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Endometriosis: A general review and rationale for surgical therapy

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Abstract

This review article aims to give a comprehensive insight into both the historical and current thoughts on all aspects of endometriosis including aetiology, diagnosis, and medical and surgical treatments. The prevalence of endometriosis is about 6–8%, and may affect up to two million women in the United Kingdom. It causes, through pain and infertility, a significant problem for sufferers, their families and society as a whole. There is no conclusive evidence to explain its aetiology although our understanding of the basic pathophysiology is improving. However, there remains a substantial lack of understanding in all areas of disease.

A rationale is presented for surgical therapy as the preferred approach for diagnosis and treatment. The recognised gold standard for diagnosis is laparoscopy. Surgery is the only treatment modality that consistently eradicates all macroscopic diseases and can be carried out at the same time as diagnosis. There is no evidence that medical treatment is superior to surgical treatment. Surgical removal of endometriotic lesions is the only treatment that improves spontaneous conception rates in endometriosis-associated infertility. The evidence for the surgical techniques and energy modalities used for the surgical management of minimal to moderate endometriosis, endometriomas and recto-vaginal disease are described in greater detail.

Keywords: Endometriosis; Laparoscopy; Pelvis

1. Introduction

Endometriosis is a common condition of disputed aetiology that can cause pain and infertility in women. It is defined as the presence of endometrial glands and stroma present outside the uterus [1] and is most commonly found in premenopausal women.

2. Prevalence

The prevalence of endometriosis has probably increased over the last 100 years or so for several reasons. Modern women have many more menses than their predecessors as they spend less of their reproductive lives in a state of pregnancy. Women now have an estimated 450 menses in their lifetime compared to the 100 that their Victorian forebears had. Consequently the frequency of dysmenorrhoea has increased. The introduction of laparoscopy over the last three decades has allowed us to diagnose endometriosis more easily as most gynaecologists are able to perform a diagnostic laparoscopy at least. The current prevalence of endometriosis is estimated to be up to 10% [2–4]. Vercellini stated in his address to the World Congress on Endometriosis in Maastricht in 2005 that the incidence has not increased in the last 30 years and remains at 2.37–2.49 per 1000 women per year, equating to an approximate prevalence of 6–8% [5,6].

3. Localisation and appearance

Endometriosis is most commonly found in the pelvis [7]. More rarely it is found in remote sites like the lungs and

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brain [8,9]. It has even been found in men [10]. In the pelvis, it is more frequently left sided [11]. The reason for this left sided dominance is unclear, although the direction of flow of peritoneal fluid been proposed as a possible cause [12].

Endometriotic lesions can infiltrate any structure in the pelvis. They are seen more commonly on the pelvic peritoneum, utero-sacral ligaments, bladder, sigmoid colon and rectum, the most severe presentation being the recto-vaginal nodule. On the ovaries, endometriotic cysts can form that contain a thick ''chocolate-like'' substance formed from old blood. These cysts are often adherent to the pelvic sidewall. Rarely, the ureter can be infiltrated with endometriosis itself, though stenosis is more commonly due to fibrotic impingement by a proximal utero-sacral nodule [13].

Endometriotic deposits can appear in a range of colours and textures from transparent vesicles and white fibrotic plaques to red haemorrhagic flares and blue/black nodular lesions. These may be superficial or described as deep if they extend more than 5 mm beneath the peritoneal surface. This wide variety of appearance is not always recognised by the general gynaecologist and the diagnosis may be easily missed. Overall appearance may vary from the barely visible through to the ''frozen pelvis''.

4. Epidemiology

There are several large epidemiological studies that provide an insight into the disease. The results reveal the depth of the problem for women, their families and society in general, as well as highlighting current inadequacies in the management of endometriosis.

The All Party Parliamentary Group in the UK has so far collected epidemiological data via its web-based questionnaire from 7025 women who reside in 52 different countries. It estimates that two million women in the UK suffer from endometriosis. The results of this survey currently show a delay in diagnosis in this cohort averaging 8.3 years with 65% of women reporting that they had initially received an incorrect diagnosis. Moreover, only a third of the sample believed their treatment to be effective. A startling 78% reported taking an average of 5.3 days off work per month and 72% reported relationship problems [5]. These results are similar to those found in a recent Australian genetic epidemiological study of 3895 women diagnosed with endometriosis where the average age of onset of endometriosis symptoms was 20.1 ± 6.8 years [14]. The youngest diagnosed participant in this study was 13 years old, although the disease, or its antecedent, has been diagnosed in females as young as 8 years old [15].

Endometriosis is reported to be less common in Black Africans and more common in East Asians compared with Caucasians, but it is found in all ethnic groups [16,17].

Endometriosis is found in 40–60% of women with pelvic pain and in 20–30% of women suffering from infertility [18–20]. Women with more advanced disease have a higher rate of infertility (19.5% for rASRM stage 1% versus 28.7% for rASRM stage 2–4) [21].

Risk factors for endometriosis include early age of menarche, short menstrual cycles, long duration of menstrual flow, a family history of endometriosis. There is also an inverse relationship with parity [4].

5. Aetiology

Many theories of the aetiology of endometriosis have been postulated since Rokitansky first described the disease in 1860 [22]. Meyer proposed the theory of coelomic metaplasia in 1909 and postulated that tissue, with the potential to develop into endometrial-like cells later in life, was laid down in the trans-embryonic coelom [23] and Halban suggested the possibility of haematological or lymphatic spread from the endometrium in 1924 [24]. Sampson proposed the theory of retrograde menstruation in 1927 [25] and this has become the ''default'' explanation.

However, there remain several problems with Sampson's theory. There is little doubt that reflux menstruation occurs. Bloody peritoneal fluid is found in 80–90% of menstruating women [26,27] compared with only 15% of women with occluded fallopian tubes. However, most women do not develop endometriosis. Moreover, the cells found in endometriosis are not identical to normal endometrium [28] and endometriosis does not generally recur if treated surgically as one might expect if retrograde menstruation was to continue [29,30]. Also, it does not explain the small, but nevertheless relevant, occurrence in men, premenarchal girls and postmenopausal women.

Consequently, all of the above theories remain in the frame to this day with no clear evidence having emerged for either one. Despite being the dominant theory, it seems likely that Sampson's view was over simplistic.

More recent advances in technology have permitted the emergence of new theories. Altered immune function has gained credibility with those seeking to find a basis for the discrepancy between the frequency of retrograde menstruation and the infrequency of endometriosis. This theory was first postulated in 1987 by Gleicher et al. [31], who suggested that immune system alterations result in a failure to ''mop up'' ectopic endometrial cells, and therefore allow them to infiltrate at the site of disease. Immune system alterations in endometriosis sufferers have been shown in natural killer (NK) and cytotoxic T cells, and aberrations have been found in immune mediators such as tumour necrosis factor-α, Interferon-γ and polyclonal B-cell autoantibodies [32].

Simpson et al. first suggested a genetic basis for endometriosis in 1980 [33]. This is likely to be complex and polygenic in nature. Linkage study work has now shown a susceptibility locus on Chromosome 10q26 [34]. Work is also progressing in other areas including studies of