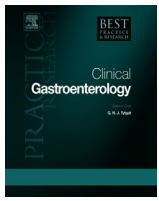
Best Practice & Research Clinical Gastroenterology 28 (2014) 53–67

Contents lists available at ScienceDirect

Best Practice & Research Clinical Gastroenterology



5

Multidisciplinary laparoscopic treatment for bowel endometriosis

Albert M. Wolthuis, MD, Adjunct Head of Clinic [a,*], Carla Tomassetti, MD, Adjunct Head of Clinic [b]

[a] Department of Abdominal Surgery, University Hospital Leuven, Herestraat 49, 3000 Leuven, Belgium
[b] Leuven University Fertility Centre, Department of Obstetrics and Gynaecology, University Hospital Leuven, Herestraat 49, 3000 Leuven, Belgium

Keywords:
Endometriosis
Colorectal endometriosis
Deeply infiltrative endometriosis
Laparoscopy
Diagnosis
Treatment

A B S T R A C T

Endometriosis is a handicapping disease affecting young females in the reproductive period. It mainly occurs in the pelvis and affects the bowel in 3–37%. Endometriosis can cause menstrual and non-menstrual pelvic pain and infertility. Colorectal involvement results in alterations of bowel habit such as constipation, diarrhoea, tenesmus, and rarely rectal bleeding. A precise diagnosis about the presence, location and extent is necessary. Based on clinical examination, the diagnosis of bowel endometriosis can be made by transvaginal ultrasound, barium enema examination and magnetic resonance imaging. Multidisciplinary laparoscopic treatment has become the standard of care and depending on size of the lesion and site of involvement full-thickness disc excision or bowel resection is performed by an experienced colorectal surgeon. Anastomotic complications occur around 1%. Long-term outcome after bowel resection for severe endometriosis is good with a pregnancy rate of 50%.

Introduction

Endometriosis is defined as the presence of endometrial-like tissue outside the uterus and it induces a chronic inflammatory reaction. Three clinical presentations have been described [1]: peritoneal endometriosis, endometriotic ovarian cysts (i.e. endometriomas), and deeply infiltrative endometriosis

* Corresponding author. Department of Abdominal Surgery, University Hospital Gasthuisberg Leuven, Herestraat 49, 3000 Leuven, Belgium. Tel.: +32 16 34 42 65; fax: +32 16 34 48 32.
E-mail addresses: albert.wolthuis@uzleuven.be, amwolthuis@hotmail.com (A.M. Wolthuis).

1521-6918/$ – see front matter © 2013 Elsevier Ltd. All rights reserved.
http://dx.doi.org/10.1016/j.bpg.2013.11.008

(DIE). DIE is defined as the invasion of anatomical structures and organs deeper than 5 mm beyond the peritoneum. DIE can cause a complete distorsion of the pelvic anatomy and it mainly involves the uterosacral ligaments, the vagina, the bladder, the rectum, and rectosigmoid colon [2]. This review specifically focuses on the diagnosis and treatment of DIE of the bowel.

Bowel endometriosis

Epidemiology

Endometriosis occurs during the active reproductive period. Endometriotic implants depend on ovarian steroids for growth and maintenance [3]. Therefore, the disease is uncommon in pre- or post-menarchal girls and rare in post-menopausal women who are not on hormonal replacement therapy. Little is known about the prevalence of endometriosis. It is estimated that up to 15% of all women of reproductive age have endometriosis. About one-third of infertile women suffer from endometriosis [4,5]. One population-based study estimated that 6% of premenopausal women have endometriosis [6]. Japanese women have a doubled incidence of endometriosis compared to Caucasian women [7]. Nulliparity, polymenorrhea, hypermenorrhea and early menarche are important risk factors for endometriosis [8]. It is also associated with a taller, thinner body habitus and a lower body mass index [9]. Endometriosis affects the bowel in 3–37% of all cases [10] and in 90% of these cases the rectum or sigmoid colon are involved [11–14].

Pathogenesis

Three theories have been proposed to explain the pathogenesis of endometriosis: ectopic transplantation of endometrial tissue, coelomic metaplasia, and the induction theory. No single theory can account for the location of endometriosis in all cases [15].

The most widely accepted and supported explanation is the transplantation theory [16], which suggests that endometriosis is caused by the seeding or implantation of endometrial cells by transtubal regurgitation during menstruation. These ectopic cells can attach to, and invade the pelvic peritoneal membrane. Retrograde menstruation occurs in 70%–90% of women [17,18] and leads to the presence of endometrial cells in the peritoneal fluid [19]. However, this mechanism fails to explain why not all women with retrograde menstruation develop endometriosis. The implantation theory suggests the establishment of endometriosis in the peritoneal cavity requiring endometrial cells to complete a process of adhesion, invasion and proliferation. This requires interaction between dislocated endometrial cells and peritoneum. The contribution of the pelvic peritoneal mesothelium to the development and progression of endometriosis was recently reviewed [20], and constitutes of the following: differential expression of peritoneal mesothelial adhesion factors, changes in peritoneal mesothelial cell phenotype (loss of tight junctions) allowing ectopic cells to bind to or invade into the extracellular matrix, epithelial-to-mesenchymal transition of peritoneal mesothelial cells leading to an increase in lesion invasion and formation of fibrotic tissue. The menstrual reflux theory combined with the clockwise peritoneal fluid current explains why endometriosis is predominantly located on the left side of the pelvis (refluxed endometrials cells get stuck more easily in the rectosigmoidal area) [21] and why diaphragmatic endometriosis is found more frequently on the right side (refluxed endometrial cells are stuck by the falciform ligament) [22].

According to anatomical, surgical, and pathological findings, DIE seems to originate intraperitoneally rather than extra-peritoneally [15]. Also, the lateral asymmetry in the occurrence of ureteral endometriosis is compatible with the menstrual reflux theory and with the anatomical differences of the left and right hemipelvis [21], supporting the notion that endometriosis starts as peritoneal disease [15]. Extra-pelvic endometriosis, although rare (1%–2%), potentially may result from vascular or lymphatic dissemination of cells to many gynaecologic (vulva, vagina, cervix) and non-gynaecologic sites. The latter include bowel, lungs and pleural cavity, skin (episiotomy or other surgical scars, inguinal region, extremities, umbilicus), lymph glands, nerves, and brain [15,23].

The coelomic metaplasia theory, i. e. transformation (metaplasia) of coelomic epithelium into endometrial tissue, has been proposed as a second mechanism for the origin of endometriosis. Up to