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Evaluating Transvaginal Ultrasound for Monitoring Treatment Response in Endometrial Hyperplasia: Clinical Utility of a 5-mm Endometrial Thickness Cutoff

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Abstract

Objectives:

To evaluate the diagnostic performance of transvaginal ultrasonographic endometrial thickness (ET) in predicting histopathological response to progestin therapy in patients with medically treated endometrial hyperplasia.

Methods:

In this prospective observational study, patients with histologically confirmed endometrial hyperplasia receiving progestin therapy underwent transvaginal ultrasonography and repeat histopathologic evaluation at 3 and 6 months. Endometrial thickness was categorized using predefined cut-off values (≤ 5 mm, 5.5–10 mm, 10.5–15 mm, and > 15 mm). Receiver operating characteristic (ROC) analysis was performed to assess the discriminatory performance of ET in predicting histopathologic outcomes. The influence of baseline histopathology and the presence of endometrial polyps was also evaluated

Results:

Across both follow-up intervals, histopathological regression was observed in all ET categories. ROC analysis demonstrated poor overall discriminatory ability of ET (AUC = 0.369 at 3 months; 0.424 at 6 months). Although an ET ≤ 5 mm was associated with higher regression rates, no single cut-off value reliably predicted treatment response. The presence of endometrial polyps was associated with increased ET measurements even among patients showing histopathologic regression.

Conclusion:

Transvaginal ultrasonographic measurement of endometrial thickness has limited ability to predict histopathologic regression after progestin therapy and should not substitute for tissue diagnosis. However, a threshold of ≤ 5 mm may serve as a supportive indicator of favorable therapeutic response in selected patients.

Introduction

Endometrial hyperplasia represents a spectrum of proliferative endometrial disorders characterized by glandular crowding and an increased risk of progression to endometrial carcinoma, particularly in the presence of cytologic atypia (1). Histopathological evaluation remains the gold standard for diagnosis, classification, and assessment of treatment response, as emphasized by international guidelines (1, 2).

Progestin therapy is widely used as a conservative treatment option for endometrial hyperplasia, especially in women desiring fertility preservation or those who are poor surgical candidates. Previous studies have demonstrated higher regression rates in non-atypical hyperplasia, whereas atypical hyperplasia is associated with a lower response rate and a higher risk of persistence or progression (3–6). Consequently, close follow-up is essential to evaluate treatment efficacy and guide further management.

Transvaginal ultrasonography is commonly used as a noninvasive tool for assessment and follow-up of endometrial pathology. Endometrial thickness (ET) is a readily available ultrasonographic parameter and is frequently incorporated into clinical decision-making (7–9). Several cutoff values, most commonly around 4–5 mm, have been proposed to stratify risk; however, the role of ET in predicting histopathological response after medical treatment remains controversial and less investigated (10–12) .

The present study aimed to evaluate the diagnostic performance of transvaginal ultrasonographic ET in predicting histopathological response to progestin therapy at 3- and 6-month follow-up intervals. Despite the widespread use of transvaginal ultrasound in endometrial assessment, the role of endometrial thickness as a follow-up marker after progestin therapy has not been sufficiently clarified. Therefore, this study seeks to address this clinical gap by correlating sonographic findings with histopathologic outcomes at defined follow-up intervals.

Methods

Study design and participants

This prospective observational cohort study was conducted at Shahid Sadoughi Hospital, affiliated with Shahid Sadoughi University of Medical Sciences. The study protocol was approved by the Institutional Ethics Committee of Shahid Sadoughi University of Medical Sciences (IR.SSU.MEDICINE.REC.1403.216)and was conducted in accordance with the principles of the Declaration of Helsinki.Because all diagnostic and therapeutic procedures were part of standard clinical care and patient data were analyzed anonymously, the need for written study-specific informed consent was waived by the institutional ethics committee.

Participants were recruited and followed prospectively between March 2021 and December 2024. A total of 75 women with histologically confirmed endometrial hyperplasia were included, representing all consecutive patients who met the inclusion criteria during the study period. Eligible participants had undergone baseline transvaginal ultrasonography and endometrial sampling by dilation and curettage or Pipelle biopsy because of increased endometrial thickness. All transvaginal ultrasonographic examinations were performed by a single experienced gynecologist using a standardized examination protocol to minimize interobserver variability.

Treatment and follow-up protocol

All participants received continuous medical treatment with megestrol acetate at a dose of 40 mg daily for a prespecified treatment duration of 3 or 6 months, according to the clinical treatment protocol. Follow-up evaluation was scheduled immediately after completion and discontinuation of the assigned treatment course.

At the end of the 3- or 6-month treatment interval, megestrol acetate was discontinued, and transvaginal ultrason6-month treatment interval, megestrol acetate was discontinued, and transvaginal ultrason treatment discontinuation. Thus, ultrasonographic assessment and histopathologic evaluation were obtained within a predefined and short interval after completion of progestin therapy.

Because follow-up assessments were scheduled according to the predefined treatment duration and immediately after discontinuation of continuous progestin therapy, rather than according to spontaneous menstrual-cycle phases, menstrual-cycle timing was not used to determine the timing of ultrasonography or endometrial sampling.

Eligibility and exclusion criteria

Patients were excluded if they had previously received tamoxifen, aromatase inhibitors, or hormone replacement therapy; lacked baseline or follow-up ultrasonographic or histopathologic data; or had incomplete study data that precluded assessment of treatment response.

Transvaginal ultrasonographic assessment

Endometrial thickness was measured by transvaginal ultrasonography as the maximum double-layer thickness of the endometrial echo complex in the sagittal plane. Endometrial thickness was recorded in millimeters. The presence of endometrial polyps was also documented during ultrasonographic assessment and was considered a potential confounding factor.

For the primary analysis, endometrial thickness was dichotomized using a 5-mm cutoff into two groups: **≤5 mm** and **> 5 mm**. For secondary descriptive analyses, endometrial thickness was categorized into four predefined groups: **≤5 mm, > 5 to 10 mm, > 10 to 15 mm, and > 15 mm**. The ET categories (< 5, 5–10, 10–15, and > 15 mm) were chosen based on previously reported clinical thresholds in the literature regarding endometrial hyperplasia management.

Outcome definition

The primary outcome was histopathologic regression of endometrial hyperplasia on repeat endometrial sampling performed after completion and discontinuation of megestrol acetate therapy. Treatment response was defined according to the histopathologic findings obtained at follow-up sampling.

Statistical analysis

Statistical analyses were performed using IBM SPSS Statistics software (version 18.0; SPSS Inc., Chicago, IL, USA). Continuous variables are presented as mean ±\pm± standard deviation (SD) or median with interquartile range (IQR), as appropriate. Normality of quantitative data distribution was assessed using the Kolmogorov–Smirnov test. Categorical variables are reported as frequencies and percentages.For univariate comparisons, the Chi-square test was used to analyze categorical variables, while Fisher's exact test was employed whenever the expected frequency in more than 20% of the contingency table cells was less than 5.

Receiver operating characteristic (ROC) curve analysis was performed to determine the optimal cutoff value and evaluate the overall diagnostic performance of endometrial thickness in predicting histopathologic regression. The area under the ROC curve (AUC), sensitivity, specificity, positive predictive value (PPV), negative predictive value (NPV), and corresponding 95% confidence intervals (CIs) were calculated. A two-sided P-value < 0.05P\text{-value} < 0.05P-value < 0.05 was considered statistically significant for all analyses.

Use of generative AI

During the preparation of this work, the author(s) used ChatGPT (generative AI) for language editing and formatting assistance. The authors reviewed and edited the output and take full responsibility for the content.

Results

1. Base line characteristics

Seventy-five women with biopsy-proven endometrial hyperplasia, aged 27 to 65 years (mean ± SD: 38.80 ± 7.37), were evaluated. The baseline histopathologic distribution showed that simple hyperplasia without atypia was the most common diagnosis (n = 37, 49.3%), followed by complex hyperplasia without atypia (n = 16, 21.3%). Simple atypical hyperplasia and complex atypical hyperplasia each accounted for 12% of cases (n = 9 for each). The least frequent finding (n = 4, 5.3%) was in disordered proliferative endometrium group (Table 1).

2. Diagnostic performance of TVUS in relation to post-treatment pathology

Transvaginal ultrasonography demonstrated limited diagnostic performance in predicting histopathological outcomes. Receiver operating characteristic (ROC) analysis showed poor discriminatory ability, with an area under the curve (AUC) of 0.369 (95% CI, 0.228–0.510; P = 0.103) at 3 months and 0.424 (95% CI, 0.129–0.719; P = 0.512) at 6 months. Overall, transvaginal ultrasonography yielded a sensitivity of 71% and a specificity of 5.5%, with a positive predictive value of 23% and a negative predictive value of 33.3%.Although a statistically significant association was observed between

sonographic findings and histopathological results (Chi-square P = 0.012), this finding did not translate into clinical utility, as evidenced by the poor agreement (Cohen's kappa coefficient κ = −0.139; P = 0.006) and limited predictive performance (Table 2).

3. Three-month outcomes

At the three-month follow-up, histopathologic evaluation demonstrated regression of endometrial hyperplasia in 58 patients (77.3%), while 17 patients (22.7%) showed persistent disease. Endometrial thickness measured by transvaginal ultrasonography was categorized as ≤ 5 mm, 5.5–10 mm, 10.5-15mm and > 15mm. Comparison between endometrial thickness categories and histopathologic findings showed **no statistically significant association** (P = 0.422). Receiver operating characteristic analysis for predicting persistent disease yielded an AUC of 0.369 (P = 0.103), indicating poor discriminatory performance (Table 3).

Table 3
Cross-tabulation of Three-Month Histopathology and Endometrial Thickness Measured by TVUS

4. Six-month outcomes

At the 6-month follow-up, 68 patients were available for analysis, while 7 patients were lost to follow-up. Regression of endometrial hyperplasia was observed in 61 patients (89.7%), whereas 7 patients (10.3%) had persistent disease. Endometrial thickness was again classified into four categories (≤ 5 mm, 5.5–10 mm, 10.5–15 mm and < 15mm). Similar to the three-month assessment, **no statistically significant association** was found between endometrial thickness category and histopathologic outcome (P = 0.353) (Table 4).

Table 4
Cross-tabulation of Six-Month Histopathology and Endometrial Thickness

Six-months Pathology	**Endometrial Thickness by TVUS (Month 6)**				
	≤ 5 mm	**5.1–10 mm**	**10.1–15 mm**	**> 15 mm**	**Total**
Persistent disease	0 (0.0)	5(11.6)	2 (15.4)	0 (0.0)	7 (10.3)
Regressed/Treated	14 (100)	38 (88.4)	11 (84.6)	7 (100)	61 (89.7)
Total	14(100)	43 (100)	13 (100)	7 (100)	68 (100)

Abbreviations: TVUS, transvaginal ultrasound. Data are presented as n (%). P-value derived from Chi-square test; P = 0.553.

5. Impact of ET cutoff ≤ 5 mm

When endometrial thickness (ET) was dichotomized into ≤ 5 mm and > 5 mm, patients with ET ≤ 5 mm showed a higher rate of histopathological regression.

At the three-month follow-up, all patients with ET ≤ 5 mm demonstrated regression (6/6, 100%). In contrast, among patients with ET > 5 mm, regression was observed in 52 of 69 patients (75.4%), while 17 patients (24.6%) had persistent disease. However, this difference did not reach statistical significance (Fisher's exact test, P = 0.327) (Table 5).

Table 5
Association between Endometrial Thickness Cutoff (≤ 5 mm) and Histopathological Outcome at 3 Months

Histopathological outcome	**Endometrial thickness at 3-month ultrasound**		
	ET ≤ 5 mm	**ET > 5 mm**	**Total**
Regression	6 (100%)	52 (75.4%)	58 (77.3%)
Persistent disease	0 (0%)	17 (24.6%)	17 (22.7%)
Total	6	69	75

Abbreviations: ET, endometrial thickness. Data are presented as n (%). Fisher's exact test was used to compare histopathological outcomes between ET groups; P = 0.327.

Similarly, at the six-month follow-up, all patients with ET ≤ 5 mm showed histopathological regression (14/14, 100%). Among patients with ET > 5 mm, regression occurred in 47 of 54 patients (87%), while 7 patients (13%) had persistent disease. Statistical analysis indicated that this difference was not statistically significant (Fisher's exact test P = 0.353) (Table 6).

Table 6
Impact of ET Cutoff ≤ 5 mm at 6-Month Follow-up

Histopathological outcome	**ET ≤ 5 mm**	**ET > 5 mm**	**Total**
Regression	14 (100.0%)	47(87%)	61(89.7%)
Persistent disease	0 (0.0%)	7 (13%)	7 (10.3%)
Total	14 (100%)	54 (100%)	68 (100%)

Abbreviations: ET, endometrial thickness. Data are presented as n (%). Fisher's exact test was used to compare histopathological outcomes between ET groups; P = 0.353.

Discussion

In this study, we evaluated the diagnostic performance of transvaginal ultrasonographic endometrial thickness (ET) in predicting histopathological response to progestin therapy in patients with endometrial

hyperplasia. Our findings indicate that ET has limited discriminatory capacity and cannot reliably serve as a surrogate marker for histopathological regression. To our knowledge, limited data are available specifically evaluating endometrial thickness as a follow-up parameter in patients with medically treated endometrial hyperplasia. While several studies have assessed the diagnostic role of ultrasound in detecting endometrial pathology, few have focused on the dynamic changes in endometrial thickness after progestin therapy and its correlation with histopathologic outcomes. Therefore, our findings contribute additional evidence regarding the clinical utility and limitations of ultrasound monitoring in this specific patient population.

In the present study, ROC curve analyses at both the third and sixth months demonstrated limited overall discriminatory ability of endometrial thickness (ET) in predicting histopathologic outcomes, as reflected by non-significant AUC values. These findings indicate that when ET is evaluated as a continuous variable across all possible thresholds, its global diagnostic performance is modest and may not be sufficient as a standalone predictor of treatment response.

When a clinically meaningful threshold was applied, a distinct pattern emerged. An ET cutoff of ≤ 5 mm appeared to be associated with histopathologic regression. At 3 months, regression occurred in all patients with ET ≤ 5 mm, compared with 75.4% of those with ET > 5 mm (P = 0.177). A similar trend persisted at 6 months, where all patients with ET ≤ 5 mm demonstrated regression, whereas 87% of patients with ET > 5 mm showed regression (P = 0.353). While these differences did not reach statistical significance, likely due to the small sample size in the ET ≤ 5 mm subgroup, the consistent 100% regression rate observed in this group highlights the potential prognostic value of a ≤ 5 mm ET threshold in predicting early and sustained treatment response

However, when endometrial thickness was analyzed using the four predefined categories (≤ 5 mm, 5.1–10 mm, 10.1–15 mm, and > 15 mm), the association was not significant. At the 3-month evaluation, histopathologic outcomes did not differ significantly across these ET categories (Table 3, P = 0.422). Similarly, at the 6-month follow-up, no statistically significant relationship was found between ET category and treatment response (Table 4, P = 0.553). These findings suggest that stratifying endometrial thickness into broader, multiple ranges does not effectively distinguish between patients who achieve regression and those with persistent disease.

The lack of statistical significance when ET is categorized into wider intervals indicates that only a very thin endometrium (≤ 5 mm) may hold potential predictive value, whereas intermediate and higher thickness ranges show substantial overlap between responders and non-responders. In other words, while an ET ≤ 5 mm appears to be a useful indicator of a favorable response, increasing endometrial thickness beyond this threshold does not reliably correspond to treatment failure.

Beyond the limited discriminatory performance of ET categories, the overall diagnostic value of transvaginal ultrasonography in predicting post-treatment histopathology was poor. Consistent with our findings, ROC analyses demonstrated weak discriminatory ability at both follow-up intervals, with AUC

values of 0.369 at 3 months and 0.424 at 6 months. These values indicate that ET, even as a continuous variable, lacks sufficient accuracy for distinguishing regression from persistence.

Similarly, diagnostic indices further highlighted substantial limitations. Despite a statistically significant (though clinically paradoxical) association between TVUS findings and pathology ($p = 0.012$), the sensitivity of 71% was offset by a negligible specificity of 5.5%, leading to high rates of false-positive interpretations. The poor agreement between the two modalities, reflected by a negative Cohen's kappa coefficient ($\kappa = -0.139$), reinforces the limited reliability of TVUS as an independent tool for monitoring treatment response.

Taken together, these results suggest that while specific thresholds such as ≤ 5 mm may offer some limited guidance, transvaginal ultrasonography alone is insufficient as a surrogate for histopathological monitoring, and should not replace tissue sampling in the conservative management of endometrial hyperplasia.

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From a clinical perspective, ET > 5 mm was associated with a substantially lower regression rate, suggesting that persistent endometrial thickening after progestin therapy may reflect residual disease or incomplete response. Conversely, an ET ≤ 5 mm appears to identify a subgroup of patients with a high probability of regression. This cutoff has been widely used in postmenopausal screening contexts (7, 10). Several contemporary studies in postmenopausal women with bleeding suggest that thin endometrium in appropriately selected patients may be associated with a low likelihood of residual pathology, potentially supporting a surveillance-based approach rather than immediate biopsy in low-risk cases (7, 13). On the other hand some other met analysis concluded that ET alone could not reliably exclude malignancy (11). However all these studies were not conducted in treated patients with endometrial hyperplasia. Although ultrasonography cannot replace histopathology confirmation, these findings suggest that in carefully selected low-risk patients, particularly those without atypia and with reassuring clinical features, a cautious surveillance-based approach could be considered rather than immediate repeat biopsy.

Importantly, regression was observed across all ET categories, including patients with measurements exceeding 15 mm. This finding suggests that structural endometrial thickness does not necessarily parallel histological normalization. Similar observations have been reported in longitudinal studies where persistent thickening did not consistently correlate with histologic persistence (6, 16). Conversely, a subset of patients with relatively low ET values demonstrated persistent disease, further limiting the reliability of a fixed cutoff threshold.

During the follow-up period, endometrial polyps emerged as an important confounding factor. The strong association between endometrial thickness ≥ 10 mm and the presence of polyps in treated patients aligns with prior studies demonstrating that focal polyps can significantly increase ET measurements even among patients who ultimately achieve histopathologic regression. Because conventional

transvaginal ultrasonography may not reliably differentiate diffuse endometrial hyperplasia from focal intracavitary lesions, ET overestimation is likely to occur in such scenarios (15, 16). Adjunctive techniques such as saline infusion sonography or hysteroscopy may therefore be necessary to improve diagnostic clarity, particularly when sonographic thickening is disproportionate to clinical expectations or when persistent thickening is observed despite documented regression (17).

The higher regression rate observed at 6 months compared with 3 months reinforces the time-dependent effect of progestin therapy (14, 15). Early imaging assessment may underestimate therapeutic response and should be interpreted cautiously. From a clinical standpoint, ET measurement may provide supportive information when evaluated longitudinally; however, histopathological confirmation remains essential, particularly in atypical hyperplasia.

Detailed associations between baseline histopathology and therapeutic response at both 3 and 6 months are presented in Supplementary Tables 1 and 2. Notably, our analysis revealed a statistically significant correlation between baseline histopathological subtypes and treatment outcomes at both follow-up intervals ($p = 0.006$ at 3 months; $p = 0.001$ at 6 months). In line with the time-dependent nature of treatment response, regression patterns also varied across different histopathological subtypes. Differences in regression rates among these subtypes were consistent with their known biological behavior. Non-atypical hyperplasia demonstrated higher response rates, whereas atypical hyperplasia showed lower regression rates, likely reflecting its premalignant potential and relative progesterone resistance (5, 6). International guidelines emphasize that atypical hyperplasia requires close surveillance because of its higher progression risk (4, 14). In this subgroup, reliance on imaging parameters alone is particularly inappropriate."Although statistical association between ultrasonography and histopathology was observed, agreement was poor, and specificity was notably low. This discordance highlights a fundamental limitation: ultrasonography reflects macroscopic structural changes, whereas histopathology evaluates glandular architecture and cytologic atypia. Poor agreement between diagnostic modalities is commonly interpreted using kappa statistics, where low or negative values indicate lack of meaningful concordance (20, 21). Therefore, ET measurement cannot replace tissue-based evaluation in therapeutic follow-up.

This study has several limitations. The sample size, particularly within certain histopathologic subgroups, may have influenced the precision of diagnostic estimates. In addition, ultrasound assessment was based primarily on endometrial thickness without incorporating Doppler indices or detailed morphologic criteria, which may have limited overall discriminatory performance. The presence of focal intracavitary lesions, such as polyps, may also have contributed to thickness overestimation. Finally, as a single-center study, external generalizability may be limited.

Taken together, these findings indicate that although transvaginal ultrasonographic endometrial thickness has limited global discriminatory capacity, selected thresholds—particularly ≤ 5 mm—may offer clinically meaningful risk stratification in the follow-up of medically treated endometrial hyperplasia. A thin endometrium after progestin therapy appears to be associated with a high probability of

histopathologic regression, whereas persistent thickening (> 5 mm) should not be automatically interpreted as treatment failure. Endometrial thickness should therefore be integrated within a comprehensive clinical and pathological assessment rather than used as a standalone marker of therapeutic response.

Abbreviations

AUC
Area Under the Curve
ET
Endometrial Thickness
ROC
Receiver Operating Characteristic
TVUS
Transvaginal Ultrasonograph

Declarations

· Ethics **approval and consent to participate:**

The study protocol was approved by the Institutional Ethics Committee of Shahid Sadoughi University of Medical Sciences (Approval number: IR.SSU.MEDICINE.REC.1403.216). Informed consent was obtained from all participants.

· Consent **for publication:**

Not applicable

· Availability **of data and materials:**

The datasets used and/or analyzed during the current study are available from the corresponding author on reasonable request.

· Competing **interests:**

The authors declare that they have no competing interests.

· Funding**:**

This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.

· Authors' **contributions:**

All authors contributed to the study conception and design .M.Hajisafari Tafti: Conceptualization, Methodology, Supervision and Writing – original draft. F.akvani: Data Curation, Formal analysis and software .Z. Shiravani and A.Azad: Investigation and Visualization .All authors read and approved the final manuscript.

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Table 1 To 3

Table 1 To 3 are available in the Supplementary Files section.

Supplementary Files

This is a list of supplementary files associated with this preprint. Click to download.

- SupplementaryTables.docx
- Table1to3.docx