

## Progression of bowel endometriosis during treatment with the oral contraceptive pill

Simone Ferrero · Giovanni Camerini ·  
Pier Luigi Venturini · Ennio Biscaldi ·  
Valentino Remorgida

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### Introduction

Bowel endometriosis affects between 3.8% and 37% of women with endometriosis [1]. The extent of bowel endometriotic nodules is variable; smaller nodules infiltrate only the intestinal serosa and the subserous fat tissue, whereas larger lesions may infiltrate the muscular layer of the bowel and reach the submucosa or the mucosa. The depth of infiltration of the endometriotic nodules in the bowel wall has been correlated with the severity of symptoms [2]. Small endometriotic nodules infiltrating only the serosal surface rarely cause symptoms [2]. Larger nodules may cause abdominal pain and a wide range of intestinal symptoms including dyschezia, diarrhea, constipation, abdominal bloating, painful bowel movements and

cyclical rectal bleeding [1–3]. It is well known that the surgical excision of bowel endometriosis determines an improvement in pain, intestinal symptoms and quality of life [4–7]. However, patients with bowel endometriosis may want to avoid or postpone surgery. Therefore, endocrine therapies (such as gonadotropin releasing hormone analogs, progestins and aromatase inhibitors) have recently been proposed for the treatment of pain and intestinal symptoms of patients with bowel endometriosis [8–10]. However, limited data are available on the natural history of bowel endometriotic nodules; in particular, there is no evidence that these lesions may progress during endocrine therapies [1]. In this report, we present the evidence of progression of bowel endometriosis during the use of endocrine therapy.

### Methods and findings

A 25-year-old woman was referred to our endometriosis center in 2005. She complained of dysmenorrhea and deep dyspareunia; she had no gastrointestinal symptom. She had never previously used hormonal treatments for endometriosis. The gynecological examination and transvaginal ultrasonography revealed the presence of a 2.5-cm rectovaginal nodule. The patient underwent multidetector computerized tomography enteroclysis (MDCT-e). The exam revealed the presence of a small sigmoid endometriotic nodule of the serosal surface that did not infiltrate the muscularis propria. The nodule was clearly visible during the exam as a retraction of the serosal surface of the sigmoid colon despite the optimal distension (Fig. 1). The patient refused the surgical excision of endometriosis and decided to use continuous oral contraceptive pill (desogestrel 150 µg and ethinylestradiol 20 µg; Mercilon, Rome, Italy). The patient was lost to follow-up

S. Ferrero (✉) · P. L. Venturini · V. Remorgida  
Department of Obstetrics and Gynecology,  
San Martino Hospital and University of Genoa,  
Largo R. Benzi 1,  
16132 Genoa, Italy  
e-mail: dr@simoneferrero.com

G. Camerini  
Department of Surgery,  
San Martino Hospital and University of Genoa,  
Largo R. Benzi 1,  
16132 Genoa, Italy

E. Biscaldi  
Department of Radiology, Galliera Hospital,  
Via Mura delle Cappuccine 14,  
16128 Genoa, Italy

**Fig. 1** Left, sagittal reconstruction of MDCT-e performed in 2005; a small endometriotic nodule (indicated by the red arrow) determines a retraction of the serosal surface of the sigmoid colon. Right, sagittal reconstruction of MR-e performed in 2009; the blue arrow indicates a large endometriotic nodule infiltrating the muscular layer of the sigmoid colon



until February 2009 when she had a consultation at our center because of pain and persistent gastrointestinal symptoms of increasing severity (constipation and dyschezia); she was still under continuous oral contraceptive pill. Magnetic resonance enteroclysis (MR-e) was performed. The exam revealed the presence of a large sigmoid nodule in the same site where, 41 months before, MDCT-e revealed the small serosal sigmoid nodule (Fig. 1). Hormonal therapy was continued until laparoscopy; the presence of the sigmoid endometriotic nodule was confirmed and it was excised by bowel resection. The histological examination of the bowel specimen demonstrated that the endometriotic nodule reached the bowel submucosa; the immunohistochemical staining with S100 showed that the infiltration reached the submucosal (Meissner) nervous plexus. The postoperative course was uneventful and the patient was discharged 5 days after surgery.

## Conclusion

Until now, there has been no evidence of progression of bowel endometriosis despite the use of endocrine therapies. This report shows, for the first time, that the oral contraceptive pill does not prevent the progression of bowel endometriosis. Nowadays, modern radiological techniques and transvaginal ultrasonography often allow a non-invasive diagnosis of rectosigmoid endometriosis [1, 11, 12]. These patients should be fully informed of the potential benefits and limitations of endocrine and surgical therapies. In particular, patients refusing surgery and using medical treatments may have an improvement in pain and intestinal symptoms [8–10]; however, they should be aware that a silent progression of bowel endometriotic nodules might occur despite endocrine treatments. Furthermore, these patients should be carefully monitored for potential progression of intestinal lesions. A limitation of this case report consists in the fact that the first diagnosis of sigmoid

endometriosis was based on MDCT-e and not on surgery and histology. However, previous studies demonstrated that MDCT-e has high sensibility and specificity for detecting intestinal endometriotic lesions and for determining the depth of infiltration of the nodules in the bowel wall [13–15]. A prospective study including 98 women showed that MDCT-e is reliable in identifying the depth of infiltration of serosal and muscular lesions, while an underestimation of the lesions reaching the submucosa may be observed [13]. In conclusion, to the best of our knowledge, this case report demonstrates for the first time that a progression of bowel endometriosis might occur despite hormonal therapies. This information should be given to all women choosing to avoid the surgical excision of colorectal endometriosis.

**Conflict of interest** The authors report no conflicts of interest. The authors alone are responsible for the content and writing of the paper.

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