

Hormonal treatments for adenomyosis

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Like endometriosis and uterine myomas, adenomyosis presents the typical characteristics of oestrogen-dependent diseases. The medical treatment of adenomyosis is based on the hormonal dependency of the disease and its strongly debated similarities with endometriosis. Infact, despite the evident differences between the two conditions, the therapies that treat endometriosis effectively have also been successful for the treatment of adenomyosis. Although the two diseases have distinct epidemiological features, they have the same 'target tissue' for hormonal therapy, namely ectopic endometrium. Recognized approaches are systemic hormonal treatments, which are generally used for endometriosis and are capable of suppressing the oestrogenic induction of the disease, and local hormonal treatment that targets the ectopic endometrium directly. Gonadotropin-releasing hormone agonists, danazol and intrauterine levonorgestrel- or danazol-releasing devices have been used in the treatment of adenomyosis. Despite the solid rational basis for its hormonal treatment, few studies have been performed on medical therapy for adenomyosis.

Key words: adenomyosis; medical therapy; levonorgestrel-IUD; pelvic pain; menorrhagia.

Adenomyosis is characterized by the presence of endometrial glands and stroma within the myometrium. The ectopic endometrium is typically surrounded by reactive, hypertrophic and hyperplastic myometrium.¹ The disease can range from small microscopical isolated endometrial islands within the myometrium to extensive infiltration of uterine walls, or grossly visible nodules without a capsule, allowing clear distinction

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from normal myometrium.² The former situation is more common and is referred to as 'diffuse adenomyosis', while the latter case describes nodular adenomyosis (also called 'adenomyomas') which is relatively rare. There are many uncertainties regarding the true incidence of the disease and the correlation with its commonly associated symptoms, namely metrorrhagia and pelvic pain.³ These doubts are due to the fact that it is impossible to confirm or exclude the presence of adenomyosis without prior adequate pathological assessment of the uterus, and there is no consensus regarding which histological diagnostic criteria should be applied for diagnosis of the disease.^{1,4-7} Study populations have invariably been constituted by women undergoing hysterectomy for menorrhagia and an enlarged uterus, especially in the fifth decade of life, so it should not be surprising that adenomyosis is commonly believed to be prevalent in women between 40 and 50 years of age. In fact, there are insufficient data, i.e. hysterectomies, regarding younger women. From the literature, it can be estimated that adenomyosis is present in approximately 20–30% of women who undergo hysterectomy³, and that, at least in the more severe forms of endometrial infiltration, there is a causal relationship between the disease, metrorrhagia and dysmenorrhoea.^{8,9} The relationship between adenomyosis, metrorrhagia and pelvic pain is less clear in the more limited forms, and the correlation between adenomyosis and infertility, which is strongly maintained by some authors^{10,11}, needs to be confirmed. With the exception of some nodular lesions, conservative surgical treatment is impracticable as it is not possible to isolate the adenomyotic tissue distinctly, so hysterectomy is the only rational and complete procedure. Recently, imaging studies for the diagnosis of adenomyosis by magnetic resonance imaging (MRI) and transvaginal ultrasound have shown these methods to be accurate and reliable.¹²⁻¹⁶ The definition of imaging diagnostic criteria for adenomyosis has allowed effective assessment of the medical treatments and of new conservative operative techniques, i.e. uterine artery embolization and MRI-focused ultrasound surgery.¹⁷

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RATIONAL BASIS OF HORMONAL MEDICAL THERAPY

Like endometriosis and uterine myomas, adenomyosis presents the typical characteristics of oestrogen-dependent diseases. In fact, it is absent before menarche and regresses rapidly after menopause. Studies on hormone–ligand binding in adenomyosis report that oestrogen receptors are always present, whereas progesterone receptors are found less frequently.¹⁸ Androgen receptors have also been encountered in adenomyotic tissue. The first studies in adenomyosis demonstrated that the levels of receptors for sex steroids were generally lower than those present in the endometrium, although this was disclaimed recently.¹⁹ Aromatase is an enzyme that converts androgens to oestrogens, i.e. $\Delta 4$ -androstenedione in oestrone, and can be found in adenomyotic tissue.^{20,21} The enzyme oestrone sulphatase is also present^{21,22}, and converts oestrone-3-sulphate, the most prevalent circulating oestrogen, into 17β -oestradiol. The combined activity of aromatase and oestrone sulphatase increases the local oestrogenic activity that, along with the circulating oestrogens, stimulates

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