MORPHOLOGICAL REMODELING OF THE ENDOMETRIUM AND MYOMETRIUM IN ADENOMYOSIS

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Background. Adenomyosis is a benign but clinically significant gynecological disorder characterized by the presence and progressive invasion of endometrial glands and stroma into the myometrium. The disease is associated with dysmenorrhea, chronic pelvic pain, abnormal uterine bleeding, infertility, and reduced quality of life. Although the pathogenesis of adenomyosis remains incompletely understood, increasing evidence indicates that the disease is accompanied by profound structural and functional remodeling of both the endometrium and myometrium. These changes are not limited to the ectopic endometrial foci but involve the surrounding junctional zone and the eutopic endometrium, suggesting a complex interaction between epithelial, stromal, smooth muscle, vascular, and inflammatory components.

Aim. To characterize the principal morphological changes and tissue remodeling processes occurring in the endometrium and myometrium in adenomyosis and to assess their potential contribution to disease progression and clinical manifestations.

Materials and Methods. A morphological analysis of uterine tissue in adenomyosis was considered with emphasis on routine histological examination and morphometric assessment. Tissue sections were evaluated for the structural organization of the endometrial glands and stroma, epithelial changes, stromal cellularity, myometrial architecture, smooth muscle hypertrophy and hyperplasia, fibrosis, vascular remodeling, and the distribution of ectopic endometrial structures within the myometrium. Particular attention was paid to the junctional zone, which

represents a critical anatomical and functional interface between the endometrium and myometrium. The morphological findings were interpreted in relation to the mechanisms of tissue invasion, inflammation, fibrosis, and abnormal uterine contractility.

Results. Adenomyosis was associated with marked remodeling of the normal endometrial–myometrial architecture. The endometrium demonstrated heterogeneous structural changes, including irregularity of glandular architecture, alterations in gland-to-stroma relationships, variable epithelial proliferation, stromal remodeling, and focal inflammatory infiltration. In some areas, endometrial glands and stroma exhibited morphological features extending beyond the expected endometrial–myometrial boundary, indicating disruption of the normal tissue interface.

The most characteristic morphological feature was the presence of endometrial glands and endometrial-type stroma within the myometrial tissue. These ectopic structures were frequently accompanied by reactive changes in the surrounding smooth muscle. The myometrium demonstrated focal or diffuse hypertrophy and hyperplasia of smooth muscle cells, resulting in increased muscular density and distortion of the normal fascicular arrangement. In areas surrounding adenomyotic foci, myometrial fibers frequently showed disorganization and variable degrees of hypertrophy.

Another important component of tissue remodeling was the development of fibrosis. Increased deposition of collagen and expansion of fibrous connective tissue were observed around adenomyotic lesions and between smooth muscle bundles. Fibrotic remodeling contributed to increased tissue stiffness and architectural distortion of the myometrium. In addition, vascular changes, including increased vascular density

and structural alterations of small vessels, were consistent with active tissue remodeling and chronic inflammatory stimulation.

The junctional zone appeared to represent a particularly important site of pathological remodeling. Disruption of its normal architecture was associated with increased smooth muscle activity, structural disorganization, and penetration of endometrial components into the underlying myometrium. These changes may contribute to abnormal uterine peristalsis and impaired coordination between endometrial and myometrial functions.

The morphological alterations were accompanied by features consistent with chronic inflammatory activation. Stromal and myometrial cellular responses, together with fibrosis and vascular remodeling, indicate that adenomyosis should be regarded not simply as ectopic localization of endometrial tissue but as a complex process of tissue injury, repair, inflammation, and pathological remodeling.

Conclusion. Adenomyosis is characterized by profound remodeling of the endometrium–myometrium interface involving ectopic endometrial invasion, smooth muscle hypertrophy and hyperplasia, disruption of myometrial architecture, fibrosis, vascular remodeling, and chronic inflammatory changes. The junctional zone appears to play a central role in the development and progression of these structural abnormalities. The interaction between ectopic endometrial tissue and the surrounding myometrium may establish a self-perpetuating cycle of inflammation, tissue injury, fibrosis, and abnormal smooth muscle remodeling. Consequently, morphological assessment of the endometrium, junctional zone, and myometrium provides important information for understanding the pathogenesis of adenomyosis and may contribute to

the identification of potential morphological and molecular targets for diagnosis and treatment.

Keywords: adenomyosis; endometrium; myometrium; junctional zone; morphological remodeling; fibrosis; smooth muscle hypertrophy; ectopic endometrium; inflammation; uterine tissue.