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MORPHOLOGICAL CHANGES OF THE UTERUS DURING MENOPAUSE

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Abstract

Menopause is a physiological stage of female aging associated with permanent cessation of ovarian activity and progressive estrogen deficiency, resulting in significant structural and functional changes in reproductive organs. The uterus is particularly sensitive to hormonal alterations because of its dependence on cyclic estrogen and progesterone stimulation. Menopausal hypoestrogenism induces endometrial atrophy, myometrial fibrosis, vascular insufficiency, and connective tissue remodeling, leading to progressive uterine involution.

This literature review analyzes current evidence regarding morphological and histopathological changes of the uterus during menopause. Methods included systematic searches of international databases for studies published between 2000 and 2025 addressing uterine aging, menopausal histology, endometrial atrophy, myometrial remodeling, and vascular changes. Results demonstrate that estrogen deficiency reduces proliferative activity of the endometrium, decreases vascularization, promotes stromal fibrosis, and alters smooth muscle architecture. Degenerative cellular processes and extracellular matrix remodeling contribute to reduced uterine elasticity and adaptive capacity.

The review highlights the importance of understanding normal menopausal uterine morphology for differential diagnosis of pathological conditions, optimization of gynecological care, and development of preventive strategies aimed at preserving women's reproductive health during aging.

Keywords: menopause, uterus, endometrium, myometrium, uterine atrophy, histopathology, estrogen deficiency, reproductive aging, fibrosis, vascular remodeling

Introduction

Menopause is characterized by the permanent cessation of menstruation resulting from ovarian follicular depletion and reduced production of estrogen and progesterone. This physiological transition causes profound endocrine and metabolic alterations affecting multiple organ systems, particularly the female reproductive tract. The uterus demonstrates marked sensitivity to hormonal fluctuations because its structure and function are largely regulated by ovarian steroids.

During reproductive life, estrogens stimulate endometrial proliferation, angiogenesis, glandular secretion, and myometrial trophism. Progesterone modulates cyclic differentiation and maintains endometrial stability. Menopause disrupts these regulatory mechanisms, resulting in progressive uterine involution and tissue remodeling.

Morphological changes of the uterus during menopause include reduction in uterine size, endometrial thinning, stromal fibrosis, degeneration of smooth muscle fibers, and vascular sclerosis.

These changes not only reflect physiological aging but also influence susceptibility to gynecological disorders including endometrial hyperplasia, postmenopausal bleeding, adenomyosis, and malignant transformation.

This review analyzes the role of hormonal deficiency in uterine remodeling during menopause and summarizes major histopathological changes observed in uterine tissues.

Methods

This review was conducted through systematic searches of databases including PubMed, Google Scholar, Scopus, ResearchGate, and international gynecological journals for studies published from 2000 to 2025. Search terms included "menopause uterine morphology," "endometrial atrophy," "myometrial remodeling during menopause," "uterine aging," and "histopathological changes in postmenopausal uterus."

Inclusion criteria involved peer-reviewed English-language studies addressing histological, structural, vascular, and cellular changes in the uterus during menopause. Articles focused exclusively on malignant uterine pathology unrelated to menopausal physiology were excluded.

Approximately 30–40 scientific sources were screened, with 15–20 studies selected according to relevance and scientific quality. Data synthesis focused on hormonal mechanisms, tissue remodeling, vascular alterations, and clinical implications of uterine aging.

Results

Endometrial Atrophy During Menopause

The most characteristic morphological feature of the menopausal uterus is endometrial atrophy. Reduced estrogen stimulation decreases glandular proliferation and suppresses mitotic activity within the endometrial epithelium.

Histologically, the postmenopausal endometrium demonstrates:

- thinning of the functional layer;
- reduced glandular density;
- cystic dilation of residual glands;
- stromal fibrosis;
- diminished vascularization.

Endometrial glands become narrow and inactive, while stromal tissue gradually loses cellularity and elasticity. These changes contribute to decreased regenerative potential and increased tissue fragility.

Myometrial Remodeling

The myometrium undergoes progressive structural degeneration during menopause. Estrogen deficiency reduces trophic support for smooth muscle cells, leading to partial replacement of muscular tissue by fibrous connective tissue.

Microscopic examination reveals:

- atrophy of smooth muscle fibers;
- collagen accumulation;
- reduced vascular perfusion;
- decreased elasticity of the uterine wall.

Fibrotic remodeling contributes to reduction in uterine volume and impaired contractile function. Age-related connective tissue deposition additionally affects pelvic support structures and uterine mobility.

Vascular Alterations

Menopause is associated with significant vascular remodeling within uterine tissues. Estrogen normally exerts vasodilatory and angiogenic effects; therefore, hormonal decline contributes to progressive vascular insufficiency.

Observed vascular changes include:

- sclerosis of small arteries;
- narrowing of vascular lumens;
- decreased capillary density;
- impaired microcirculation.

Reduced blood supply promotes tissue hypoxia, degenerative processes, and impaired cellular repair mechanisms.

Connective Tissue Remodeling

Extracellular matrix metabolism changes considerably during menopause. Imbalance between collagen synthesis and degradation leads to excessive accumulation of connective tissue components.

Fibrotic changes are particularly pronounced in the endometrial stroma and myometrial interstitium. Increased collagen deposition reduces tissue flexibility and contributes to uterine rigidity.

Cellular Degeneration and Aging

Cellular senescence is an important component of menopausal uterine involution. Aging uterine cells demonstrate reduced proliferative activity, mitochondrial dysfunction, and increased oxidative stress.

Accumulation of reactive oxygen species damages intracellular structures and accelerates tissue degeneration. Impaired DNA repair mechanisms further contribute to progressive structural deterioration.

Discussion

The available evidence demonstrates that menopause induces complex morphological remodeling of uterine tissues primarily through estrogen deficiency and age-related degenerative mechanisms. Similar to dyshormonal changes described in hormonally sensitive organs such as the mammary gland, uterine tissues exhibit marked structural dependence on endocrine regulation.

Endometrial atrophy represents the dominant morphological process during menopause. Reduced hormonal stimulation suppresses proliferative activity and vascularization, producing a thin and fragile endometrial lining. Although physiological in most cases, excessive atrophy may contribute to postmenopausal bleeding because of superficial vessel fragility.

Myometrial fibrosis and vascular sclerosis further reduce uterine adaptive capacity. Connective tissue accumulation and impaired perfusion decrease tissue elasticity and regenerative potential. These alterations may predispose women to chronic pelvic disorders and delayed tissue repair.

The relationship between vascular insufficiency, oxidative stress, and extracellular matrix remodeling highlights the multifactorial nature of uterine aging. Similar pathogenic mechanisms have been observed in other estrogen-dependent tissues undergoing menopausal involution.

Clinically, understanding normal menopausal morphology is essential for differentiation between physiological aging and pathological conditions including hyperplasia, polyps, adenomyosis, and endometrial carcinoma. Regular gynecological monitoring and histopathological evaluation remain important components of postmenopausal healthcare.

Conclusion

In summary, menopause induces profound morphological and histopathological changes in the uterus resulting from progressive estrogen deficiency and tissue aging. Endometrial atrophy, myometrial fibrosis, vascular insufficiency, connective tissue remodeling, and cellular degeneration represent the principal structural alterations observed during the menopausal period.

These changes significantly influence uterine physiology, regenerative capacity, and susceptibility to gynecological pathology. Understanding mechanisms of uterine involution is essential for accurate diagnosis, optimization of gynecological management, and development of preventive strategies aimed at preserving women's reproductive health during aging.

Future studies should focus on molecular biomarkers of uterine aging, mechanisms of fibrosis progression, and therapeutic interventions capable of reducing degenerative changes in menopausal uterine tissues.

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