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Laparoscopic Surgery in the Treatment of Endometriosis

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Abstract

Endometriosis is a benign disease, which affects about 10% of reproductive age women and almost 50% of infertile women. Although every year at least 300 new articles deal with this topic, endometriosis is still a enigmatic disease starting with theories of etiopathogenesis where there is still no consensus about the major cause of endometriosis. Also there is still no consensus about the management of the disease, mainly when there is an infertile patient who is preparing for *in vitro* fertilization procedure.

Keywords: disease, endometriosis, surgery, infertility, IVF

1. Introduction

Endometriosis is classically defined as the presence of endometrial glands and stroma in ectopic locations, primarily the pelvic peritoneum, ovaries, and rectovaginal septum. This benign disease affecting 6–10% of women of reproductive age, but the prevalence is as high as 35–50% if the women are infertile or experience chronic pelvic pain. Yet endometriosis is underdiagnosed and mean latency from onset of symptoms to definitive diagnosis is 6.7 years. Endometriosis is the third main indication for hysterectomy after endometrial carcinoma and different kind of bleeding connected to myomas [1, 2].

2. Etiopathogenesis of endometriosis

The major dilemma is always understanding the etiology and pathogenesis of this condition. The past several decades have witnessed substantial progress toward unraveling the enigma associated with this disorder [3].

2.1. Retrograde menstruation theory

The theory of retrograde menstruation is proposed by Sampson in the 1920s, both attractive and supported by multiple scientific evidences. According to this theory, eutopic endometrium is transferred through patent fallopian tubes into the peritoneal cavity during menstruation. The facts supporting this theory are next: The prevalence of endometriosis is high in adolescent girls with congenital obstructed or compromised tracts. Also in up to 90% of women with patent tubes undergoing laparoscopy during the perimenstrual time of the cycle, the menstrual blood is found in the pouch of Douglas. The larger volume of retrograde menstrual fluid found in the pelvises of patients with endometriosis when compared with healthy women may increase the risk of endometriotic lesion implantation. Escape from immune clearance, attachment to peritoneal epithelium, invasion of the epithelium, establishment of local neurovascularity, and continued growth and survival are necessary if endometriosis is to develop from retrograde passage of endometrium. However, this theory has been disputed in the past since it cannot explain the occurrence of endometriosis in prepubertal girls, newborns, or males [4–6].

2.2. Coelomic metaplasia theory

According to this theory, endometriosis originates from the metaplasia of cells, which are present in the mesothelial lining of the visceral and abdominal peritoneum. Transformation of normal peritoneal cells into endometrium-like cells is supported by hormonal or immunological background. This theory may explain the occurrence of disease in girls before puberty. However, main supporting factor, estrogen, is not present in the pre-pubertal girls and therefore this condition may be different from endometriosis that is found in women of reproductive age. Ectopic endometrial tissue has also been detected in female fetuses and therefore supported the theory of abnormal embryogenesis. This theory postulates that residual cells of the Wolffian or Mullerian ducts persist and develop into endometriotic lesions that respond to estrogen. Furthermore, recent theories suggest coelomic metaplasia to be the origin of adolescent variant of severe and progressive form of endometriosis. However, possible error of this theory is found in the fact that endometriotic lesions can also be found in areas outside of the course of Mullerian duct. It is stated by many studies that undifferentiated cells can differentiate into endometrial-like tissue. This process is induced by endogenous biochemical or immunological factors [7–10].

2.3. Stem cell theory

The existence of a stem cell pool is supported by the repetitive regeneration of the endometrium after menses and re-epithelialisation of the endometrium after any kind of artificial damage

(curettage). The stem cells are supposed to exist in the basalis layer of the endometrium since the basalis layer of the endometrium is not shed with the monthly menstrual shedding of the functional layer. Recently, clonogenic cells have been identified and proposed to be involved in the formation of ectopic endometrial lesions. These cells are thought to represent the stem cell population in the human endometrium.

Stem cells are pluripotent cells. This means that they have ability to differentiate into one or several types of specialized cells. Differentiation is defined as a change in cell's gene expression, which leads to the alteration of the cell phenotype enabling the cell to have a specific function. The undifferentiated endometrial stem cells may be less responsive to ovarian steroids than the terminally differentiated cells due to lack of expression of hormone receptors. Retrograde menstruation can lead to the abnormal translocation of normal endometrial basalis, which can result in the involvement of stem cells in the formation of endometriotic deposits. Brosens et al. postulated that the uterine bleeding in neonatal girls contains a high amount of endometrial progenitor cells and that some of these cells may reside and reactivate in response to ovarian hormones later in life.

Leyendecker et al. proposed that women with endometriosis abnormally shed the endometrial basalis tissue, which, in combination with retrograde menstruation, initiates endometriotic deposits. The baboon model of endometriosis induction, where placement of the stem cell-rich endometrial basalis in the pelvic cavity resulting in 100% induction of endometriosis in all animals, may further support Leyendecker's theory.

Alternatively, these stem cells may be transported through the lymphatic or blood vessels to ectopic sites. The evidence that some of the endometrial stem cells have bone marrow origin further support the hematogenous dissemination theory of these cells. On the other hand, aberrant stem cell can relocate from the endometrium to an ectopic site and can generate endometrium-like lesions. Endometrial tissue produces several chemokines and angiogenic cytokines so this lesion can reside in the ectopic sites due to neovascularization.

The last possibility of stem cell involvement in endometriosis is the differentiation of the peritoneal, hematopoietic, or ovarian stem cells into endometrium-like tissue. Cytokines flow between uterine cavity and peritoneal cavity through the fallopian tubes. This connection may regulate the endometrium-like differentiation of resident stem cell population in the peritoneal cavity. Although possible, the reasons for such differentiation of the peritoneal stem cells into endometrium-like tissue in only up to 10% of the female population remain unexplained [11–21].

2.4. Inflammatory disease theory

There is a growing body of evidence that supports the concept of endometriosis as a pelvic inflammatory condition. In women with endometriosis, the peritoneal fluid is rich in activated macrophages and the cytokine/chemokine profile of it is different from the peritoneal fluid of women without endometriosis. In the peritoneal fluid of patients with endometriosis, one protein is identified, which is structurally similar to haptoglobin. This protein can bind to macrophages. That way it increases the production of IL-6 by macrophages and reduces

their phagocytic activity. Some other cytokines, such as MIF (macrophage migration inhibitory factor), tumor necrosis factor (TNF)-a, interleukin (IL)-1b, and IL-6, can be found in the peritoneal fluid of women with endometriosis. Also chemoattractants such as IL-8, RANTES (Regulated on Activation, Normal T Cell Expressed and Secreted), and MCP-1/CCL2 (monocyte chemoattractant protein-1) can be identified in peritoneal fluid of the women with the disease. Yet, it is not clear if the observed cytokine profile is a cause or a consequence what remains to be definitively determined.

The peritoneal microenvironment in the setting of endometriosis is significantly richer in prostaglandins, and these mediators likely play a central role in the development of the disease as well as in its clinical presentation (pain and infertility). Peritoneal macrophages from women with endometriosis express higher levels of cyclo-oxygenase-2 (COX-2) and release significantly higher amounts of prostaglandins than macrophages from healthy women [22–24].

2.5. Hormonal disease theory

Hormonal alterations may influence the ability of endometrial cells to proliferate, attach to the mesothelium, and/or escape immune-mediated clearance. The concept of endometriosis as an estrogen-dependent disorder is well supported by molecular evidences. Crucial finding is that when we compare ectopic endometriotic tissue with eutopic increased activity of the aromatase enzyme and decreased activity of 17b-hydroxysteroid dehydrogenase (17b-HSD) type 2 is found. Increased aromatase activity is the consequence of the higher production of prostaglandin E_2 (PGE$_2$), which is stimulated by higher locally bioavailable E_2 concentration. These findings support the ability of endometriotic lesions for E_2 biosynthesis and confirm treatments, which supposed to lead to hypoestrogenism [25, 26].

2.6. Autoimmune disease theory

One very attractive theory claims that endometriosis occurs when defective immune response is present. This theory is supported by the fact that autoimmune diseases are found to be more common in women with endometriosis. Women with endometriosis have a higher concentration of activated macrophages, decreased cellular immunity, and a repressed NK cell function. Ectopic endometrial cells in the peritoneum induce an inflammatory response, and macrophages and leukocytes are activated locally. This inflammatory response may have negative influence which prevents elimination of ectopic endometrial cells that now can grow in ectopic sites [27–29].

2.7. Genetic disease theory

A genetic theory of endometriosis is supported by the familiar occurrence of the disease, by the existence of endometriosis in twins, and by the fact that women are in higher risk for the disease if their mothers were affected. A great number of studies have found that endometriosis has a polygenic mode of inheritance that is likely to involve multiple loci. Some chromosomal regions were reported to be associated with the corresponding endometriosis phenotype [30].

3. Symptoms of endometriosis

Symptoms of endometriosis primarily depend on its localization. The major symptoms are infertility, chronic pelvic pain, dysmenorrhea, and dyspareunia. Dyschezia appears predominantly in the cases when the endometriosis is localized on the rectovaginal septum, while bowel involvement leads to constipation, diarrhea, and meteorism. Dysuria and hematuria are present in the cases where endometriosis involves urinary bladder, as well as symptoms of ureteral obstruction with consequential hydronephrosis and deprivation of renal function on the side of involved ureter. In the rare cases, in which endometriosis involves pleura and lungs, hemoptysis, hemathorax, or pneumothorax may appear [31].

4. Diagnosis of endometriosis

Diagnosis of endometriosis is based on a specific symptomatology, clinical, and ultrasonographic presentation, but definitive confirmation is based on a videolaparoscopic visualization of the lesions and on a histological verification of endometrial glands and stroma. Combination of videolaparoscopy and histology is a "Golden standard" in the diagnosis of endometriosis. Sole laparoscopy can lead to a greater incidence of false positive diagnoses. From the other hand, endometriosis can be unrecognized if the laparoscopy is performed by an inexperienced surgeon.

Having the knowledge about the most frequent localization and the appearance of endometriosis is crucial in laparoscopic exploration. It is important, for the didactical reasons, to distinguish three morphological forms of the disease that can be seen throughout the laparoscopy: superficial endometriotic implants, endometriomas, and deep infiltrative endometriosis (DIE).

Diagnosis of the superficial peritoneal endometriosis is based on a laparoscopic evaluation with or without histological confirmation. Superficial peritoneal implants are most often localized on the uterosacral ligaments, in the pouch of Douglas, ovarian fossa, and on the lateral pelvic wall. In the rare cases, we can find them in the upper parts of the abdominal cavity, on the bladder surface, or on the surface of the intestines. For that reason, it is important to explore the whole abdominal cavity. The diagnosis of superficial peritoneal endometriosis is purely laparoscopic visualization with or without histological verification. Superficial peritoneal implants are usually localized to the uterosacral ligaments, Douglas string, ovarian fossa, and lateral pelvic walls. Less commonly, they can be found in the upper parts of the abdomen, on the surface of the bladder, and bowel. Therefore, we must make a full inspection of the abdomen. Superficial implants can be dark (brown and black) red and white. The dark lesion is found in moderate-to-severe stages of advanced endometriosis. Red lesions are presented as red hot fields, petechiae on the peritoneum, the fields of increased vascularization, and vesicular *ekrescence*: a sign of active endometriosis. Bright field in the form of white *scarified* lesions or very light brown boxes are signs of healed or latent endometriosis. Magnification

received during laparoscopy allows visualization of very small lesions, 400–180 microns, and red to white lesions (**Figure 1**).

Figure 1. Superficial peritoneal endometriosis: dark, white and red lesions.

Diagnosis endometrioma is simple if they are over 2 cm and on the surface of the ovary (**Figure 2**). These endometriomas are detected on sonographic examination as opalescent hyperechogenic cystic changes whose descriptive echogenicity is compared with echogenicity of "the milky glass" (Ground-glass opacification—GGO—figure corresponded to pathomorphological substrate terr-chocolate cysts).However, small endometriomas can be found in as many as 48% of outside normal and enlarged ovaries. Preoperative ultrasound ovary examination is useful for the diagnosis of subcortical small endometrioma to be explored during the laparoscopy puncture.

Figure 2. Endometrioma.

The deep infiltrative endometriosis (DIE) is defined by subperitoneal invasion of endometriotic lesions with a depth of penetration of more than 5 mm. It occurs to fibromuscular structures, primarily in the rectovaginal septum and *sacro uterine* connections (nearly 70%), vagina (14.5%), digestive system (about 10%), and urinary system (about 10%).The deep infiltrative endometriosis is an entity totally different from superficial endometriosis and endometrioma. A crucial

argument in favor for this is the discovery that these lesions show no expression of HOX10 gene (associated with the endometrium), but 11 I 12 HOX genes (associated with the vagina and cervix), so it is considered that these nodules are actually the result of a tumor process, which originates from the cervix and the vagina. In less than 10% of cases, it can arise from metaplasia remains of Miller's channels [32]. The nodes of deep pelvic endometriosis can be clearly seen on MR images and endometriosis of rectovaginal septum at the transrectal sonographic examination. Endometriosis of rectovaginal septum and sacrouterine relations during the laparascopy should always be suspected in cases of obliteration of the Douglas pouch with dense adhesions and reports of "frozen pelvis" during laparoscopic exploration. However, given that these lesions are predominantly retroperitoneal, very often, they may remain unrecognized. Sometimes, just a small defect in the rectovaginal septum in the form of "peritoneal window" indicates a deep pelvic endometriosis (**Figure 3**).
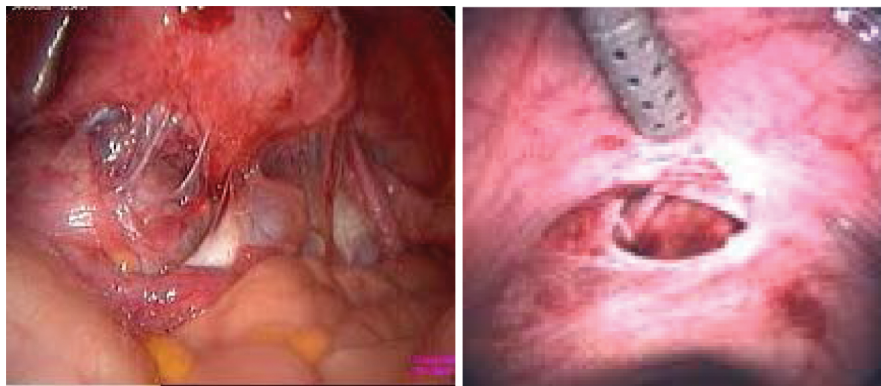

Figure 3. Deep infiltrative endometriosis: frozen pelvis and peritoneal window.

Diagnosis of endometriosis is missed in close to 7% as a lower stage in as many as 50% of cases. The classification is still mainly done by ASRM system (Revised American Society for Reproductive Medicine classification of endometriosis: 1996, 1997). Although there are many shortcomings, it is still used as the only internationally accepted classification. This classification was amended in 2005 with ENZIAN classification in terms of description and deep pelvic endometriosis and in 2010 EFI (Endometriosis Fertility Index) classification was released and it is applicable only to infertile patients [33]. A unique prognostic classification with clear guidelines still does not exist.

5. Surgery for endometriosis

Indications for video laparoscopic exploration are infertility, pain, or the presence of both symptoms. When planning the treatment, the following are taken into account: the patient's age, extent of the disease, the degree of severity of symptoms, and the desire to preserve fertility. The main guideline for the treatment is the need to preserve fertility. If this is the main symptom, the surgery is minimally extensive, especially when it comes to endometrioma. The main objective is not to reduce the ovarian reserve. If, on the other hand, the main symptom

is the pain, the operation is maximum extensive in order to resolve the pain and prevent recurrence of the disease.

The first phase of the operation is to prepare the patient, the operating room, the team, and the equipment. This phase of work often determines the fate and success of the next stage in the surgical treatment and is as important as the possession of skills in endoscopic resolution of endometriosis. Patients with endometriosis require preoperative imaging processing that is necessary to clearly define the potential lesion preoperatively. "Bowel prep" or preparation for a potential digestive tract resection or segmental resection of the lesion of the "shaving" type is necessary in the case of anticipated ureteral endometriosis, and there is a possibility for preoperative placement of ureteral catheters or the same phase delay for a moment of resolving the ureteral endometriosis. Standard is a urethral catheterization. The patient is placed in dorsal litotomic gynecological position with the upper legs that are in the plane of the body with the mandatory setting of uterine manipulator (HUMI; Rumi, Valtchev, Koh, and others). Endoscopic team consists of gynecologist-operator, assistant, assistant to the manipulator, nurse, auxiliary nurse, nurses, and anesthesia team. We emphasize that the role of operators and assistants is almost equal in terms of responsibility for the success of surgery and that philosophy of endoscopic resolution of endometriosis is based on the assumption that the surgeon operator and assistants possess full doctrinal matching tie and technical approach to surgical treatment. The lesion is "attacked" by the operator and assistant with coordinated operation, using the safe techniques for dissection where the organization of a team and the mutual anticipation is a focal point in the work. Therefore, we recommend a team approach, i.e., engagement of previously cohesive teams with fully harmonized attitudes and knowledge to work. Since the surgery for endometriosis is one of the most complex, often more demanding than oncological endoscopic surgery, it is necessary to insist on the very thorough education of a surgeon who will determine endometriosis. The equipment consists of a trocar (umbilical —optical 5 or 10 mm depending on the telescope) and extra-ancelliar (two of 5 mm or three trocars out of which the third can be paraumbilically lateral or suprapubic), a telescope of 0 or 30 degrees, a system for suction and irrigation, bipolar forceps and scissors, dissecting and grasping forceps, laser system (optional), and harmonic system (optional).

After the introduction of optical and ancilliary trocar and creation of pneumoperitoneum, follows the detailed exploration of the pelvis and abdomen with a determination of the extension of the disease. Then follows the approach to adhesiolysis and the restoration of normal anatomic relationships. Adhesiolysis can be done by hydrosection, atraumatic forceps, bipolar or monopolar forceps, and scissors or Harmonic ultracision system (Ethicon US), depending on thickness and localization of adhesions.

Peritoneal implants may coagulate (by bipolar or unipolar currency), vaporize by using laser, or by excision. Any of these three techniques can be applied to lesions smaller than 2 mm. During the coagulation of the spots, we need to know that the depth of penetration can go from 20 mm for bipolar and even to 50 mm for monopolar energy. For lesions sized 3–5 mm laser vaporization or excision is used, while for those over 5 mm only excision is used. Prior to the excision, saline solution or Ringer lactate is injected retroperitoneally around the eye lesions (grasper is previously used to catch the lesion itself and transcend it to pick the

peritoneum). This method of hydrodisection enables uplifting the peritoneal endometriotic implants from structures that may be below (ureter, blood vessels, and bladder). After this, an excision is performed around the implant of about 2 cm. This wide excision is suggested by the fact that one visible lesion of endometriosis is often accompanied by microscopic lesions around. The advantage of the excision technique is the possibility of histological verification of endometriosis.

Implants of endometriosis on the ovarian surface and endometriomas less than 2 cm can be coagulated, resected by laser, or excidated using scissors, bipolar, or accordion. These small endometriomas generally fall into type 1 endometrioma occurring by intussusception of superficial endometriosis implants, and it is very difficult to make excision on endometrioma capsules. When it comes to endometriomas larger than 3 cm, there are two surgical techniques: an ablative technique that involves drainage with aspiration of endometrioma content and then the destruction of the endometrioma wall by using laser or bipolar cautery. These techniques risk incomplete surgery and early endometrioma recurrence. Another technique involves the excision and "stripping" endometrioma capsules. Endometrioma excision begins with incision and aspiration of endometrioma content and then is repeatedly performed irrigation and aspiration of endometrioma cavity as well as inspection of the capsule walls (**Figure 4**). Incision is always done on the antimesenteric side of the ovary, as far as possible from the hilus. Then comes the dissecting plan or cleavage capsules to access the space between the capsule endometrioma and the ovarian tissue and then access the "stripping" of the capsule. This technique actually involves denudation of endometrioma using two grasping forceps that are used for traction and contractions. Applying these techniques means excessive risk of a surgery (removal of healthy ovarian tissue along with the capsule). The third technique combines the first two. Endometriomas larger than 3 cm generally fall into type 2 endometrioma occurring by infiltration of endometriosis in the corpus luteum. Depending on the depth of penetration of the implant, this technique can be simple and often complicated. In shallow penetration, endometrioma capsule can be fully removed. In deeper infiltration, insisting on complete removal of the capsule should not be the case. The existence of endometriosis in the ovary means that it threatens the healthy ovarian tissue and that the application of additional surgical techniques can significantly reduce the ovarian reserve. This is especially important for a group of infertile patients.

Although the original studies have indicated less damage to the ovary by applying ablative techniques, recent studies indicate increased rates of spontaneous conception to 2.5 times after excision techniques as well as to 4 times lower recurrence of chronic pelvic pain and 3 times lower risk of recurrence after this technique [34]. However, it has been proven that when removing the endometrioma capsule, in many cases, we have the healthy ovarian tissue with the capsule. Near the ovarian hilum, healthy ovarian tissue was found in 69% of cases, whereas away from the hilum in a small percentage only primordial follicles are found [35]. Therefore, if the "stripping" requires the application of force, it is necessary to apply a combination of excision and ablative techniques. The traction and contraction should be very carefully carried out as long as possible until the ovarian hilum is reached. Then, the process should be stopped, cut the capsule, and carefully coagulate the rest of endometrioma capsule. The point is not to

approach to hilus too close where the bleeding can be caused and subsequent necessary coagulation can completely distort the vascularization of the ovary and consequently ovarian reserve. An alternative technique is called stripping, i.e., "inversion" in whose basis comes to the initial derivation of ovarian endometrioma, ovarium inversion through derivational ovarian incision, incision of endometrioma pseudocapsule on the site of its bottom, and finding the cleavage plan, whereby the denudation of the capsule is carried in the opposite direction from classic technique that goes from edges and moves to the bottom (**Figure 5**). This technique is promoted as a potentially more optimal approach to finding the correct avascular cleavage with minimal loss of ovarian tissue and minimal disruption of ovarian vascularization. After the excision of endometrioma, infertile patients not only have a greater chance of spontaneous conception but also a good outcome in the process of in vitro fertilization. The majority of studies indicate that the damage caused by the operation is rather quantitative than qualitative and that these patients in the process of in vitro fertilization require a larger amount in the ampoules stimulation and longer stimulation but they have the same rate of pregnancy and childbirth compared to patients with tubal infertility [36]. The operation of endometrioma recurrence is technically demanding and complicated. Except in cases of symptomatic endometriosis, it should be avoided, especially in infertile patients with recurrency to be involved in the process of artificial insemination. Patients two times operated from endometriosis have much less chance of spontaneous conception as well as significantly lower rate of pregnancy in in vitro fertilization procedure [37].
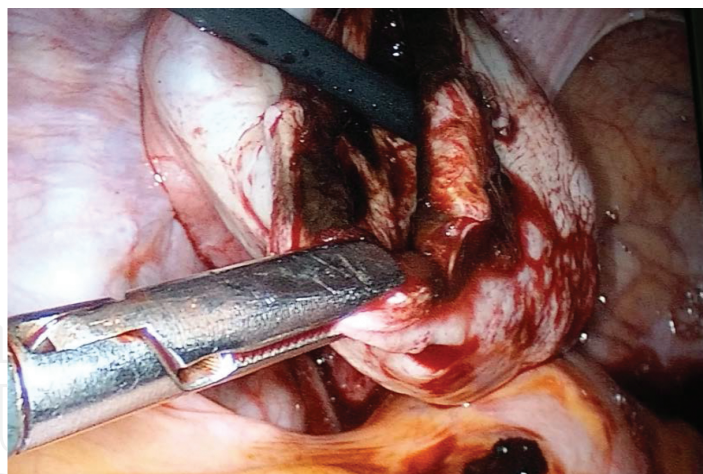

Figure 4. Incision and drainage of endometrioma.

In dealing with the DIE, the first postulate is the restoration of normal anatomic relationships in the pelvis. In order to have better access to the Douglas area, it is best to first empty the contents of endometrioma, release ovarian adhesions with the last leaf broad ligament, and temporarily fix them with a needle to the peritoneum of the anterior abdominal wall (System T-LIFT A. Wattiez-a is optimized for this maneuver). Maneuver of adnexal masses fixation is a "released hand" assistant who would otherwise be "trapped" by the need for the permanent removal of adnexal of the operative field. Then, we move on to identify the ureter. Given that

endometriosis is often a surgery of uterosacral ligaments and fibrosis medialises ureters, approach at this level is usually not safe. The simplest is to lift and incise the peritoneum at the level of "pelvic brim" (additus pelvis minoris-linea terminalis), laterally to the promontorium i a. iliacae communis and so access the ureter. Then, the downstream is followed and all the time it is lateralised. Laterally from a medial to the ureters, this internal iliac artery is the lateral aspect of pararectal fosse (Latzkov space), avascular space whose development ensures integrity of iliac vessels laterally, ureter medially and uterine arteries that is its front border and separating it from paravesicle fossa. The medial aspect of pararectal fossa is located medially to sacrouterine connections, and it is developed by peritoneal incision medial to sacrouterine relationship, sometimes even at the level of rectosigmoid and with blunt dissection, led by postulates that the fat belongs to the rectum, descend to the bottom of the medial aspect of pararectal fossa, the level of levatory panels and clearly show rectovaginal septum, rectum and vagina stub and define a proximal point of rectovaginal cleavage from which the rectal dissection is started as well as the separation from the rectum Denonvilliers's fascia at the level where the nodule DIE is present. Excision of endometriosis in this segment can be relatively simple, without compromising the integrity of the wall of the rectum or vagina. In the space of pararectal fossa, the position of the hypogastric nerve should be respected, and it is mostly freely compared to endometrioid lesions that in rare cases is affected by the disease when impossible to avoid his sacrifice. If the nodule affects vaginal mucosa or penetrates below the lamina muscularis mucosae rectum, then the rectal wall discoid resection or segmental resection of the rectum is performed, with or without resection of the vagina affected by the disease. "Rectal shaving" is a surgical principle applicable when a nodule of endometriosis rectum is superficial and does not penetrate to the mucosa. It implies "shaving" of rectal serosa with scissors along with meticulous and nonpenetrative hemostasis and with optional *reseroziranje* of the part of the rectum, which is exempt from endometriosis. Discoid resection of rectal wall involves removing the entire wall of the rectum, which is affected by the disease but not the entire sigmoid segment. Optimal embodiment is the use of circular staplers. Segmental resection of the rectum requires good mezorectal dissection and preparation for the placement of a circular stapler, which realizes the resection and T-T anastomoses at the same time.
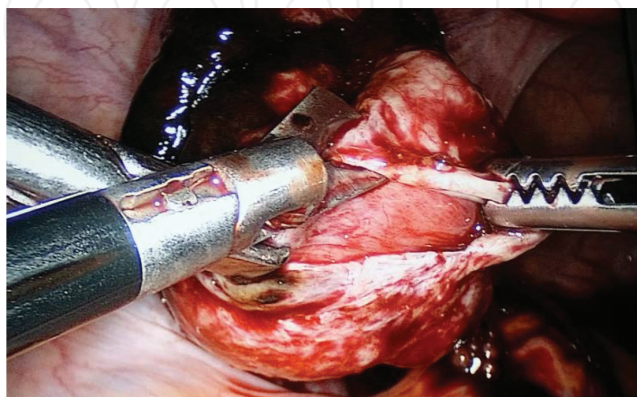

Figure 5. Stripping technique.

Ureteral endometriosis is manifested by hydroureter above the place of compression or obstruction node infiltration into the ureter with possible consequent hydronephrosis to a complete loss of kidney function on the affected side. The surgical approach involves a clear definition of the earlier mentioned positions of the ureter and its way through a small pelvis, developing pararectal and paravesicle fossa in order to clearly define the relationship of the ureter and blood vessels, and their preservation, and defining the degree and length of lesion. Compressive fibro-endometriotic "muff," which only compresses the urethra without destruction of the wall, is removed by precise and blunt dissection with the preservation of adventitial blood vessels of the ureter. When there is a "crush" lesion of the ureter or partial involvement of lumen in endometriosis nodule, the proximal and distal ureteral mobilization with ureteral resection is done, as well as the placement pig-tail ureteral (double J) catheter with termino-terminal anastomosis.

Endometriosis of the bladder may require the resection only of the part of serosa (superficial lesions), or if the nodule is transmural with mucosa affection, the resection of the bladder wall is done along with a suture in one or two or sometimes three layers with a mandatory catheterization next 2 weeks.

Treatment of deep pelvic endometriosis primarily aims to solve the pain. Regarding spontaneous conception after solving DIE, data from the literature are different, and the percentages range from 24% to 36% [38]. The impact of the success of solving DIE on VTO procedures that follow is still unclear.

Therefore, if an operation is performed on infertile patients, the treatment of endometriosis includes adhesiolysis, excision and coagulation of superficial endometriosis implants, stripping of endometrioma, and resolving of deep infiltrative endometriosis especially when infertility is associated with pain. When it comes to addressing chronic pelvic pain in patients who have completed the reproduction and do not want to preserve fertility, radical treatment should be taken into consideration. Extension of the disease does not affect much the intensity of the pain so that the removal of adhesions and endometriosis spots will, in some women, resolve the problem, especially if a deep pelvic endometriosis is solved, whereas the others require adnexectomy or hysterectomy. One optional technique is presacral neurectomy. This technique involves access to hypogastric plexus at the level of promontorium and removal of all fibers in interiliac triangle.

Given the fact that the endoscopic surgery for endometriosis is one of the most challenging and the most invasive in terms of the manipulation of tissues and organs of the pelvis, and that in the process of the excision of the disease, very vulnerable structures (iliac vessels, uterine blood vessels, ureters, bladder and bowel) are often approached, the percentage of complications (intra or perioperative) is greater than in the treatment of other benign conditions.

Blood vessel injuries occur most commonly during the excision of endometriosis from the lateral compartment during adhesiolysis and ovarium deliberation, which are captured in ovarian fossae or during the deliberation of the ureter. The most common reason is the inadequate implementation of energy or force of traction on the wall of the blood vessel or adhesiolysis method (sharp precise dissection or large force of blunt dissection). Injuries could

be resolved with a ligature of blood vessels (if they are not vital and whose "sacrifice" does not jeopardize the function) or with wall suture (iliac blood vessels, aorta, and vena cava). Complications are endoscopically solvable as well, if the team that solves them is familiar with the endoscopic method of treatment.

Intestine injuries (usually rectosigmoid segment or the small intestine) occur during adhesiolysis or inadequate application of energy or the wrong assessment of the initial approach to solve endometrioid nodes (selection of rectal shaving in case the disease spread to the entire wall of the rectum and when it is necessary to open the intestinal lumen). Intraoperative complications are primarily unrecognized lesions of the intestinal wall and rectovaginal fistula or intestinal obstruction is postoperative.

Urinary complications are the result of removing the disease from the wall of the bladder or ureter complications but they are considered complications only in the case of same result of inadequate implementation of operational strategy in relation to the real state of the disease (segmental resection of the ureter with the ureteral endometriosis is not considered a complication but a planned surgical treatment, while ureteral resection because of the incidental ureteral injuries due to an inadequate use of force or energy is a complication).

Loss of ovarian reserves and POF is considered a very significant complication of fertility-preserving operative treatment, and in this sense there is a consensus that provides for the abandonment of excision surgery at the ovarium level on endometrioma less than 2 cm or those that affect almost the entire volume of the ovary or ovarian hilus and in patients with preoperative low ovarian reserve (estimated levels of AMH, AFC). Then, the derivative of endometrioma with partial destruction of zones of endometriosis in pseudocapsule is a surgical method of choice. In other cases, it is decided on the stripping of the capsule with all the above principles to achieve preservation of ovarian vascularization and residual ovarian function.

In order to prevent "seeding" or implantation of endometrioid tissue in the anterior abdominal wall at the site of trocar-incision, as one of the possible complications, it is an imperative to use a ENDOBAG when extracting the material at the end of the operation (pseudocapsule of endometrioma, implants, and nodules). Extraction way is usually on the side or umbilical incision on the skin [39].

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References

[1] L. C. Giudice. Endometriosis. The New England Journal of Medicine. 2010; 362(25): 2389–2398

[2] M. M. Carneiro, I. D. D. S. Filogônio, L. M. P. Costa, I. De Ávila, and M. C. Ferreira, Accuracy of clinical signs and symptoms in the diagnosis of endometriosis. Journal of Endometriosis. 2010; 2(2):63–70

[3] R. O. Burney and L. Giudice. Pathogenesis and pathophysiology of endometriosis. Fertility and Sterility. 2012;98(3):511–519

[4] P. R. Koninckx, D. Barlow, and S. Kennedy. Implantation versus infiltration: the Sampson versus the endometriotic disease theory. Gynecologic and Obstetric Investigation. 1999; 47(1):3–10

[5] P. R. Koninckx, A. Ussia, L. Adamyan, A. Wattiez, and J. Donnez. Deep endometriosis: definition, diagnosis, and treatment. Fertility and Sterility. 2012; 98(7):564–571

[6] J. A. Sampson. Heterotopic or misplaced endometrial tissue. American Journal of Obstetrics and Gynecology. 1925; 10(5):649–664

[7] A. Valentijn, H. Al-Lamee, K. Palial, et al. SSEA-1 Isolates human endometrial basal glandular epithelial cells: phenotypic and functional characterisation and implications in pathogenesis of endometriosis. Human Reproduction. 2013; 28(4): 2695–2708

[8] W. P. Dmowski and E. Radwanska. Current concepts on pathology, histogenesis and etiology of endometriosis. Acta Obstetricia et Gynecologica Scandinavica. 1984; 63(123):29–33.

[9] I. Brosens and G. Benagiano. Is neonatal uterine bleeding involved in the pathogenesis of endometriosis as a source of stem cells. Fertility and Sterility. 2013; 100(22):622–623

[10] P. Gruenwald. Origin of endometriosis from the mesenchyme of the celomic walls. American Journal of Obstetrics and Gynecology. 1942; 44(3):470–474

[11] H. M. Albertsen, R. Chettier, P. Farrington, and K. Ward. Genome-wide association study link novel loci to endometriosis. PLoS One. 2013; 8: Article ID e5825, 22–25.

[12] K. E. May, J. Villar, S. Kirtley, S. H. Kennedy, and C. M. Becker. Endometrial alterations in endometriosis: a systematic review of putative biomarkers. Human Reproduction Update. 2011; 17(5):637–653

[13] Y. Afshar, J. Hastings, D. Roqueiro, J. W. Jeong, L. C. Giudice, and A. T. Fazlebas. Changes in eutopic endometrial gene expression during the progression of experimental endometriosis in the baboon, *Papio anubis*. Biology of Reproduction. 2013; 88(3):44

[14] M. A. Khan, J. Sengupta, S. Mittal, and D. Ghosh. Genome-wide expressions in autologous eutopic and ectopic endometrium of fertile women with endometriosis. Reproductive Biology and Endocrinology. 2012; 10(8):84

[15] S. Tsuji, M. Yoshimoto, K. Takahashi, Y. Noda, T. Nakahata, and T. Heike. Side population cells contribute to the genesis of human endometrium. Fertility and Sterility. 2008;90(4):1528–1537

[16] H. A. Padykula. Regeneration in the primate uterus: the role of stem cells. Annals of the New York Academy of Sciences. 1991; 622:47-56.

[17] J. A. Deane, R. C. Gualano, and C. E. Gargett. Regenerating endometrium from stem/progenitor cells; is it abnormal in endometriosis, Asherman's syndrome and infertility? Current Opinion in Obstetrics and Gynecology. 2013; 25:193–200

[18] K. Kato. Stem cells in human normal endometrium and endometrial cancer cells: characterization of side population cells. Kaohsiung Journal of Medical Sciences. 2012;28(2):63-71

[19] F. R. Oliveira, C. D. Cruz, H. L. del Puerto, Q. T. M. F. Vilamil, F. M. Reis, and A. F. Camargos. Stem cells: are they the answer to the puzzling etiology of endometriosis? Histology and Histopathology. 2012;27(1):23–29

[20] C. E. Gargett, R. W. S. Chan, and K. E. Schwab. Hormone and growth factor signaling in endometrial renewal: role of stem/progenitor cells. Molecular and Cellular Endocrinology. 2008;288(1-2):22-29

[21] H. S. Taylor. Endometrial cells derived from donor stem cells in bone marrow transplant recipients. Journal of the American Medical Association. 2004;292(1):81-85

[22] H. Masuda, Y. Matsuzaki, E. Hiratsu et al. Stem cell-like properties of the endometrial side population: implication in endometrial regeneration. PLoS One. 2010;5(4): Article ID e10387, 121–123

[23] S. Gupta, A. Agarwal, N. Krajcir, and J. G. Alvarez. Role of oxidative stress in endometriosis. Reproductive BioMedicine Online. 2006;13(1):126-134

[24] A. Augoulea, A. Alexandrou, M. Creatsa, N. Vrachnis, and I. Lambrinoudaki. Pathogenesis of endometriosis: the role of genetics, inflammation and oxidative stress. Archives of Gynecology and Obstetrics. 2012; 5(1):1–5

[25] C. Parente Barbosa, A. M. Bentes De Souza, B. Bianco, and D. M. Christofolini. The effect of hormones on endometriosis development. Minerva Ginecologica. 2011;63(4): 375–386

[26] J. J. Kim, T. Kurita, and S. E. Bulun. Progesterone action in endometrial cancer, endometriosis, uterine fibroids, and breast cancer. Endocrine Reviews. 2013;34(2):130-162

[27] N. Sinaii, S. D. Cleary, M. L. Ballweg, L. K. Nieman, and P. Stratton. High rates of autoimmune and endocrine disorders, fibromyalgia, chronic fatigue syndrome and

atopic diseases among women with endometriosis: a survey analysis. Human Reproduction. 2002;17(10):2715–2724

[28] J. Sikora, A. Mielczarek-Palacz, and Z. Kondera-Anasz. Role of natural killer cell activity in the pathogenesis of endometriosis. Current Medicinal Chemistry. 2011;18(2):200-208.

[29] Y. Osuga, K. Koga, Y. Hirota, T. Hirata, O. Yoshino, and Y. Taketani. Lymphocytes in endometriosis. American Journal of Reproductive Immunology. 2011;65(1):1-10

[30] H. M. Albertsen, R. Chettier, P. Farrington, and K. Ward. Genome-wide association study link novel loci to endometriosis. PLoS One. 2013;8:Article ID e58257

[31] Bellelis P, Dias JA, Jr., Podgaec S, Gonzales M, Baracat EC and Abrão MS. Epidemiological and clinical aspects of pelvic endometriosis—a case series. Revista de Associacao Medica Brasileira 2010;56:467–471.

[32] Kulp, J. L., Cakmak, H. and Taylor, H. S. HOX Genes and Endometriosis, in Endometriosis: Science and Practice (eds L. C. Giudice, J. L. H. Evers and D. L. Healy), 2011. Wiley-Blackwell, Oxford, UK.

[33] Adamson GD. Endometriosis Fertility Index: is it better than the present staging systems? Current Opinion in Obstetrics & Gynecology. 2013; 25(3):186–92.

[34] Hart RJ, Hickey M, Maouris P, Buckett W. Excisional surgery versus ablative surgery for ovarian endometriomata. Cochrane Database Systematic Review 2008; 2: CD004992.

[35] Muzii L, Panici PB. Combined technique of excision and ablation for the surgical treatment of ovarian endometriomas: the way forward? Reproduction Biomedicine Online. 2010;20(2):300–2.

[36] Ruiz-Flores FJ and Garcia-Velascoa JA. Is there a benefit for surgery in endometrioma associated infertility? Current Opinion in Obstetrics & Gynecology 2012;24(3):136–140

[37] Berlanda D, Vercellini P and Fedele L. The outcome of repeat surgery for recurrent symptomatic endometriosis. Current Opinion in Obstetrics & Gynecology 2010; 22(4): 320–325

[38] Kondo W, Bourdel N, Tamburro S, Cavoli D, Jardon K, Rabischong B, Botchorishvili R, Pouly J, Mage G, Canis M. Complications after surgery for deeply infiltrating pelvic endometriosis. BJOG 2011; 118(3):292–8.

[39] deZiegler D, Borghese B, Chapron C. Endometriosis and infertility: pathophysiology and management. Lancet 2010;376(9742):730–8.