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Research Progress on Risk Factors for Endometrial Lesions in Asymptomatic Postmenopausal Women with Endometrial Thickening

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Abstract: As of 2023, endometrial cancer (EC) ranks second among malignant tumors of the female reproductive system in China, following cervical cancer, posing a significant burden on the country's healthcare system. Postmenopausal asymptomatic endometrial thickening is primarily benign, often involving endometrial polyps. However, previous clinical studies indicate a relatively high malignancy rate for postmenopausal endometrial polyps, suggesting the necessity for active intervention, particularly in cases with high-risk factors for EC. This article reviews the research progress on risk factors for endometrial lesions in postmenopausal patients with asymptomatic endometrial thickening, aiming to provide insights for clinical diagnosis and treatment.

Keywords: Postmenopause; Asymptomatic endometrial thickening; Endometrial lesions; Risk factors

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1. Introduction

The incidence of endometrial cancer (EC) in China is on the rise. According to data released by the National Cancer Center in 2023, there were 71,100 new cases of uterine cancer, making it the second most common malignancy in the female reproductive system, following cervical cancer [1]. After menopause, ovarian function gradually declines, and estrogen levels decrease significantly, typically resulting in the thinning of the endometrium. However, in some women, the endometrium may abnormally thicken, which, if left untreated, could

increase the risk of developing EC. The exact mechanisms of malignant transformation in endometrial lesions remain unclear but are influenced by multiple factors, including obesity, hormone replacement therapy during menopause, and hypertension. Studies have shown that a body mass index (BMI) exceeding 23 kg/m^2, the presence of hypertension or diabetes, more than three pregnancies, more than two deliveries, and menopausal status are independent risk factors for malignant transformation in patients with endometrial lesions [2]. In clinical practice, the management of postmenopausal patients with asymptomatic endometrial thickening requires a multi-layered screening approach and individualized treatment strategies to mitigate the risk of malignant transformation. This study aims to review the primary risk factors and potential mechanisms of endometrial lesions in postmenopausal patients with asymptomatic endometrial thickening, explore the relationship between metabolic diseases such as obesity and hypertension and endometrial lesions, and summarize related screening methods and treatment strategies to provide scientific evidence for clinical management.

2. Causes of asymptomatic endometrial thickening after menopause

The formation of asymptomatic endometrial thickening in postmenopausal women involves a variety of physiological and pathological factors, including changes in the endocrine environment and localized tissue hyperplasia. Normal menstrual cycles rely on stable regulation by the hypothalamic-pituitary-gonadal axis. However, in postmenopausal women, the declining function of this axis and fluctuating hormone levels lead to an inability of the endometrium to transition normally from the proliferative to the secretory phase. This results in excessive endometrial growth, breakthrough or withdrawal bleeding, and potentially endometrial lesions. Research has shown that 84.3% of patients with asymptomatic postmenopausal endometrial thickening have intrauterine lesions, and the detection rate of malignancy increases with endometrial thickness [3].

Endometrial polyps are among the common causes of asymptomatic endometrial thickening in postmenopausal women. Polyps form from localized hyperplasia of endometrial glands and stroma. In postmenopausal women, due to the progressive atrophy of the uterine cavity, polyps are often flattened and adhere closely to the endometrial surface, presenting as uniform or irregular thickening on ultrasound. These lesions are frequently asymptomatic, and even when detected during routine examinations, distinguishing between benign polyps and potential malignancies via imaging is challenging, complicating clinical management [4]. Submucosal fibroids, which grow into or protrude into the uterine cavity, are another critical factor contributing to endometrial thickening post-menopause. Although estrogen levels decrease after menopause, existing submucosal fibroids may undergo cystic degeneration or softening during hormonal regression, protruding into the uterine cavity and appearing as thickened endometrium on ultrasound. Infections and chronic inflammation also contribute to asymptomatic endometrial thickening. Declining estrogen levels weaken the natural barrier function of the vagina and uterine cavity, making local tissues more susceptible to pathogenic invasion. The resulting inflammatory responses may cause endometrial congestion and edema, manifesting as thickened endometrium or intrauterine fluid accumulation on imaging. Additionally, postmenopausal women often experience inflammation-related endocrine and immune dysfunction, disrupting the normal balance of the endometrium and promoting cellular hyperplasia [5].

3. Screening methods for asymptomatic endometrial thickening in postmenopausal women

Transvaginal ultrasonography (TVUS) is the most commonly used initial screening tool, valued for its non-

invasive, convenient, and efficient characteristics. TVUS effectively visualizes endometrial thickness and uterine cavity structures. For instance, a study by Fu *et al.* [6] demonstrated that TVUS has high accuracy in diagnosing endometrial lesions. By analyzing hemodynamic parameters such as resistance index (RI), pulsatility index (PI), total blood flow area (TAP), and imaging features, TVUS can differentiate between polyps and malignancies. These lesions show distinct hemodynamic profiles: malignancies typically present with greater endometrial thickness and clearer endometrial-myometrial junctions but lower rates of homogenous internal echoes compared to polyps. However, TVUS has limitations when screening asymptomatic postmenopausal women. Studies suggest that while the negative predictive value of endometrial thickness is high, the specificity for absolute lesion risk is insufficient, leading to false positives. The positive predictive value ranges from 0–20%, while the negative predictive value is as high as 98–100% [7]. Saline infusion sonohysterography (SIS) enhances diagnostic accuracy by injecting saline into the uterine cavity for ultrasound imaging, allowing for clearer visualization of uterine lesions and reduced false positives. For example, research by Palermo *et al.* [8] found SIS to have a specificity of 84.47% and a sensitivity of 100% in diagnosing asymptomatic postmenopausal endometrial thickening. Hysteroscopy (HS) and endometrial histopathological examination remain the gold standards for diagnosing endometrial thickening. These methods allow direct visualization of intrauterine lesions and precise sampling for pathological analysis [9]. However, HS is invasive and may pose risks such as cervical dilation difficulties, uterine perforation, or infection. Thus, it is recommended for patients with endometrial thickness exceeding critical values and significant high-risk factors.

4. Endometrial sampling techniques for screening endometrial lesions

Endometrial cytology testing (ECT) uses an endometrial sampling device to collect exfoliated cells, which are then analyzed using thin-layer liquid-based cytology (LBC) technology. This non-invasive method is effective for screening endometrial lesions. LBC technology improves the sensitivity and specificity of screenings by removing blood and mucus and enhancing the uniformity and quality of cell samples. Common endometrial sampling devices include Tao Brush, Li-Brush, and SAP-1, which mechanically collect exfoliated cells from the uterine cavity. Cytological smear technology clearly presents pathological features of nuclei and cytoplasm, aiding in the diagnosis of lesions. Research indicates that ECT achieves sensitivity rates of 75–96% and specificity rates of 83–100% for diagnosing endometrial cancer and atypical hyperplasia, demonstrating significant diagnostic value. Wen *et al.* [10] conducted a study using ECT combined with the SAP-1 endometrial sampler on 1,045 postmenopausal women. The results showed an accuracy rate of 93.4%, a sensitivity of 72.4%, a specificity of 99.1%, a positive predictive value of 95.5%, and a negative predictive value of 93.0% for diagnosing endometrial cancer and atypical hyperplasia. Similarly, research by Wu [11] demonstrated that liquid-based cytology testing of the endometrium is highly reliable for diagnosing endometrial lesions in postmenopausal women. This method is simple, minimally invasive, and associated with minimal bleeding, offering significant advantages.

5. Risk factors for endometrial lesions in postmenopausal women with asymptomatic endometrial thickening

5.1. Obesity and postmenopausal hormone therapy

Obesity is a significant risk factor for asymptomatic endometrial thickening in postmenopausal women, closely associated with endocrine disorders, metabolic imbalances, and chronic inflammation. Obesity affects female

fertility through metabolic disturbances, such as hyperandrogenism, insulin resistance-related hyperandrogenemia, and high leptin levels. After menopause, when ovarian function declines, adipose tissue becomes the primary source of estrogen. Aromatase, highly expressed in adipose tissue, converts androstenedione to estrone, which is subsequently converted to estradiol. This continuous estrogen stimulation, without progesterone counteraction, leads to prolonged endometrial hyperplasia, increasing the risk of lesions and potential malignancy [12]. For instance, research by Wang *et al.* [13] found that in patients with postmenopausal asymptomatic endometrial thickening of ≥ 5 mm, the incidence of endometrial lesions was significantly higher in obese individuals compared to those with normal weight. Chronic low-grade inflammation is another hallmark of obesity, with adipose tissue secreting large amounts of pro-inflammatory factors such as tumor necrosis factor-α and interleukin-6. These factors exacerbate systemic metabolic imbalances, promote abnormal cell proliferation, and increase the risk of endometrial cancer [14]. Menopausal hormone therapy, while alleviating menopausal symptoms, also increases the risk of endometrial thickening by elevating estrogen levels, especially in the absence of progesterone counteraction. For example, Zheng's [15] research found that combining triptorelin acetate with hormone replacement therapy to treat infertility in patients with polycystic ovary syndrome resulted in increased endometrial thickness, raising the risk of endometrial lesions.

5.2. Hypertension

Hypertension influences the development of endometrial lesions through multiple physiological pathways. It causes structural damage to vascular walls and endothelial dysfunction, increasing endometrial inflammation and angiogenesis, thereby creating favorable conditions for lesion development. Prolonged hypertension also stimulates the hypothalamic-pituitary-ovarian axis, disrupting normal function and causing fluctuations in estrone levels, which may trigger endometrial-related lesions [16]. For instance, Long's [17] research indicated that hypertension significantly increases the risk of endometrial atypical hyperplasia and endometrial cancer, highlighting it as a critical risk factor for endometrial lesions in postmenopausal asymptomatic endometrial thickening.

6. Conclusion

This article emphasizes the role of risk factors such as obesity, menopausal hormone therapy, and hypertension in promoting endometrial thickening and lesions, exploring their underlying mechanisms. Addressing these risk factors requires strengthening individualized screening strategies in clinical management. High-risk populations should be monitored early using transvaginal ultrasound, endometrial sampling, and hysteroscopy. If endometrial thickening exceeds clinical thresholds or imaging suggests potential lesions, prompt intervention is essential to reduce the incidence of malignant transformation, ultimately improving patient prognosis and quality of life.

Disclosure statement

The authors declare no conflict of interest.

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