIAGNOSIS

The ability to visually identify endometriosis during surgery makes it an essential tool for both diagnosis and therapy. Due to the limited reliability of eye assessment alone, excision and confirmation by histology are strongly advised. Endometrial glands, stroma, and macrophages loaded with Endometriosis are characterized histologically by hemosiderin. Endometriosis is rated by stage I and stage II), stage III, and stage IV according to the American Society for Reproductive Medicine.[25] The model has limited clinical use since it is not associated with quality-of-life measures. The staging score developed by the American Society for Reproductive Medicine is widely used to measure disease burden and promote consistency in research and patient treatment. It was originally established for fertility evaluation, but it is now used for other purposes. There are additional taxonomies used in surgery, such as the Enzian classification, which measures the extent to which endometriosis has penetrated a patient's body, and the endometriosis fertility index, which uses surgical results to forecast the patient's fertility. Endometriosis often manifests itself in the uterine lining, the pelvic peritoneum (namely the cul de sac and broad ligament), and the uterosacral ligaments. No lesions will be overlooked during surgery if the pelvis is approached methodically.[59] Taking pictures of each part will help when talking to the patient and other doctors.

Table 2: Symptoms of endometriosis.[58]

Symptoms	Conditions displaying comparable symptomatology
Dyspareunia	Problems with the pelvic floor and mental health
Bowel symptoms (diarrhea, cramping, constipation)	Irritable bowel syndrome (IBS), hemorrhoids, and constipation
Infertility	Mysterious infertility
Tumour or lump in the ovaries	Small cyst on the ovary
Discomfort in the bladder and irregular urination	Pelvic floor issues, painful bladder syndrome, interstitial cystitis
Dysmenorrhea	Symptoms such as adenomyosis, primary dysmenorrhea, and blocked Mullerian abnormalities in teenagers.
Nonmenstrual pelvic–abdominal pain	Syndromes involving the entrapment of nerves in the abdominal wall, neuropathic pain, adhesions, and irritable bowel syndrome
Defecation pain (dyschezia)	Problems with the pelvic floor and anal fissures

SURGICAL MANAGEMENT

Although endometriosis surgery helps treat infertility, chronic pain, and ovarian cysts, it does come with several problematic side effects. When medical management fails, is intolerable, or fails to provide the desired results, or when a prompt diagnosis or treatment is required, surgical

intervention may be considered. Infertility therapy and the identification of an adnexal mass are two further indications for its use. Endometriosis treatment options range from less invasive laparoscopic procedures to more drastic procedures like hysterectomy, which may or may not involve ovary removal. Peritoneal disease therapy options include excision or energy-based lesion ablation. There was a trend favoring excision, although a recent meta-analysis of three randomized trials failed to find a statistically significant variation in patient reports of pain.[52]

Endometriosis close to vital organs like the ureter, colon, or bladder should not be ablation since the lateral dispersion of the energy beam might harm these tissues. In addition, without adequate dissection, the danger of damaging underlying tissues is great when attempting to ablate deeply infiltrating illness using any energy type. Thus, the best and most comprehensive surgical treatment for endometriosis typically involves excision.

Results of surgery for women suffering from chronic pelvic pain

Odds ratio [OR] (10.00, CI 3.21–31.17) was the difference between diagnostic laparoscopy alone and endometriosis laparoscopic surgery at 6 and 12 months in terms of total discomfort.[43] Additionally, compared to laparoscopic surgery, diagnostic laparoscopy with GnRH agonist medicine did not yield the same results. The anticipated rate of symptom recurrence is around 10% after 1 year and 40-50% after 5-7 years.[38] The use of long-term postoperative medicinal suppressive treatment has been inconsistent in several surgical investigations. Medical therapy that suppresses hormones immediately after surgery is recommended by society's standards since it reduces the likelihood of symptom recurrence and the need for additional surgeries.[36,37] Six to twenty-four months is the sweet spot for suppression. Several methods have been proposed to enhance the success rate of surgical procedures. The primary idea is to make endometriosis easier to see during surgery by using a fluorescent dye, such as indocyanine green. When it comes to surgical endometriosis therapy, no high-quality trials have shown this medicine to be effective. A recent randomized multicenter clinical trial comparing robotic surgery to conventional laparoscopy did not find any advantage in short-term postoperative parameters or improvement in pain scores or other quality-of-life indicators, despite robotic surgery providing a new dimension to the visualization and treatment of endometriosis.[42] The second strategy to enhance surgical results is uterine denervation or nerve transection. Except for presacral neurectomy, no procedure has improved results according to much research. Even if midline pain can be effectively reduced by presacral neurectomy (dysmenorrhea, for example), surgery comes with the risk of bowel and bladder denervation, which can lead to issues including constipation and malfunction in the bladder.

Management of endometriomas

Surgery for endometrioma can reduce ovarian reserve and harm the ovaries, so patients need to know what they're getting out of the procedure before it happens. When the goal of surgery is to diagnose the tissue and alleviate symptoms, a full excision is the most successful and least invasive method with a lower risk of recurrence. A Cochrane review synthesized the findings from two randomized clinical trials and concluded that, in comparison to ablative surgery, cyst excision had better outcomes, including lower rates of recurrence and symptom recurrence as well as higher rates of spontaneous pregnancy (OR 5.1, CI 2.04-13.29).[41]

Treatment for endometriosis that has spread outside the pelvic areas

Many organs outside of the genitourinary system are affected by deeply invading endometriosis. The rectovaginal septum, uterosacral ligaments, intestine, urinary system, ureter, and bladder are all potential sites of involvement. Thorough familiarity with the retroperitoneal area is necessary for the excision of endometriosis that has penetrated deeply.[49] When a lesion is deep enough, it might show up in the ureter, bladder, or rectal area posterior to the cervix. Making simple ablation an ineffective treatment option. While suppressive medication does alleviate discomfort for women suffering from deeply invasive endometriosis, there is no evidence that it enhances reproductive prospects.[42]

Hysterectomy

For women who suffer from severe endometriosis discomfort, a hysterectomy is the best option. After 2, 5, and 7 years, the reoperation-free percentage for women who underwent a hysterectomy with bilateral oophorectomy was 92% as shown by ongoing monitoring. For women who underwent hysterectomy alone, it was 96%, 87%, and 77%. Reoperation is 2.44 times more likely when ovaries are conserved for 7 years, there was no difference in the probability of reoperation among subgroups of hysterectomy patients younger than 40 years old. Between individuals with endometriosis and preserved normal ovaries. We recommend that women under the age of 40 have their natural ovaries preserved. Surgical menopause is a final option that the patient must consider, whether carries a lower chance of recurrence or not.[35]

CONCLUSION

It is commonly believed that a hysterectomy will permanently alleviate the severe pelvic discomfort experienced by women who suffer from endometriosis. Yeah, that's the case for a lot of ladies. Nevertheless, a handful of women may have a return of their symptoms following the "definitive" surgery. Although the risks of disease recurrence are higher for women who opt to keep

one or both ovaries or begin hormone replacement therapy (HRT) after undergoing an oophorectomy, the advantages of these choices, especially for younger women, probably exceed these risks.

The recurrence of endometriosis following a hysterectomy has been the subject of much speculation. Possible causes include hormonal variables, ovarian remains, uterine morcellation, lymphovascular invasion, de-novo illness, and persistent microscopic foci. But it's more probable that disease persistence than recurrence following hysterectomy is the real culprit in most situations. It is unclear from the research on disease recurrence following hysterectomy if all endometriotic lesions were surgically removed during the initial procedure. Although it is a more technically demanding operation, recurrence rates are lower when endometriotic lesions are removed entirely during hysterectomy. Because of this, hysterectomy for the main diagnosis of endometriosis should only be performed by surgeons who are well-versed in identifying and removing endometriosis samples. In any other instance, it would be wise to send the patient to a surgical facility that specializes in endometriosis.

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