Chapter

Minimally Invasive Surgical Treatment of Pelvic Pain in Teenagers and Young Women

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Abstract

Pelvic pain could be acute or chronic but rarely could be life threatening with various reasons such as pathological, physiological or functional. Clinical evaluation and management should be performed simultaneously, especially in emergencies that carry a high risk of mortality. Clinical evaluation and management should be performed simultaneously, especially in emergencies that carry a high risk of mortality. Although a detailed history, physical and gynecological examination, supplemented with imaging modalities can itself be diagnostic, the role of laparoscopy for diagnosis should not be overlooked. The common causes of pelvic pain with focus on a minimally invasive approach in this age group are as following: endometriosis, rupture of ovarian cyst, infection, ovarian torsion, pelvic vein syndrome, adhesions pain due to previous surgery and unsatisfactory treated infections.

Keywords: acute and chronic pelvic pain, gynecologic, non-gynecologic, endometriosis, cyst accident, torsion, ectopic pregnancy

1. Introduction

Pelvic pain is categorized as chronic or acute. Moreover should be mentioned that the incidence of pathology is different by age and it is essential to distinguish the causes that could be gynecological or non-gynecological. In adolescents appendicitis, cyst eruption or intussusception consist the most common causes of pelvic pain and on the other hand ectopic pregnancy, uterine fibroids, torsion of ovarian cyst, and pain are Mittelschmerz the most common causes of pelvic pain in women

of reproductive age. Additionally, in postmenopausal woman diverticulitis, tumors and renal stones are the most frequent pathology.

There are multiple causes **of** pelvic pain with a broad spectrum of possible pathologies, physiological **processes** and functional syndromes that can cause pain. The pain is usually noncyclic. Chronic pain is defined as lasting for 3 to 6 months or more) [1]. The condition can be combined with abdominal pain. Contrary to the impression that chronic pelvic pain is usually related to gynecologic conditions, the majority of cases is related to gastrointestinal and urologic conditions [2]. Pelvic pain, in its severe form, can cause functional disability requiring an etiological or symptomatic treatment. Considering that the range of etiologies is diverse, both symptomatic and etiological management is often necessary. Among others, endometriosis, leiomyomas, adenomyosis, pelvic inflammatory disease and pelvic adhesions are included in the gynecologic conditions; interstitial cystitis, recurrent cystitis and recurrent urolithiasis are included in the urologic conditions; and irritable bowel syndrome, inflammatory bowel disease and celiac disease are included in the gastroenterologic disorders causing pelvic pain. Minimally invasive techniques could be proposed in both investigating and managing some of the above conditions. As an example, laparo-endoscopic single-site surgery technique could be considered in the management of endometriosis [1, 3]. Similarly, although routine laparoscopic adhesiolysis is not advised for pelvic adhesions, in selected cases, such as in one or two scars around an ovary, this specific adhesiolysis could be proved beneficial.

The surgical treatment of gynecological diseases in adolescence differs from that of adults. The way of coping with is affected by the high life expectancy and the necessity to maintain reproductive capacity [3, 4].

The sensitive psychology of adolescents should not be overlooked. Finding a mass inside the pelvis is an unpleasant experience at any age, but, in adolescence, is considered traumatic for both of the patient herself and her family [5, 6].

From the data of the international literature, which involve a large number of adolescent patients, it appears that:

- ovarian tumors are the most common tumors of the genital system at this age. Of these, 65% are benign, while 35% reveal signs of malignancy.

- Uterine body tumors are very rare in adolescence [3–7]. They are usually malignant and manifest with vaginal bleeding as the primary symptom.

- Tumors of external genitalia also have a low incidence, perhaps because the environmental factors that are causally related to their appearance have not yet been affected [8–12].

- In addition, vaginal and cervical tumors are very rare in adolescence.

A variety of gynecologic and non- gynecologic conditions such as approximately in 20% of cases endometriosis, pelvic inflammatory, adhesive disease, irritable bowel diverticulitis can lead to pelvic pain.

In this review is presented pelvic pain resulting from several gynecologic conditions and referred according to our experience [8–12].

2. Acute pelvic pain

Acute pelvic pain in this age group is characterized with the following symptoms: sharp, sudden pelvic pain, exacerbating pain with great intensity, short duration

(<24 hours) and usually occur in lower abdomen. In most cases, appeared also symptoms based on irritation of neural system like nausea, vomiting, sweating and tremor. Acute pelvic pain is the most often cause of visiting emergency department of gynecological clinic and it is a challenge for gynecologists for prompt diagnosis and treatment. The most often causes acute pelvic pain are as following [13–15]:

1. Causes that are not associated with pregnancy: pelvic inflammatory disease, degeneration of fibroids, ovarian torsion, cyst eruption, abscess.

2. Associated with pregnancy: ectopic pregnancy, miscarriage

3. Non- gynecological causes: appendicitis, diverticulitis, nephrolithiasis, inflammatory bowel disease, mesenteric lymphadenitis, typhlitis, gastroenteritis, bowel obstruction, cystitis, pyelonephritis, inguinal hernia, acute thromboembolic disease, coronary heart disease and abdominal aneurysm. Surgical treatment is most time with performance of laparoscopy or laparotomy, depending on the condition and the experience and training of the doctor [15–17].

3. Chronic pelvic pain (CPP)

Chronic pelvic pain is referred in pain has duration more than 6 months, occur lower abdomen and is associated to pain intensity great influence in life quality and sometimes requires multiple doctors visits and hospital admissions. The most common causes which could be functional, pathological, physiological are as following:

1. From gastrointestinal system

2. From urogenital system

3. From female reproductive system

4. From myoskeletal system

5. From neurological system

6. From psychological causes

Frequency is approximately 4–25%, however the minority of woman in this age group (33%) visit doctor.

Various studies have studied the incidence of chronic pelvic pain (CPP) in 5,000 women, aged 12 - -22 years, in the United States. In these studies, it was revealed that 15% suffer from CPP. Of these, only 10% visited a gynecologist, while 75% had no visits in any health care provider.

Despite the fact that such an increased number of women did not visit a doctor, CPP was the cause of 10% of gynecological visits, 10 - -40% of laparoscopic surgeries, and 10 - -16% of hysterectomies [18–23].

In the USA, every year are performed approximately 4000,000. If 10 - -16% of them done for CPP, then we could assume that 65000 to 104000 hysterectomies are performed due to CPP.

In addition, while the mortality of hysterectomies is about 0.1%, 65 - -104 of deaths can be attributed to surgeries for the treatment of CPP each year. About 35% had no visible abnormal CPP findings on laparoscopy. It is estimated that 58% of

women limit their physical activity to at least one day a month, 10% seek for a doctor other than a gynecologist, while 1% seek for treatment with a psychiatrist [18–23].

The majority of the patients with chronic pelvic pain and negative laparoscopic findings (41%) were taken contraceptives, while 25% GnRH analogues for 3 months [18–23]. The American College of Obstetricians & Gynecologists recently argued that it is appropriate to prescribe empirical treatment with GnRH analogues for 3 months to women with CPP, without an exact diagnosis, who have not had any other anti-inflammatory drugs [18–23].

This approach is considered relatively safe and effective. If there is no response to this treatment, a laparoscopy or other surgical approach should be performed [18–23].

An empirical protocol is proposed by Winkel, which, after excluding other pathologies such as pelvic inflammatory disease or ovarian cysts, administers GnRH in proportion to an improvement effort. According to the results of various women, there is an improvement of 80% in patients with, or without, endometriosis, r17-22egardless of whether they have undergone laparoscopy or not [13–21].

4. Endometriosis

4.1 Definition and incidence

Endometriosis is characterized by the presence of endometrium outside the uterus, the proliferation and differentiation of tissue similar to the endometrium outside the cavity of the uterus. It is the one of most common cause of pelvic pain in women of reproductive age and the second most common gynecological condition after fibroids. Any peritoneal surface including tube ovary (where it may form endometriomas, peritoneal, bowel, bladder etc.

- If involving myometrium – adeno

- That it can be superficial, deeply infiltrating or form nodules. When penetration into fibrous tissue is revealed, the term external adenomyosis (adenomyosis, externa) characterizes this pathological condition due to its important similarity to adenomyosis (adenomyosis interna).

The term ‘“deep endometriosis” includes the morphological classification of endometriosis, which has a penetration depth greater than 5 mm [22–26].

The clear differentiation of deep endometriosis from other forms of the disease was observed by Donnez [24–28]. The nodular form of these lesions is due to the proliferation of smooth muscle fibers, their localization is retroperitoneal and may extend laterally to the anterior part of the rectum.

Although this entity is a common gynecological disease, responsible for 10 - -15% of infertility cases, little is known about the pathogenesis of the disease. For its etiology, several pathophysiological mechanisms have been proposed -, anatomical, immunological, endocrinological, environmental and finally genetic factors with genetic predisposition [24–28].

The genetic processes that regulate endometrial cell proliferation have not been fully elucidated. The etiology of endometriosis is unknown, but there is evidence to suggest that the genetic factor is associated with the development of endometriosis. The notion that endometriosis is a genetic disease was initially relied on poorly controlled studies in which a familial impact of the disease was found [24–28]. A familial tendency for endometriosis was first reported in 1957, but the first representative study of endometriosis genetics was conducted in Europe by Simpson et al.

in 1980 in Europe. It should be noted that relatives of second-degree patients had a 1.8% risk of endometriosis. Women with a family history of endometriosis tended to develop allergic reactions (eczema, asthma, allergic rhinitis). In addition, women with congenital endometriosis developed the disease at a younger age (mean age 22.1 years).

Finally, women with congenital endometriosis usually had the most severe form of the disease, the third and fourth stages of endometriosis, according to a revised American Fertility Society ranking. Although, the above information is consistent with the view of the existence of a genetic basis in endometriosis, other factors could explain the endogenous impact of the disease. Symptomatic women, for example, may seek medical help sooner if they have a relative diagnosed with endometriosis.

According to a study conducted by the Australian National Commission on Health and Social Research on 3,096 twin sisters, endometriosis was observed in 2% of singletons and 0.6% of twins. The increasing incidence of endometriosis in monozygotic twin sisters makes genetic factors more important in the etiology of the disease [29, 30].

5. Diagnosis

When the vaginal bleeding and pelvic pain, which consist symptoms of endometriosis are mild to moderate, the first line treatment is hormonal therapy. Depending on the diagnostic method that will be used, the diagnosis of endometriosis is stated only in 19–20% of cases. The majority of specialists use laparoscopy to diagnose endometriosis. This technique, however, is not without disadvantages. Laparoscopy will diagnose endometriosis in 20% of patients. Another problem with laparoscopy is that even if there are visible lesions, histological confirmation of endometriosis is not always possible. Histological confirmation also depends on the biopsy. In addition, to this due to different forms of the disease, it can be overestimated in more than 2/3 of the patients. Although, is helpful as it allows the patient and clinician to refocus on alternative medical strategies for management, especially in early stages (deep endometriosis). When positive findings – then laparoscopic ablation/resection can be undertaken. The appropriate treatment of endometriosis is of great importance in this age group due to the fact that that fertility should be maintained, also in cases in which tubes are not affected by endometrial lesions because in these cases could be negative laparoscopy findings but could affect fertility(negative influence of occurred endometrial lesions in ovum and sperm). Symptoms of endometriosis are as following: dysmenorrhea 60–80%, pelvic pain 30–50%, subfertility 30–40%, dyspareunia 25–40%, menstrual abnormalities 10–20%, periodical dysuria and hematuria 1–2%, constipation 1–2% and < 1% rectal bleeding and fatigue.

Another big problem for the diagnosis of CPP is that 80% of the patients who have had a laparoscopy has negative findings. And what about these patients who have negative findings? Doyle reports that of the patients with the negative findings, 53% wanted analgesics, 50% were unsatisfied with the treatment and 43% did not have a good quality of life. The presence of endometrial implants in the peritoneum can cause local peritoneal inflammation [30–34].

The cystic form of ovarian endometriosis is usually followed by adhesions, which is known that play an important role in the progression of the disease, explaining why ovarian endometriosis is classified as Π AFS. The determination of Ca-125 is a useful and reliable diagnostic tool, as well as intrarectal ultrasound, while transvaginal ultrasound and magnetic resonance imaging (NMR) are not

diagnostic methods used in the diagnosis. Donnez et al. distinguish 3 forms of the disease, endometrial septal endometriosis (Type I), posterior atrial dome endometriosis (Type II) and deep endometrial edema (Type III endometriosis). **Table 1** Koninckx and Martin report another stage of the disease **Table 2**, **Figures 1**–**5**.

Additional data on the genetic basis of endometriosis, based on sibling studies, are provided by the finding that siblings, non-twins who develop the disease, usually develop the pain symptom at the same age. The incidence of all stages of endometriosis in the sisters of afflicted women in comparison to the general population is six to nine times.

The relationship between endometriosis and increased estrogen production is a popular and biologically plausible hypothesis. Endometriosis and fibroids develop in women of childbearing potential and regress after menopause or after ovulation, which is consistent with the view that the development of these diseases is estrogen dependent.

The estrogen receptor (ER) and the aromatase gene (CYP19) are potential candidate genes. Both could enhance estrogen accumulation and produce a more abundant [34–36].

Thus, it has been investigated whether the polymorphism of the estrogen receptor gene Pa (ER-α) is associated with the development of endometriosis. It is recognized that ectopic endometrial foci have estrogen receptors. Endometrial foci also express cytochrome P450 aromatase, an enzyme that catalyzes the conversion of androgens to estrogens, recommending that local estrogen production may be increased.

Types	Location	Clinical characteristics
Type I	between the posterior vaginal wall and the anterior wall of the muscular wall of the rectum	<2 cm, and most of the lesions are exophytic
Type II	extends from the posterior vaginal septum to the rectal septum	lesions are mostly small and no extension is observed in the orthopedic septum or in the rectal wall
Type III	always located below the parietal peritoneum	infiltration of the muscular wall of the rectum is observed

Table 1.
Distinction of endometriosis according to Donnez et al.

Types	Location	Clinical characteristics	Percentage
Type I	conical shape, infiltration of the surrounding tissues	large pelvic area of endometrial lesions, which are surrounded by off-white connective tissue	4,1%(women with infertility) 10,4% (women with CPP)
Type II	clinically perceived by the diving of the bowel around a typical endometrial lesion	the result of traction of the superficial bowel	0,8%(women with infertility) 3,2%(women with CPP)
Type III	spherical endometrial nodule	most severe form, and often extends laterally around the uterine artery and the corresponding ureter	0,9%(women with infertility) 3,2%(women with CPP)

Table 2.
Distinction of endometriosis according to Koninckx and Martin.

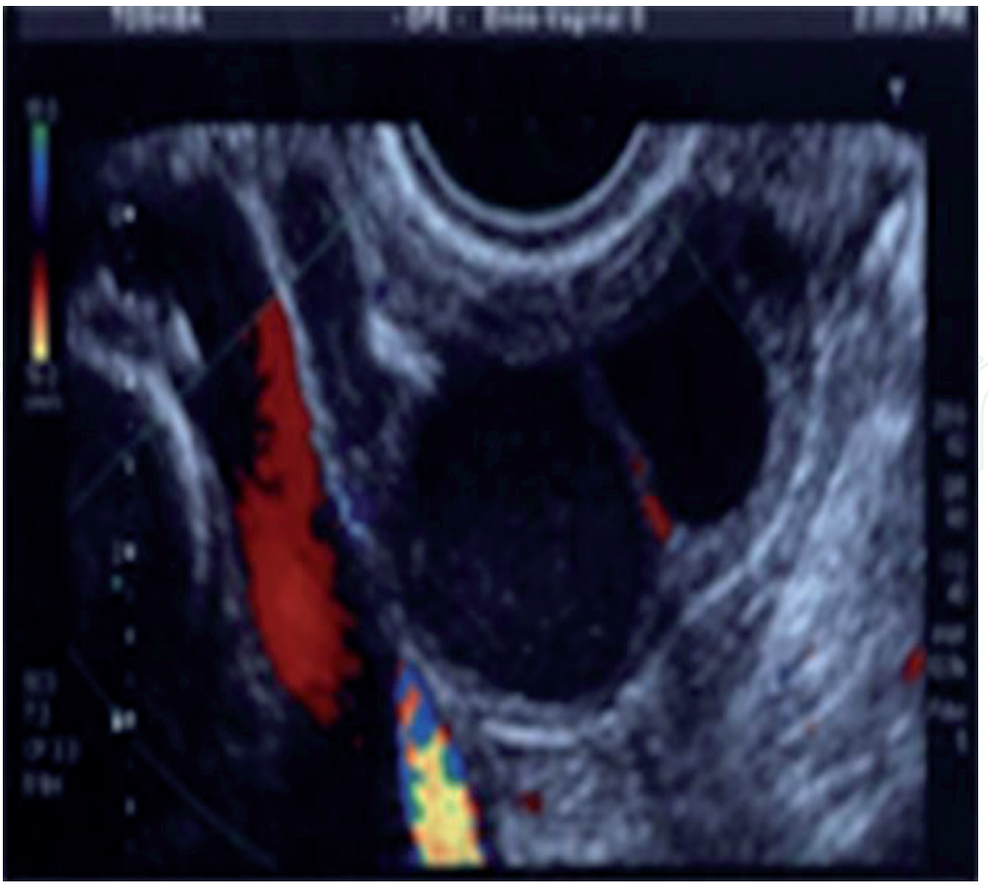

Figure 1.
Ultrasound image showing large endometriomas.

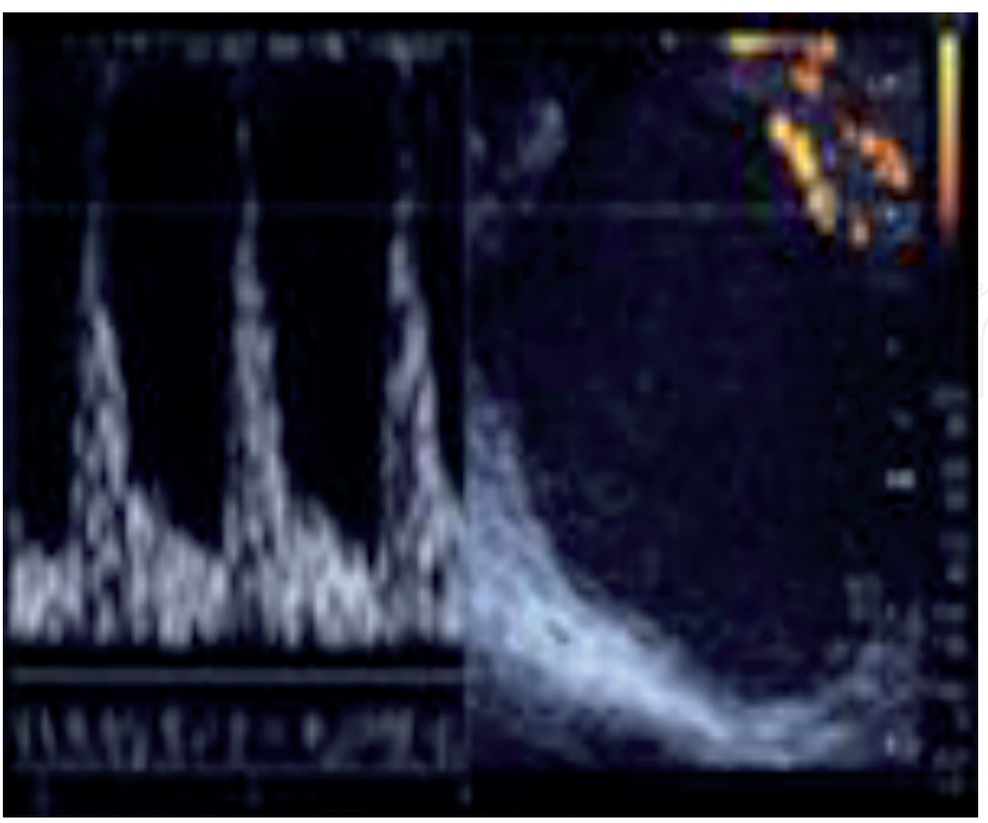

Figure 2.
Doppler ultrasound in endometriomas with high PI (pulsatility index).

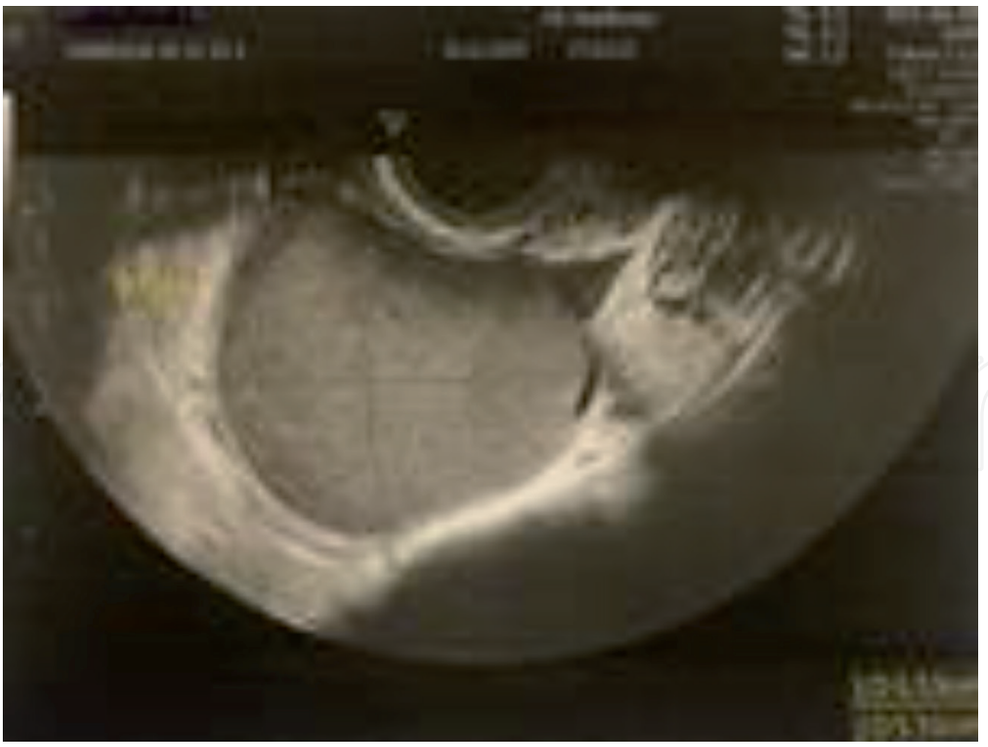

Figure 3.
Ultrasound image of endometriomas (chocolate cyst).

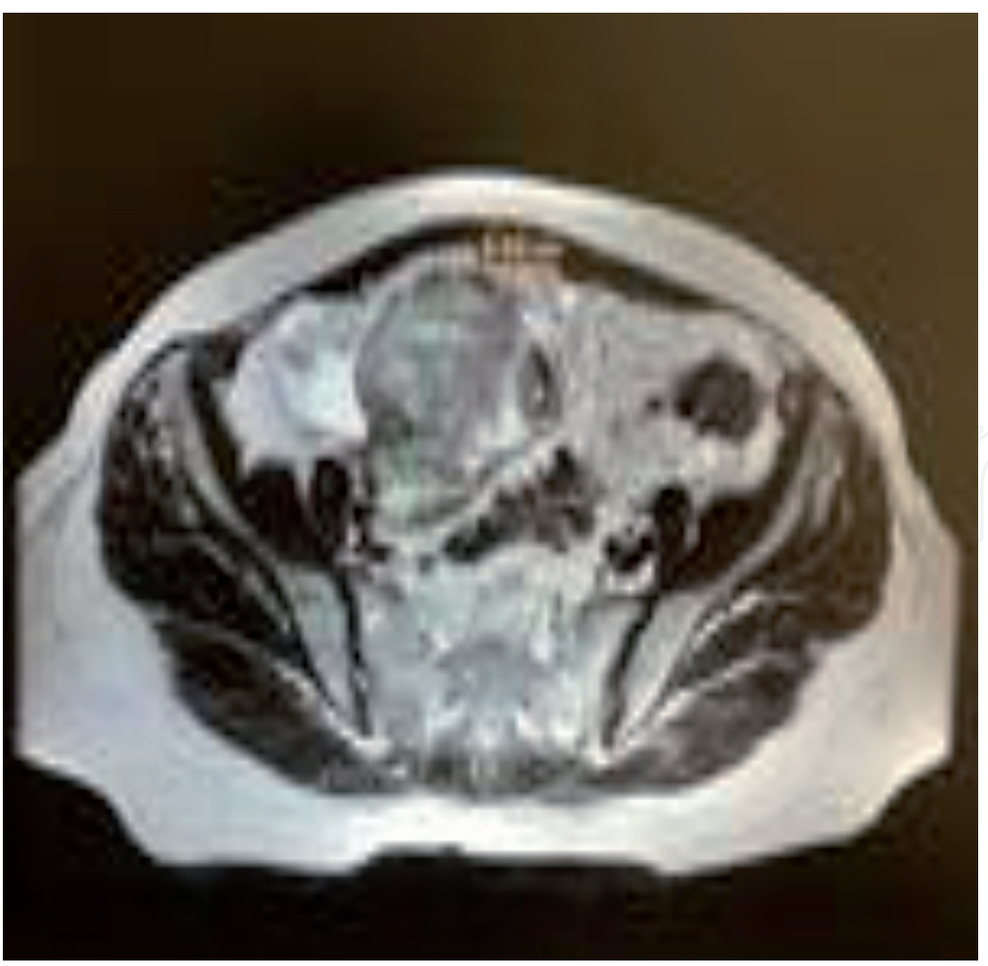

Figure 4.
Pelvic MRI scan showing large endometriomas in cross section.

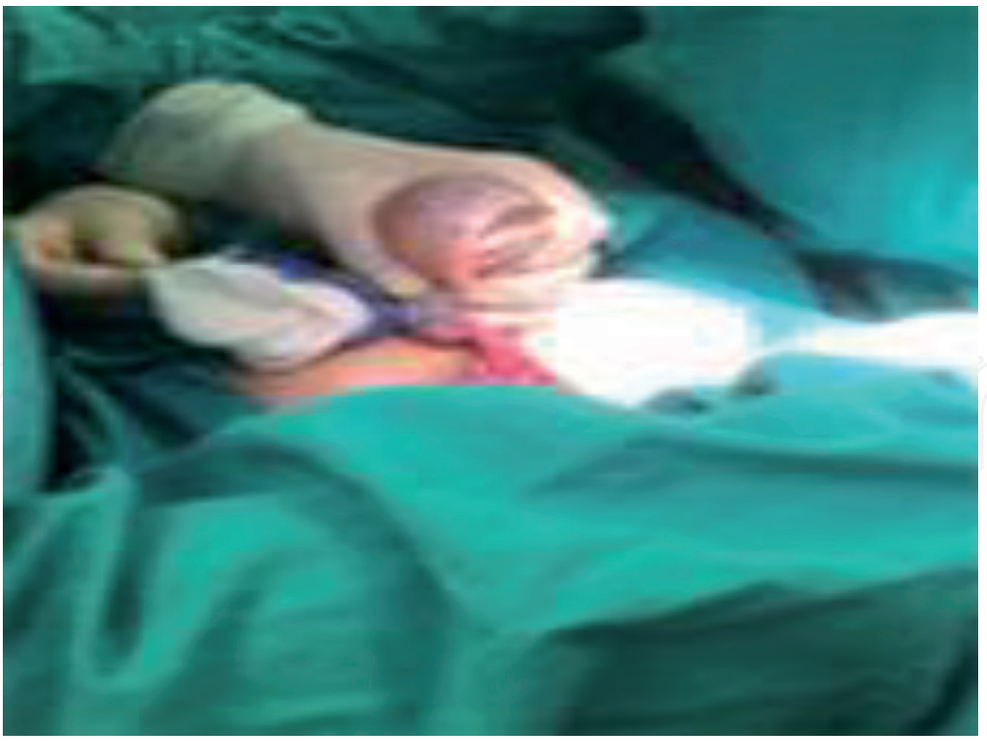

Figure 5.
Ovarian cyst during laparotomy due to ovarian torsion.

Thus, topical estrogens, together with those originating from the bloodstream, stimulate the development of endometrial foci through the estrogen receptor [37–39].

The main support for the action of aromatase in endometriosis is adrenal and ovarian androstenedione. Aromatase converts androstenedione to estrone, a weak estrogen, which must be converted to estradiol to exert a strong eff. In contrast, the enzyme 17β-hydroxysteroid dehydrogenase type II (17β-HSDIII), encoded by a different gene, inactivates estradiol by catalyzing its conversion to conversion by conversion etc.). The enzyme 17β-hydroxysteroid in endometriosis. Progesterone induces the activity of the enzyme 17β-HSDIII in endometrial cell cultures. Expression of 17β-HSD type II is absent from endometrial glandular cells during the secretory phase [37–39].

Moreover, in healthy women PP genotype was less common than PP and Pp genotype, compared with control group women and women with endometriosis. The distribution of genotypes to women in the control group was intermediate between women with endometriosis and women without disease. The frequency of the heterozygous Pp genotype did not differ between the groups. The p allele was less common than the P allele in women without the disease, compared with women with endometriosis and women in the control group from the general population. Thus, PvuII polymorphism of the ER gene is associated with the risk of developing endometriosis. The mechanism by which anonymous intron polymorphism affects the function of the estrogen receptor has not been clarified.

In the future, the clarification of this mechanism will contribute to the understanding of the pathogenesis and pathophysiology of estrogen-dependent diseases of the uterus. Loss of genetic material or DNA refers to loss of heterozygosity. Loss of heterozygosity can be caused by exogenous factors, such as carcinogens which can cause genetic damage that leads to deletions and mutations in DNA [34–39].

Mutations and deletions are particularly important in the remaining tumor suppressor genes, because if one is deleted or inactivated due to a mutation, the gene does not work properly. Such deletions can cause genetic mutations, which lead

to inactivation of genes resulting in the loss of heterozygosity. That is, the loss of heterozygosity is the result of the deletion of an area of a putative tumor suppressor gene, leading to the inactivation of that gene. Tumor suppressor genes are altered in ovarian cancers, which is consistent with the view that inactivation of these genes may play a role in the development of endometriosis. LoH heterozygous allelic mutation has been shown to occur for several DNA repair genes MSH2, MSH6, MLH1, and PMS1. The finding of LOH in regions 2p22.3-p16.1 and 3p24.2-p22, where the hMSH2 and hMLH1 genes are located, leads to the hypothesis that in some of the cases of endometriosis there may be a predisposition to cancer. Other evidence suggests the involvement of PTEN, hMLH1, p16 and INK41 in the malignancy of endometriosis. Mutation in hMLH1 methylation has been observed in four of 46 (8.6%) cases of stage III/IV endometriosis. No detectable protein expression of hMLH1 was present in these four cases, the carcinoma coexisted in two, while abnormal methylation of p16 was observed in only one case and reduced protein expression of PTEN was detected in 21 cases (15%). Both of these cases also showed hypermethylation of hMLH.

5.1 Tumor suppressor genes for endometriosis (TP53, PTEN)

Cell monoclonal expression has already been identified in endometriosis by overexpression of certain oncogenic genes (c-myc, c-erg B1). Cytogenetic abnormalities are common in malignancies. Obata and Hoshiai studied: (a) the determination of the cloning of endometriosis foci, (b) the presence of mutations in the TP53 and RASK genes, (c) the lack of heterozygosity at the sites of ovarian cancer, and (d) in endometrial carcinoma. These authors showed a lack of heterozygosity on chromosomes 9p (18%), 11q (18%) and 22q (15%). Overall, 28% of endometriosis foci showed a lack of heterozygosity at one or more sites [33–39].

There is a view that two or more genes are sequentially needed to cause endometriosis. Unlike neoplasms, not all genes involved in endometriosis need to be oncogenes or tumor suppressor genes. We believe that the first "hit" involves a gene that manifests a growing predisposition to attach and implant retrograde menstrual tissue [33–39].

This gene includes the cytoskeleton (MMPs), the cell adhesion molecule (ICAM1)), or macrophage accumulation. The second gene (`hit') may include genes that support endometrial growth, such as the estrogen receptor (ER) or steroid perturbations (CYP19). We also assume that the additional shocks involve a tumor suppressor gene, which leads to uncontrolled cell proliferation. If a tumor suppressor gene is involved in endometriosis, LoH heterozygosity could be lost at such a genetic locus. Iang et al. found the loss of LoH heterozygosity in endometriosis, by studying the chromosomal regions at 9p, 11q, and 22q in endometrial tissue. In a second study, chromosomal alterations were observed in nine of the 11 cases in which ovarian carcinoma had occurred within, or adjacent to, endometriosis [33–39].

Changes in chromosomal regions 5q, 6q, 9p, 11q, and 22q were observed in 25 - -30% of cancer-associated endometriosis cases. This is consistent with the view of some deletion in the areas where the supposed tumor suppressor genes are present, a condition seen in ovarian cancer that coexists with endometriosis. No lack of heterozygosity was found in the normal endometrium. The normal endometrium does not show molecular genetic damage. Tissue samples from endometriosis, adjacent to endometrial carcinoma, atypical endometriosis and endometrial carcinoma of the ovary were examined and common genetic changes were found with stability and joint [33–39].

These common changes did not exist in foci that were far apart. In endometrial cancers, an increased incidence of mutations in the PTEN/MMAC tumor suppressor genes were observed, while no corresponding mutations were found in serous carcinomas and clear cell carcinomas, which is evident for the tumor [33–39].

A second tumor suppressor gene studied in endometriosis is PTEN, which is located on chromosome 10q23. The gene is perturbed in a variety of cancers, including autosomal dominant disorders (Cowden syndrome and Bannayan-Zonana syndrome). PTEN mutations have been reported in endometrial cancers and in an epithelial ovarian tumor, which show some association with endometriosis but not with the serous or mucous epithelial tumors of the ovaries. Mutations involving PTEN have been observed only in endometrial tumors, at 21%. %Sato et al. [33–39] found that LoH heterozygosity was lost for PTEN in eight of 19 endometrial carcinomas (42.1%), in six of 22 clear-cell carcinomas (27.3%), and in 13 of the 23 endometrial cysts. The relationship was even greater when endometrial carcinomas were synonymous with endometriosis [33–39]

Mutations of TP53, PTEN could be associated with the transformation of benign endometrial cells into malignant cells.

In conclusion, in endometriosis there is significant damage to the molecules and to somatic cells. Also, in a percentage of cases, the lesion can continue the progression of the disease. Moreover, regions with chromosomal losses may contain important tumor suppressor genes for the pathogenesis of the disease. Lastly, in a very small percentage of cases, the disease develops into endometrial carcinoma.

6. Abdominal discomfort and pregnancy

In particular, abdominal pain during pregnancy is a the most common phenomenon due to hyperemia of the pelvic vessels or dilation of the ligaments. Due to the frequent occurrence of abdominal pain during pregnancy, it is possible not to diagnose early or even possible pathological causes of abdominal pain.

Fortunately, it is not always associated with a potential risk to pregnancy or to a woman's health, due to pelvic vascular hyperemia or dilation of the round ligaments.

The purpose of this report is to describe some areas of abdominal pain that are less troubling, which of course should be appreciated, and others that dictate immediate medical attention.

Abdominal discomfort should be associated with either a pregnancy complication or a pregnancy-related acute condition. Sometimes after the second trimester, palpable uterine contractions occasionally are painful and frequent in pregnancy.

The diagnosis of childbirth presupposes the existence of rhythmic contractions of a completely eliminated cervix and dilation of the cervix.

Physiological causes of abdominal pain in pregnancy:

Delivery.
Ectopic Pregnancy.
Ovarian cysts.
Uterine torsion.
Preeclampsia.
Placental abruption.
Acid fatty liver in pregnancy.
Musculoskeletal Pain 20–30% of women refer experience lumbar pain and sacroiliac pain.
Natural gases and bloating.

It is very likely that you will have pain caused by gas during pregnancy, due to hormones that slow down the digestion and increasing pressure of the uterus on stomach and intestines.

6.1 Constipation

Sexual intercourse remains one of most common causes of abdominal pain during pregnancy. It is known that the sperm contains prostaglandins, which can "activate" the uterus in some way after ejaculation in the vagina and cause its gentle contractions, which become even more noticeable after orgasm [40–42].

Another point, which means that abdominal pain does not seem to be associated with a serious problem, is its change depending on whether the abdominal pain decreases as soon as gas is released. Rather it is associated with a common and annoying phenomenon, which occurs during pregnancy -: bloating and constipation.

However, there are signs, which, combined with abdominal pain, during pregnancy, are a kind of "alarm" and need immediate investigation:

First of all, if 5 or more uterine contractions occur within an hour, then there is a possibility that premature birth is imminent and it is necessary to administer tocolytic treatment [40–42].

The combination of abdominal pain and vaginal bleeding at any stage of pregnancy is a point of concern. Vaginal bleeding of intense red color, in combination with abdominal pain, depending on the stage of pregnancy during which is, is a point that can hide, miscarriage, premature birth, ectopic pregnancy or placental abruption.

Finally, any abdominal pain during pregnancy, which is very intense, needs to be investigated immediately, because it may be associated with pathology of each of these pregnancies or with pathology independent of pregnancy, which needs immediate treatment. In fact, if this pain is associated with severe nausea and vomiting or fever, it may hide conditions such as appendicitis, nephrolithiasis or cholecystitis [40–42].

7. Treatment of endometriosis and pain

The treatment can be: 1) surgical, 2) conservative.

8. Conservative pharmaceutical treatment

In traditional laparoscopic surgery the percentage of recurrences or non-improvement of symptoms is about 40%. Regarding the corresponding rate of conservative drug treatment, in a study by Waller and Shaw in patients receiving GnRH for 6 months, 30% of patients reported no improvement.

Medical doctors in the field of CPP usually use analgesics or non-steroidal anti-inflammatory drugs or oral contraceptives or progesterone, but have not diagnosed endometriosis from the beginning.

The treatment that has been introduced and is considered as the second line, is GnRH analogues, usually after laparoscopy.

According to the American College of Obstetricians and Gynecologists, the treatment with GnRH analogues is an appropriate approach to failed diagnosis of the cause of pelvic pain [33–39].

9. Traditional surgery treatment

The traditional surgical treatment consists of destruction of the removal of the lesions from the ectopic endometrial tissue, mainly laparoscopically.

If the problem is more dysmenorrhea, there is a high probability of lesions related to the uterine ligaments. In this case, the destruction of the ectopic localized endometrial tissue in the uterosacral ligaments laser can be used.

Also, the cross-section of the uterosacral ligaments with bipolar diathermy has been used.

There are 3 different types of ovarian endometriosis [25].

1. Superficial hemorrhagic lesions

2. Bleeding lesions (endometriomas)

3. Deep ovarian endometriosis

1. In superficial lesions, endometriosis occurs with small cystic lesions in the ovarian cortex or small implants on the lateral surface of the ovary.

2. In endometriomas, the inner surface of a chocolate bladder is essentially the outer surface of the ovary. The hemorrhagic contents of a chocolate bladder can come from a chronic localized hemorrhage, from congested blood vessels, rather than from endometrial apoptosis.

3. In deep ovarian endometriosis there are active endometrial glands that infiltrate the ovarian cortex.

Laparoscopic treatment of ovarian endometriosis includes the following options:

1. Drainage

   The chocolate cyst is aspirated and the cyst cavity is flushed. Postoperatively, subcutaneous injection of GnRH analogues (e.g. goserelin, etc.) is recommended at 0, 4, 8, and 12 weeks [25].

2. Ablation

a. Ovarian endometriomas smaller than 3 cm in diameter treated during the diagnostic laparoscopy, if the cyst is not infiltrated into the ovary to a depth of more than 3 cm, and if the diameter of the cyst is not larger than 3 cm.

   Foci of endometriosis smaller than 1 cm in diameter can be ablated, while endometriomas of 1-3 cm in size are treated as follows: In the beginning, an area is removed from the top of the cyst, the chocolate fluid is aspirated, the ovarian cyst is flushed, and is performed ovarian cystoscopy. Subsequently the inner wall of the cyst is ablated with CO2 Laser at 40 W, until the epithelial layer is damaged (Donnez technique) [25]. However, cauterization of the inner wall of the bladder can be done with monopolar diathermy, endocoagulation or Ultracision as well.

10. Laparoscopic surgery IN CASE the ovarian torsion

Ovarian endometriomas larger than 3 cm in diameter. The protocol proposed by Donnez and applied is the following:

During the diagnostic laparoscopy, a biopsy of the endometrium is taken, the area is rinsed, and then a GnRH analog is administered for 3 months.

A second laparoscopy is performed, and, if the bladder is less than 3 cm, it is ablated as previously described. If the diameter is larger than 3 cm, a part of the cyst is removed (partial ovarian cystectomy) and the ablation of the residual endometroid ovarian cyst is followed.

10.1 Ovarian cystectomy

Described by Semm. The contents of the ovarian cyst are first aspirated, followed by an ovarian cystoscopy of its inner surface. Then, by the guidance/assistance of 2 pairs of contraction forceps, the cyst wall is detached from the ovarian tissue by rotating movements of the forceps that have captured the ovarian cyst wall, and with a movement opposite to the other forceps that has the opposite forceps.

If the ovarian cyst is firmly attached to the area of the ovary, after the remaining part of the ovary has been removed in the manner described above, the part of the ovarian cyst that is firmly attached the ovary may be exposed to CO2.

10.2 Fenestration and ablation

Fayez and Vogel argued that the removal of only one part of the endometrium, in combination with the laser ablation of the remaining part of the ovarian cyst, is associated with the development of fewer adhesions compared to the total removal of the ovarian cyst.

Absence of a thickened capsule around the endometrium makes it difficult to exclude the ovarian cyst wall, resulting in the loss of healthy ovarian tissue during ovarian cystectomy.

Thus, the exclusion of the endometrium through a Fenestration removal of 2 cm of the bladder wall and destruction of the endometrium, appears as the best therapeutic method of surgery on the ovary and the best on the ovary.

Laser laparoscopic treatment, according to Sutton's work, relieved symptoms in 71% of patients after 6 months - while 29% had no improvement. In the same study, one year after laparoscopic treatment, 56% of patients were symptom-free, while 14% had not improved or had relapsed [25, 40–45].

10.3 Additional surgical procedures to treat endometriosis

It has been shown that medication does not cure endometriosis, but makes it temporarily inactive, and that's seems to be the reason of the recurrence after discontinuation of the treatment. Various surgical approaches have been described, such as the use of the CO2 laser in relation to tissue separation and the use of bipolar diathermy.

The use of high-power CO2 laser in superpulse mode has the advantage of accurate tissue cross-section and the simultaneous achievement of hemostasis. On the contrary, the large area of thermal damage after electrocautery makes this approach less accurate, while the clear separation between healthy and abnormal tissue is more difficult. The evolution of the endoscopic equipment, which has been achieved during the last decade, as well the acquired experience and knowledge, have as a result the expansion of the indications of the laparoscopic surgery. The laparoscopic approach is an alternative to laparotomy, which is accompanied by clear and undeniable advantages. However, given that the resection of deep endometriosis is technically extremely difficult and requires vast experience, it is addressed to very few endoscopists. In addition, there are no prospective randomized trials comparing the laparoscopic approach to laparotomy [46].

Laparoscopic surgery is performed by the method of triple puncture, while, when a CO2 laser is used, the surgical laparoscope is angular 12 mm for the diode of the laser beam that is inserted through.

The surgical technique involves separating the anterior surface of the rectum from the posterior surface of the vagina and cutting or sublimating the endometriosis. First, a complete and thorough separation of the anterior surface of the rectum is performed throughout the incision, until the loose tissue of the rectal space appears.

During this surgical step the endometrial catheter is pushed downwards in order to anteriorly inflate the uterus, while some endoscopes use water for separate the fluid from the uterus. After complete preparation, it is cut from the point of adhesion to the rectum. In cases of large infiltration of the vaginal wall, it is necessary to remove part of this vagina, but also the en bloc laparoscopic removal of the entire area followed by closure of the posterior wall of the vagina. In cases of significant degree of infiltration, partial resection of the intestine and final peritoneal anastomosis laparoscopically have been described. The anterior surface of the rectosigmoid junction after surgery is not lubricated, while Interceed can be used to cover the area to be removed. Also, the excision of the intestinal part is restored laparoscopically [46].

Candiani et al. report a decrease in dyspareunia and dysmenorrhea to 60% and 40% after 3.5 years. Candiani et al. also report partial - complete remission of chronic pelvic pain in 70% of patients and a recurrence rate of 5% in 5 years [47].

The pharmaceutical approach to pelvic pain with GnRH analogues also seems to be very adequate. However, the effectiveness of laparoscopic treatment is consider as the first line therapy.

11. Conclusion

The treatment of patients with endometriosis and chronic pelvic pain (COPD) is one of the most challenging situations in every day clinical gynecology medical practice. Chronic pelvic pain is a “key point” for a woman’s quality of life.

Nowadays, the diagnosis of the endometriosis still remains difficult despite the knowledge we have acquired after years of study and research. Based on the above there is strong evidence, which supports the position that the pathogenesis of endometriosis is multifactorial, and is influenced by the interaction of genetic and environmental factors. The notion that endometriosis occurs in women with reduced immune function, which is related to genetic predisposition or environmental factors, seems more acceptable in the last decade.

The analysis of the biochemical function of the gene products will lead to a better understanding of the pathophysiology and etiology of endometriosis. New genetic markers can be used to identify high-risk women.

However, the design of epidemiological studies for predisposing factors of endometriosis is insufficient in terms of sample size and phenotype.

Basic research has laid the groundwork for new treatment protocols, particularly useful in women of childbearing age, as understanding pathology is helpful in designing a new improved but permanent treatment.

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