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ANATOMICAL CHANGE OF THE UTERUS IN ENDOMETRIOSIS

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Abstract: This IMRAD-structured review addresses uterine anatomical and morphofunctional changes in endometriosis. We synthesize evidence on fibrotic–inflammatory remodeling of the myometrium and the junctional zone, coexistent adenomyosis, alterations in uterine position and mobility, neuroangiogenesis, and imaging features on transvaginal ultrasonography and MRI, alongside histopathology and laparoscopic mapping. Disruptions of uterine peristalsis, progesterone resistance, endometrial receptor changes, clinical implications (pain, infertility), and the need for multidisciplinary diagnostic and therapeutic strategies are discussed.

Keywords: endometriosis, adenomyosis, uterine anatomy, junctional zone, fibrosis, neuroangiogenesis, transvaginal ultrasound, MRI, fertility.

Introduction

Endometriosis is an estrogen-dependent, chronic inflammatory disease characterized by increased ectopic endometrial tissue and prolonged fibrosis-remodeling processes [1,2]. "Endometriosis is an estrogen-dependent inflammatory disease" [1]. The effect of this disease on the anatomy of the uterus is multifaceted: from physical appearance (enlargement of the uterus, globular shape) to its posisional state (retroversion/retroflexion and fixation), microstructure (thickening of the junctional zone, fibromuscular hyperplasia in the myometrium) and deep changes in function (peristalsis, preparation for implantation, receptor profile)occur [2-5]. Adenomyosis — a form of internal endometriosis-is often found in association with endometriosis and plays a central role precisely in the morphology of the uterus and the aggravation of its symptoms [3,5]. In the last decade, the possibilities of detecting structural changes of the localization zone (junctional zone) through transvaginal ultrasound examination (UTT) and magnetic resonance imaging (MRI) have expanded, and they are becoming important in clinical decision-making [6]. In this article, the main concepts, evidence and their clinical results about anatomical changes in the uterus in endometriosis are systematized in accordance with the requirements of IMRAD. Materials and methods (Narrative commentary). 2000-2024. o'. PubMed / Medline, scopus o'. Cochrane Library O'. o'. "Endometriosis", "adenomyosis", "uterine Anatomy", "attachment zone", "uterine peristalsis", "fibrosis", "neuroangiogenesis", "ultrasound", " MRI "o'. o'. Main selection criteria: 1) studies that provide morphological, pictorial (UTT, MRI) or histopathological information on uterine structure and function in endometriosis; 2) comprehensive reviews and consensus articles in the highest impact-factor journals; 3) work on anatomical distribution and surgical mapping in deep infiltrative endometriosis; 4) clinical-diagnostic resources that illuminate the joint course with adenomyosis. As a classic work of

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historical importance, a source for the Sampson (1927) hypothesis was also included [8]. For the purpose of quality control, the content of sources and their clinical significance were evaluated by the two authors; reasons were discussed for research with conflicting or differing conclusions (differences in design, sampling, pictorial methods). Based on the conditions that the number of links does not exceed 10, the article relied on 9 main sources elected [1-9]. The goal was to combine high clinical-coverage and robust reliability work while maintaining a wide range of evidence for discussion. Results Macroanatomic changes. Adenomyosis accompanied by endometriosis in most cases leads to globular enlargement of the uterus, deformation of the form and diffuse thickening of the myometrium. MRI depicts thickening of the localization Zone (>12 mm) and low-signal Strip-numbers, small cystic spots within the myometrium; while transvaginal Utt records asymmetric thickening, "fan-field" shading, myometrial cysts, acoustic artifacts due to fibrosis around the lesion [6]. In cases of deep infiltrative endometriosis (death), thickening and fibrosis of the uterine-sacral ligaments, rectovaginal septum and partial closure of the Douglas pit can cause retroversion/retroflexion and fixation by pulling the matka back [4]. It enhances general physical immobility and is vividly manifested by the phenotype of the "painful immobile uterus" in a gynecological examination. Microanatomic and histopathological changes. In endometriosis / adenomyosis, fibromuscular recurrence is noted in the myometrium, inflammatory infiltration and fibrosis in the respective areas, increased nerve fiber density (neuroangiogenesis) [3,5]. The zone of localization — the functional interface between the endometrium and the myometrium — becomes pathologically condensed and changes peristaltic transmittance. The hormonal microenvironment associated with relatively reduced progesterone receptors and local estrogen biosynthesis (aromatase activity) forms the phenotype of " progesterone resistance; this condition eliminates endometrial receptivity and implantation processes [2,5,7]. While such changes are clinically discriptive, their morphological substrate — inflammation-is closely related to recurrence within fibrosis. Functional results: peristalsis and fertility. Thickening of the localization zone and remodeling of the myometrium alters the patterns of uterine peristalsis: hyperperistalsis and discoordination can make sperm transport difficult, enhancing the mechanical conditions of retrograde bleeding [2,3]. There is evidence that the expression of HOXA-talents and integrins has been derailed in the implantable endometrium; this leads to a decrease in fertility and an increase in the number of cases in need of assistance [5,7]. In addition, fibrosis-adgesive processes associated with deep infiltrative foci alter the geometry of the wedge-uterine interface and form mechanical barriers [4]. Anatomical distribution and clinical phenotypes. In cases of death, it often localizes around the uterine-sacral ligaments, rectovaginal septum, Douglas pit, and parts of the () segmental Colon shown in planetary fashion [4] lesions. Such a distribution can lead to retardation of the uterus, the formation of fibrosis "grips" limiting its excitation, and acute dyspareunia. When an adenomyosis joint with endometriosis occurs, there is an "globular-asymmetric" appearance of the uterus, thickening of the zone of localization and increased peripheral sensitization of the pain signal through dense fibrosis [3,6]. Fine diagnostics. The Transvaginal UTT - first line method-identifies a number of signs characteristic of adenomyosis: asymmetric thickening of the myometrium, heterogeneous exostructure, subendometrial leaks, and small cysts. MRI specifically accurately assesses the junctional zone in T2-vzveshenny mode, increasing sensitivity with symptoms such as anomalous thickening and Phantom stripe-numbers [6]. Die Die Die, Die Die Die, [4,6]. In general, pictorial findings suggest a combination of clinical

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phenotype and morphological changes. Historical view and modern interpretation. 1927 "menstrual distribution of endometrial tissue in the abdominal cavity" RKK [8]. This hypothesis was an important historical milestone in explaining the origin of peritoneal furnaces. However, we think that it does not fully cover multifaceted processes such as hormonal-inflammatory relapses of uterine micromuxitis, neuroangiogenesis and fibrosis; modern views emphasize multifactorial pathogenesis — genetic predisposition, immune-metabolic factors and tissue-specific response integrations [2,3,5,7]. ("Menstrual distribution..."), noting that it is not "" but a fragment in an expanded pathogenetic context.closed theory along with recognition it is in this sense A Historical Quote by Sampson Debate The results presented indicate that changes in uterine anatomy in endometriosis occur simultaneously at morphological and functional levels. First, association with adenomyosis — thickening in the zone of localization and myometrium fibromuscular recurrence — affects the peristalsis and receptivity of the uterus. These changes are the central node affecting pain pathogenesis (neuroangiogenesis, sensitization) and fertility (sperm transport, embryonic implantation) [3,5,7]. The second, Associated extinct fibrosis-adgesive nodes increase anatomical fixation and deformity to form an immobile uterine phenotype on examination; this is consistent with the severity of clinical symptoms (dyspareunia, defecation pain, dysmenorrhea) [4]. Third, pictorial methods — especially MRI-have a high value in determining anatomical changes, integrating the depth and anatomical distribution of lesions into the surgical plan [6]. Thus, the connections between anatomy and function are multichannel: hormonal-inflammatory signals, Matrix recurrence, innervation and sosudation (neuroangiogenesis), mechanical fixation and peristalsis. Many authors have commented on the modern definition of endometriosis and areas of clinical practice. "Endometriosis is an estrogen-dependent inflammatory disease" [1]. In our opinion, although this definition is short and luminous, the inclusion of Matrix remodeling inside it, functional interface changes in the localization zone, local hormonogenesis and progesterone resistance as special signs makes the problem even more clear. That is, endometriosis is not just an "ectopic endometrium", but "a disease of uterine-external genitals with altered architectures, recurrence with fibrosis, prone to neuroangiogenesis"[2,3,5,7]. Practical points in diagnosis: 1) in a patient with dysmenorrhea and dyspareunia, the immobile uterus in the examination, pain in the sacrouterin ligaments and palpator thickening are grounds to suspect Death; 2) the presence of adenomyosis symptoms in the transvaginal Utt indicates the effect of endometriosis on the uterus; 3) MRI is of great importance in creating an anatomical map of death and assessing the Treatment requires surgery that, along with hormonal therapy (estrogen-suppression), pain control, and fertility strategies, avagens the dying-oriented, neuromuscular structures; in cases of Greater adenomyosis density, uterine cleavage (adenomyomestomy) and individual reproductive plans are evaluated [2,3,9]. Of course, this article is aimed at highlighting the essence of anatomical changes, and not treatment protocols; nevertheless, a deep understanding of anatomy increases the quality of therapeutic decisions.

Research limitations. In this review, sources rely on different designs and populations; the extent of deep infiltration and adenomyosis detection criteria differ in some works. The sensitivity/specificity of imaging methods depends on the hardware and operator. Prospective, multicenter, standardized protocol studies are required to show the strict cause-and-effect Binding of anatomical changes. At the same time, the general mass of evidence — the stability of MRT/UTT symptoms, the continuous presence of fibrosis-neuroangiogenesis in

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histopathological findings, and the coherence with clinical phenotypes — are reinforcing the practical value of the conclusions [2-6].

Conclusion

In endometriosis, anatomical changes in the uterus are the result of multifaceted and interconnected processes. Uterine positivity and excitability restriction with adenomyosis joint retraction, thickening of the joynization zone, fibromuscular reoccurrence in myometrium, and fibrosis, neuroangiogenesis, as well as Die-specific fibrosis-adgesive "grips", are the main morphological advantages. These changes affect fertility through uterine peristalsis and endometrial receptivity, shape the pain phenotype, and determine diagnostic and therapeutic strategies. While the historical hypothesis of "Menstrual dissemination" serves to explain anatomical distribution, the modern interpretation places polyphactor pathogenesis-a combination of hormonal — inflammatory, immune and tissue recurrence mechanisms-at the center. Imaging techniques, particularly MRI, play a leading role in mapping these changes; while histopathology supports fibrosis-neurovascular reversibility. The multidisciplinary approach — the collaboration of a gynecologist, radiologist, reproductologist and multidisciplinary surgeons-provides an individual plan corresponding to the volume of anatomical changes and clinical goals. While further research focuses on linking peristalsis, receptivity, and fibrosis biomarkers with clinical outcomes based on standardized criteria, targeted conservation or regeneration strategies for uterine anatomy in endometriosis are further refined.

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