ABDOMINAL WALL AND CESAREAN SCAR ENDOMETRIOSIS: REPORT OF A TWO CASE
Eugeniu Cazacu, Ala Cerbadji, Ruslan Pretula, Olga Focșa
Department of Morphopathology State Medical and Pharmaceutical University „Nicolae Testemitanu”

Summary
Endometriosis is described as the presence of functioning endometrial tissue (glands and stroma) outside the uterine cavity. The most common location is within the pelvis. However, extra pelvic endometriosis is a fairly uncommon disorder and difficult to diagnose. It can sometimes occur in a surgical scar. Scar endometriosis is a rare condition and difficult to diagnose. It mostly follows obstetrical and gynecological surgeries. It presents as a painful, slowly growing mass in or near a surgical scar. We report two cases of abdominal wall scar endometriosis after cesarean section and strangulated umbelical hernia. Consequently the pathogenesis, diagnosis, morphology and complication are discussed.

Rezumat

Introduction
Endometriosis (from endo, "inside", and metra, "womb") is a gynecological medical condition in which endometrial-like cells appear and flourish in areas outside the uterine cavity. Endometriosis has been known for more than 300 years (Rokitansky, 1860), at the beginning as a purely gynecological disorder, caused by functionally active ectopic endometrial areas located in the genital tract or adjacent pelvic structures and was defined as the presence and proliferation of endometrium outside the uterine cavity, commonest site being pelvis. However, extra pelvic endometriosis a fairly uncommon disorder and difficult to diagnose. Nowadays endometriosis is
considered as a “career woman’s” disease, a disease of industrialization, etc., statistically occurring at a rate of 10%, and affecting mostly young women. In this paper we describe two cases of abdominal wall scar endometriosis and strangulated umbelical hernia.

**Patients, Methods and Results**

**Case No.1** A 52-year-old woman, was admitted to the surgical department with diagnosis: „Strangulated ombelical herniar. Small intestine perforation. Flegmon of hernial sac. Acute intestinal occlusion.” She underwent an operation to the in patient department: Herniotomia. Plastia Mayor and resection of small intestine with anastomosis FT. Macroscopically, Small intestine segment 25 cm, the serous was smooth opaque. Seminal sac with umbilical cord removed in block. The 8 cm cystick section with bothers smooth yellow – fluid contents.

![Image](image1.png)

**Fig.1.** The anterior abdominal wall, umbilical region endometriosis: a) endometrioid cyst wall vessels with hyperemia and hemorrhage; b) chronic inflammatory infiltration and deciduous reaction; c) deposits of hemosiderin. Coloration hematoxylin and eosin; a – objective 10, eyepiece – 10, b, c – objective 20, eyepiece – 10.

**Case No.2**

A 31-year-old woman, gravida:2, parity:2, was admitted in the obstetric and gynecology department with diagnosis: „39 weeks pregnancy nr. II (scar endometriosis)”.

![Image](image2.png)

**Fig.2.** Postcesariane scar endometriosis: a) scar tissue in deciduous; b) focus of myxomatosis with deciduous tissue; c) deciduous tissue surrounded by collagen bundles fiber. Coloration hematoxyllin and eosin; a – objective 10, eyepiece – 10, b, c – objective 20, eyepiece – 10.
Complications: scar endometriosis.


Obstetrical anamnesis:
I – 2001 cesarean section (b) – 1,900.
II – 2009 present pregnancy (cesarean section).

Macroscopically, Laminal fragment of skin with subcutaneous tissue, 5 – 6 cm, thick white – yellowish colour, flaccid content.

Discussion
Endometriosis is a disease that affects women during their childbearing age. It exists in approximately 8-15% of all menstruating women. It is defined as the presence of functional endometrial tissue in anatomical location outside the uterus. Its usual site is the pelvic peritoneum and pelvic organs. Extrapelvic or extragonadal endometriosis is rare and was reported to affect the appendix, small and large bowel, inguinal canal, umbilicus and the abdominal wall as localized disease or as an extension of the pelvic disease. A scar related endometrioma usually presents as a mass or a nodule related to a gynecological operation. It is noticed usually around one to 3 years after the procedure.

Materials and methods
The surgically excised materials were placed in 10% formalin solution overnight. The surgical borders were painted with ink. Tissue sections, 0.25 cm thick, were formalin – fixed and paraffin – embedded. The slides were stained with hematoxylin and eosin and PAS stain for light microscopic examination.

Epidemiology
Endometriosis is found predominantly in women of childbearing age. The mean age at diagnosis is 25-29 years, but it is often greater in women who present with infertility rather than pelvic pain. Endometriosis is not uncommon among adolescents. Approximately half of women under 20 years of age who have chronic pelvic pain or dyspareunia have the disease. Obstructive mullerian duct abdomalities of the cervix or vagina account for most cases of endometriosis in girls under the age of 17 years. About 5% of endometriosis cases are seen in postmenopausal women, and exogenous estrogen replacement therapy is suggested to play a role. In rare cases, men undergoing long-term estrogen therapy may be affected.

Etiology, pathogenesis
The etiology of the disease has not yet been fully demonstrated. Of the many theories considered along the years, only two have remained valid. The theory of celomic epithelium metaplasia sustains that endometriosis might originate in peritoneal endothelium metaplasia which, in its turn, derives from celomic epithelium, the same as genital organs do. The cause of such metaplasia might be hyperestrogenemia, in the presence of which mesenchymal elements, either covered or not by epithelium layer, would be likely to turn itself into endometrium or inflammation (embryonal celomic cells are supposed to turn into endometrium under the impact of inflammatory phenomena).

The third is the theory of endometrial origin backed up by three hypotheses:
Three theories of histogenesis have been proposed: (a) metastatic theory (retrograde menstrual implantation, vascular and lymphatic spread, and intraoperative implantation), (b) metaplastic theory, and (c) induction theory (Fig 1)
Figure 1. Drawing depicts the potential mechanisms of endometriosis pathogenesis, including retrograde menstruation (a), lymphatic spread (b), hematogenous spread (c), and coelomic metaplasia (d). Induction of undifferentiated mesenchyma by unidentified endometrial substances is also theorized.

Figure 2. Drawing illustrates common sites of endometriosis.

- The “back flow” theory (retrograde menstruation) upheld by John Sampson, 1921: endometrial fragments in menstrual blood are taken over, through the tubal pavilion, into the peritoneal cavity, where they get affixed.
- The theory of vascular metaplasia, ratified by Sampson and Halban, explains the other endometrial locations: the proliferation reaching myometrial interstices may penetrate blood and lymph vessels causing real endometrial embolism in any tissue or organ of the body.

In addition, researchers are presently investigating the role of growth factors, immunity, and other mechanisms that may contribute to the development of this disorder.

The most widely accepted theory is that endometriosis results from metastatic implantation from retrograde menstruation. The theory assumes transportation of endometrial tissue from the uterus in a retrograde fashion into the peritoneal cavity. The endometrial cells remain viable and implant on serosal surfaces outside the uterus. The occurrence of retrograde menstruation has been documented with diagnostic laparoscopy and studies of peritoneal dialysis fluid. Up to 90% of women have bloody peritoneal fluid during the perimenstrual period (15–17). Investigators have shown the in vitro growth potential of shed endometrium and have demonstrated viable endometrial cells in peritoneal fluid. Further evidence for this theory is suggested by the anatomic pattern of the disease within the dependent areas of the pelvis (Fig 2).

Endometriosis is also seen with greater frequency in women with excessive retrograde flow due to obstructive abnormalities of müllerian duct development. Other possible routes of metastatic spread include transport of endometrial cells to distant sites via the blood stream or lymphatic channels or iatrogenically during surgery or needle biopsy.
A second theory of histogenesis is that of metaplastic differentiation of serosal surfaces (coelomic epithelium) or müllerian remnant tissue. Both endometrial and peritoneal cells derive from the coelomic wall–epithelium. The theory suggests the possibility of peritoneal cells differentiating into functioning endometrial cells. The strongest evidence for this theory is the demonstration of endometriosis in women lacking functional eutopic endometrium (eg, those with Turner syndrome, gonadal dysgenesis, uterine agenesis) and in men. In the rare cases of endometriosis occurring in men, they have usually received high doses of estrogen, which is sometimes done in the treatment of prostate carcinoma. In these men, the cause is possibly hyperplasia and spread of endometrial tissue from the prostatic utricle (a müllerian remnant). A third theory, the induction theory of endometriosis, is a combination of the first two. It suggests that shed endometrium releases substances that induce undifferentiated mesenchyma to form endometriotic tissue. These substances have not been identified, but animal research has shown the formation of endometrial glands (but not stroma) when experiments have been performed to test this theory.

**Staging of endometriosis**

Staging the disorder helps physicians to formulate a treatment plan and evaluate response to therapy. According to the American Society for Reproductive Medicine, endometriosis may be classified as stage I (minimal), II (mild), III (moderate), or IV (severe), based on number, location, and depth of implants and presence of filmy or dense adhesions (Tables 1).

<table>
<thead>
<tr>
<th>Stage</th>
<th>Disease</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Minimal</td>
<td>A few superficial implants</td>
</tr>
<tr>
<td>II</td>
<td>Mild</td>
<td>More and slightly deeper implants</td>
</tr>
<tr>
<td>III</td>
<td>Moderate</td>
<td>Many deep implants, small endometriomas on one or both ovaries and some filmy adhesions</td>
</tr>
<tr>
<td>IV</td>
<td>Severe</td>
<td>Many deep implants, large endometriomas on one or both ovaries, and many dense adhesions, sometimes with the rectum adhering to the back of the uterus</td>
</tr>
</tbody>
</table>

If the fimbriated end of the fallopian tube is completely enclosed, change the point assignment to 16. Staging: Stage I (minimal):1-5; stage II (mild): 6-15; stage III (moderate): 16-40; stage IV (severe): 40

Another staging system is based primarily on the presence and severity of pelvic pain. However, because intraobserver and interobserver variability is high in the staging system, a more reliable method of staging is being sought.

**Morphology**

Endometriosis typically appears as superficial “powder burn” or “gunshot” lesions on the ovaries, serosal surfaces and peritoneum – black, dark – brown, bluish purplish lesions, nodules or small cysts containing old hemorrhage surrounded by a variable extent of fibrosis. Atypical or ‘subtle’ lesions are also common, including red implants (petechial, vesicular, polypoid, hemorrhagic, red flame – like) and serose or clear vesicles. Other appearances include white plaques or scarring and yellowish brown peritoneal discoloration of the peritoneum. Endometriomas usually contain thinck fluid like tar; such cysts are often densely adherent to the peritoneum of the ovarian fossa and the surrounding fibrosis may involve the tubes and bowel. Deeply infiltrating endometriotic nodules extend 5mm beneath the peritoneum and may involve the uterosacral ligaments, vagina, bladder, or ureters. The depth of infiltration is to the type and severity of symptoms.
Complication of endometriosis

Complications of endometriosis include the following:

- The bleeding can form bands of scar (adhesions) that can attach to the organs in the pelvis and abdomen.
- Reduced fertility that may have no obvious cause or may be caused by adhesions forming on or near to your ovaries or fallopian tubes.
- An increased risk of miscarriage or given birth prematurely.
- Cysts can bleed or rupture, causing severe pain.
- Endometriosis of the intestine can cause the bowel to become blocked or twisted.
- An increased risk of certain types of cancer, particularly ovarian.

Malignant Risk

Malignant change of endometriosis in a cesarean scar is rare. Long-standing recurrent scar endometriosis could undergo malignant changes and clinician should be aware. Only 21.3% of cases of malignant transformation of endometriosis occur at extragonadal pelvic sites, 4% of cases in scars after laparotomy. Clear-cell carcinoma is the most common histological subtype, followed by endometrioid carcinoma. In the literature, survival rate reaches only 57% after a short follow-up of 20 months. Treatment is radical surgical resection followed by prosthetic abdominal wall repair. Compared with endometriosis-associated ovarian carcinoma, the prognosis of abdominal scar complication is poor.

Conclusion

- Endometriosis is a mystery tour as it requires decision making at every stage by the physician and the patient.
- Endometriosis still remains one of the most-investigated disorders in gynecology. So is one of the highest priorities for research.

References

2. Romanian Journal of Morphology & Embryology
10. Marian D. Damewood, Pathophysiology and management of endometriosis.