

# Endometrial Thinness and Infertility: A Comparative Review of Biomedical and Unani Therapeutic Concepts

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## Abstract

### Background:

Endometrial receptivity is fundamental to successful embryo implantation, and endometrial thickness is widely regarded as a reliable clinical marker of uterine readiness. A thin endometrium, commonly defined as less than 7 mm, constitutes a significant barrier to implantation and is associated with reduced conception rates, implantation failure, and suboptimal outcomes in assisted reproductive technologies. From the Unani perspective, this condition corresponds to Sue-Mizāj-e-Raḥim, particularly Barid wa Yābis Mizāj, along with Qillat-e-Dam and weakening of Harārat-e-Gharīziyya, which together impair uterine nourishment, proliferation, and receptivity. Classical Unani scholars describe that disturbances in uterine temperament, circulation, and tissue vitality compromise the Quwwat-e-Qābilah-e-Raḥim (receptive faculty of the uterus), thereby hindering implantation. Despite advances in modern reproductive medicine, thin endometrium remains a challenging clinical entity due to its multifactorial etiology, complex pathophysiology, and limited responsiveness to available therapies, a concept that closely aligns with the Unani emphasis on individualized assessment and correction of underlying uterine derangements.

### Objective:

The objective of this review is to critically examine and synthesize contemporary evidence on the impact of endometrial thickness on implantation and pregnancy outcomes, highlighting diagnostic thresholds, underlying hormonal, vascular, and molecular mechanisms, and current evidence-based management strategies, while integrating the Unani perspective by exploring the role of uterine Mizāj, Akhlāt, and Quwā in determining endometrial receptivity and fertility potential.

## Methods:

An extensive review of the literature was conducted, including observational studies, randomized controlled trials, and meta-analyses published in leading reproductive medicine journals. Studies assessing endometrial thickness in natural, ovulation-induced, and assisted reproductive technology (ART) cycles were included. Data were extracted on implantation rates, biochemical and clinical pregnancy outcomes, and live birth rates in relation to endometrial thickness and vascular parameters. In addition, classical Unani concepts were reviewed to provide a complementary perspective on uterine temperament, humoral balance, and reproductive faculty, allowing integration of traditional principles with modern evidence. Data were synthesized qualitatively to evaluate the predictive value of thin endometrium on reproductive outcomes and to discuss individualized management strategies.

## Need of the Study

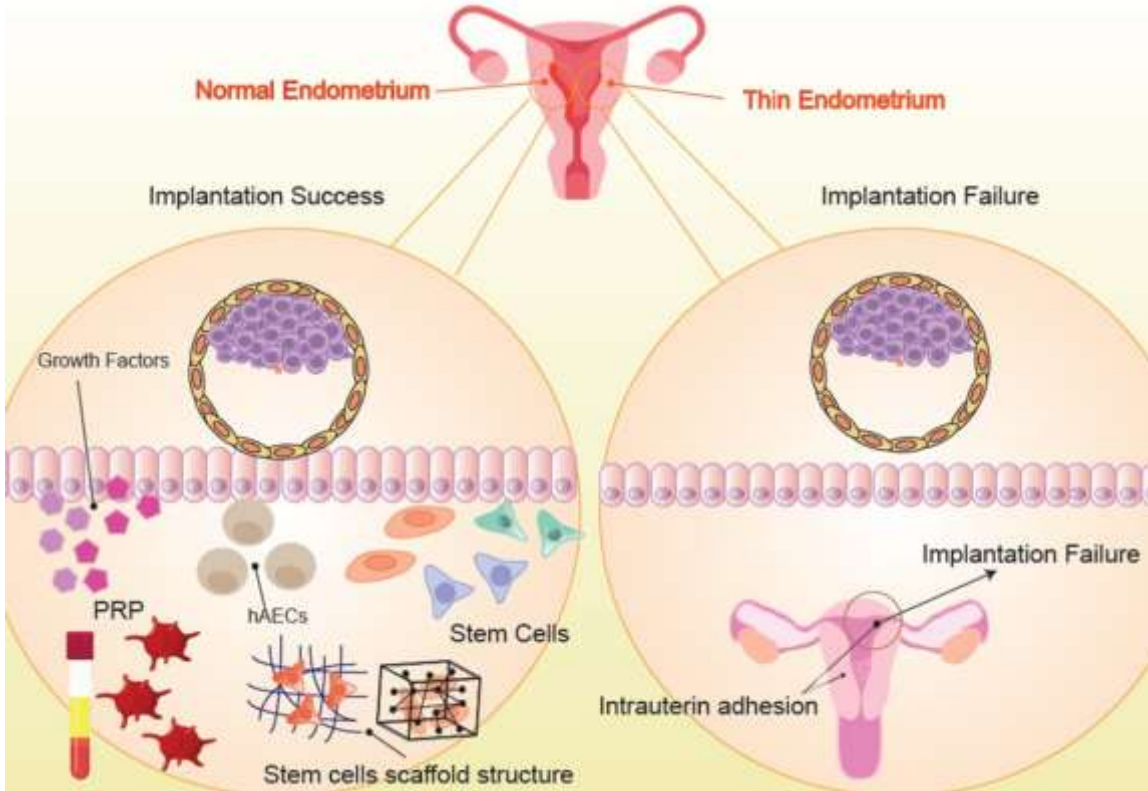
Endometrial thinness is an important yet challenging cause of female infertility, as adequate endometrial thickness and receptivity are essential for successful implantation and pregnancy. Women with thin endometrium often experience poor outcomes in natural conception as well as assisted reproductive techniques. Although several biomedical interventions such as hormonal therapy, vasodilators, and regenerative approaches are available, their effectiveness remains inconsistent, and standardized, cost-effective management strategies are still lacking.

The Unani system of medicine offers a holistic understanding of infertility by emphasizing uterine temperament, humoral balance, and nourishment of reproductive organs. Traditional Unani therapies have been used to improve uterine health and fertility, but their concepts have not been sufficiently explored in relation to modern biomedical knowledge of endometrial thinness. Therefore, a comparative review of biomedical and Unani therapeutic concepts is needed to bridge this gap, promote integrative understanding, and explore complementary approaches that may improve fertility outcomes in women with thin endometrium.

## Introduction

### Definition

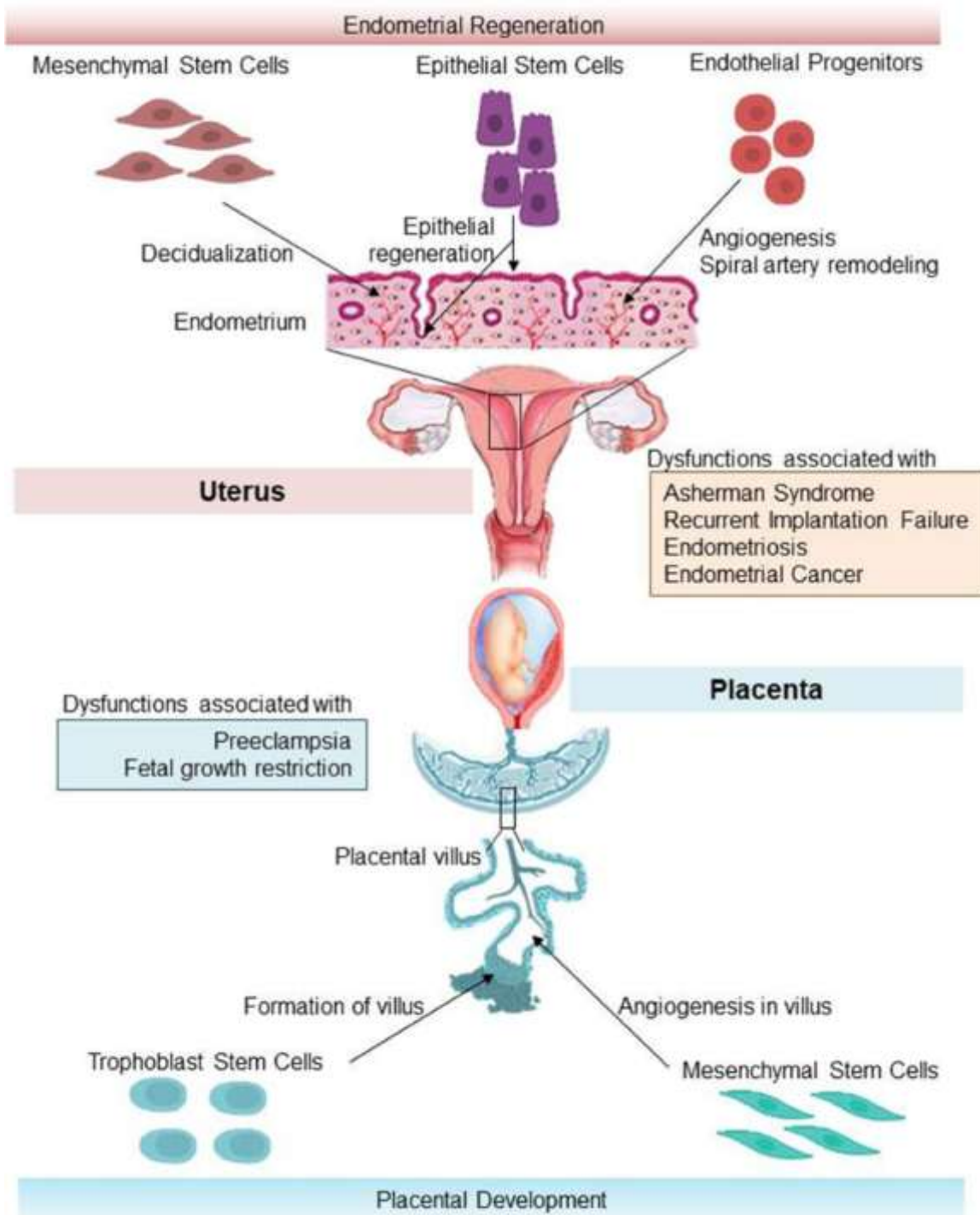
Thin endometrium (TE) is a mid-luteal or pre-transfer endometrial thickness  $\leq 7$  mm by transvaginal ultrasound (measurement in the thickest sagittal plane).



## Causes <sup>[1]</sup>

- Endometrial scarring / Asherman's syndrome — prior aggressive curettage, uterine surgery, postpartum or post-abortion infection → loss of basalis layer and scarring.
- Impaired uterine perfusion / poor angiogenesis — high uterine artery impedance, reduced vascular bed (arteriolar supply) → limited stromal proliferation.
- Defective endometrial stem/progenitor cell niche — loss or dysfunction of progenitors that regenerate the functional layer.
- Hormonal causes — low circulating estradiol, poor local estrogen signaling, dysfunctional estrogen receptor expression, or anti-estrogen effects of some drugs (e.g., clomiphene citrate)
- Chronic endometritis / infection — chronic inflammation impairs proliferation and angiogenesis.
- Radiation / pelvic chemotherapy — damage to endometrium and vasculature.
- Age and diminished ovarian reserve — lower ovarian steroid output and poorer endometrial response.

## Stem Cells in Women's Health



### Unani concept

- Ibn Sīnā (Avicenna) explains that coldness and dryness of the uterus weaken its quwwat-e-musakkinah (restorative/settling power), preventing proper imtilā' (filling) of the uterine tissues<sup>[8]</sup>
- Zakariyyā Rāzī observes that when the uterus receives insufficient dam (blood), its layers become thin and weak, and the organ loses the capacity to retain pregnancy.<sup>[9]</sup>
- Ismā'īl Jurjānī notes that uterine weakness caused by dryness reduces the organ's ability to accept the nutfah (seed), impairing conception.<sup>[10]</sup>

- Ibn Hubāl al-Baghdādī states that when the uterine layers are deprived of proper ghizā (nourishment), they become thin and lose their qābilyat-e-qabūl (receptive capacity).<sup>[11]</sup>
- Similarly, Jurjānī (further notes) that impairment of the uterus's absorbing and retaining powers leads to insufficient buildup of its inner lining.<sup>[12]</sup>

Classical Unani descriptions—such as huzūl (atrophy), qillat-e-dam (poor vascularity), su'-mizāj barīd wa yābis (cold-dry temperament), and qillat-e-ghizā (insufficient nourishment)—map closely to the modern concept of a thin endometrium and offer the theoretical basis for its etiology in Unani theory.

### Pathophysiology <sup>[2,3]</sup>

#### Reduced estrogen-driven proliferation

Estrogen (E2) acting via ER $\alpha$ /ER $\beta$  in stromal cells stimulates proliferation of stromal matrix and indirectly supports epithelial growth. Low systemic E2 or reduced ER expression  $\rightarrow$  decreased mitotic signaling (cyclins, growth factors).

#### Unani concept correlation:

In Unani medicine, proper endometrial growth depends on *Harārat-e-Gharīziyya (innate heat)* and balanced *Akhilāt*, particularly *Dam (blood)*. Reduced estrogenic activity corresponds to:

- Barūdat-e-Raḥim (uterine coldness)
  - Qillat-e-Dam (deficiency of sanguine humour)
- These conditions weaken the nutritive and formative capacity of the uterus, leading to poor endometrial proliferation.<sup>[16]</sup>

#### Impaired angiogenesis and reduced VEGF signaling

VEGF (vascular endothelial growth factor) and angiopoietins are essential for capillary sprouting and stromal perfusion. TE shows  $\downarrow$ VEGF expression, fewer microvessels  $\rightarrow$  hypoperfusion  $\rightarrow$  less nutrient/oxygen delivery for proliferative expansion. Biochemically, reduced VEGF and downstream PI3K/AKT and MAPK signaling blunt endothelial proliferation.

#### Unani concept correlation

Adequate uterine perfusion is described as Husn-e-Jaryān-e-Dam fi'l-Raḥim (proper blood flow in the uterus). Impaired angiogenesis aligns with:

- Sue-Mizāj Bārid wa Yābis (cold and dry temperament)
- Da'f-e-Quwwat-e-Ghādhīyah (weak nutritive faculty)

Poor vascular supply leads to insufficient nourishment of the endometrium, hindering its growth.<sup>[17]</sup>

#### Stem/progenitor cell dysfunction and extracellular matrix (ECM) changes

Endometrial regeneration after menses relies on basalis progenitors. Dysfunction leads to reduced epithelial/stromal expansion. Altered ECM proteins (fibronectin, collagen deposition) and increased TGF- $\beta$  signaling promote fibrosis and scarring. Biochemical hallmarks include elevated TGF- $\beta$ , altered MMP/TIMP balance (reduced MMP activity  $\rightarrow$  ECM accumulation).

### Unani concept correlation:

This process corresponds to:

*Da'f-e-Quwwat-e-Muwallidah* (weak regenerative/formative faculty)

*Ghalba-e-Balgham or Sawdā* (phlegmatic or melancholic dominance) *Excess Yubūsat* (dryness) promotes *Tasallub* (hardening/fibrosis) of uterine tissue, preventing normal regeneration after menstruation.<sup>[18]</sup>

### Impaired local growth factor and cytokine milieu

Lower local IGF, EGF, LIF (leukemia inhibitory factor) and aberrant prostaglandins reduce stromal proliferation and receptive secretory transformation. LIF and integrin expression ( $\alpha\text{v}\beta\text{3}$ ) are key to implantation — their deficiency correlates with poor receptivity.

### Unani concept correlation:

Implantation requires *Quwwat-e-Qābilah-e-Raḥim* (receptive faculty of the uterus) and a balanced uterine environment. Deficiency of local growth factors parallels:

- Sue-Mizāj Raḥim
- Ikhtilāl-e-Ratūbat-e-Mu'tadilah (disturbed optimal moisture)
- This results in failure of the uterus to accept and retain the fertilized ovum.<sup>[9]</sup>

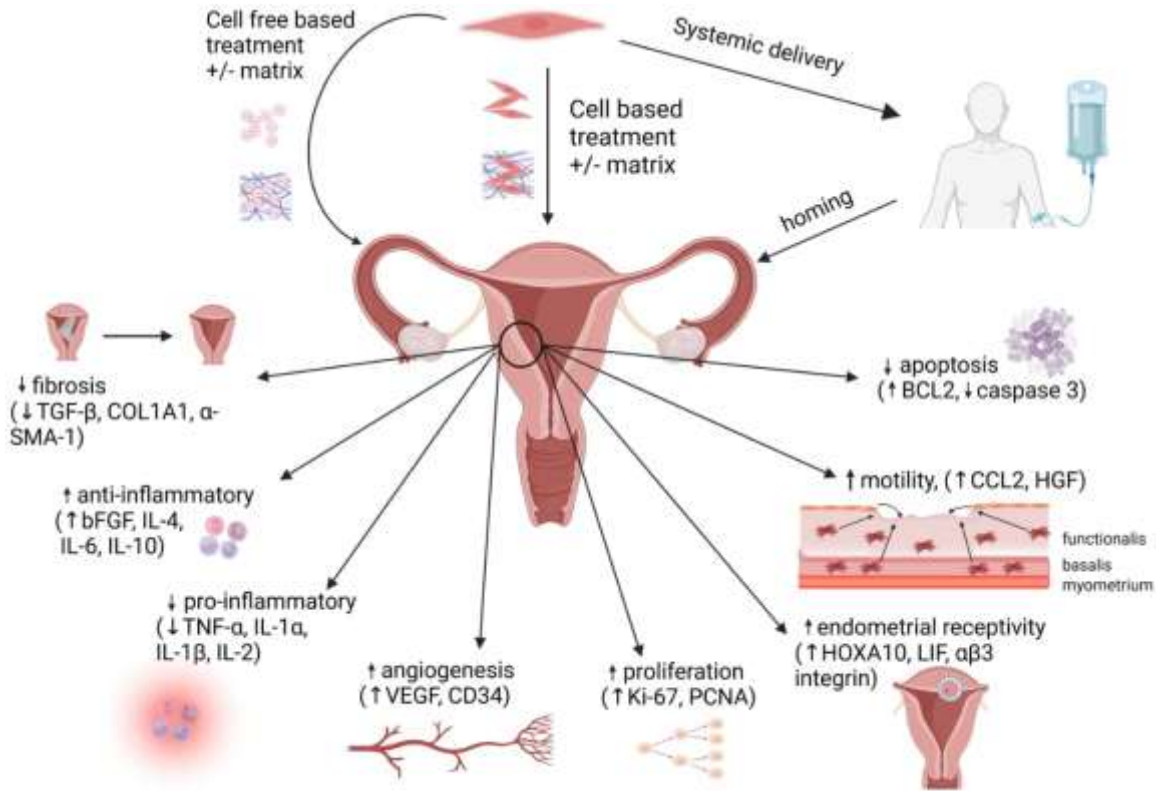
### High uterine artery impedance / poor perfusion

Elevated resistance index (RI) on Doppler indicates poor blood flow; biochemical consequence is tissue hypoxia → HIF1 $\alpha$  pathway activation but insufficient angiogenic response. Chronic hypoxia favors fibrosis rather than regeneration.

### Unani concept correlation:

This is analogous to:

- Sudda-e-Raḥim (uterine obstruction)
- Burūdat wa Yubūsat (coldness and dryness)
- Chronic hypoxia corresponds to reduced *Harārat-e-Gharīziyya*, leading to diminished tissue vitality and fibrotic changes instead of healthy endometrial growth.<sup>[19]</sup>



Modern Mechanism	Unani Concept
Low estrogen signaling	Barūdat-e-Rahim, Qillat-e-Dam
Reduced VEGF/angiogenesis	Ḍa‘f-e-Quwwat-e-Ghādhiyah
Fibrosis, ECM accumulation	Ghalba-e-Sawdā, Yubūsat
Low LIF, IGF, integrins	Ḍa‘f-e-Quwwat-e-Qābilah
Poor uterine perfusion	Sudda-e-Rahim, reduced Harārat

### clinical presentation<sup>[4,5]</sup>

Thin endometrium itself usually does not produce unique pelvic symptoms — most patients are asymptomatic and present because of infertility or implantation failure. Possible associated clinical features:

- History of prior uterine instrumentation (D&C, hysteroscopy), infection, or radiation.
- Recurrent implantation failure (RIF) or repeatedly low pregnancy rates in IVF.
- Abnormal uterine bleeding may be present if endometrial regeneration is altered (less common).
- Menstrual changes (lighter menses) possible if basalis is damaged.

### Investigations<sup>[5]</sup>

#### Essential investigations :

Transvaginal ultrasound (TVS) — measurement of endometrial thickness (EMT) in midsagittal plane (double-layer).

Note: timing matters (end of stimulation, day of hCG, or day of embryo transfer). Also report: endometrial pattern (triple-line vs homogeneous), volume (3D if available), and subendometrial blood flow (color Doppler).

Doppler ultrasound of uterine/ radial arteries — report resistance index (RI), pulsatility index (PI) — high impedance correlates with thin/poorly perfused endometrium.

Hysteroscopy — direct inspection for adhesions (Asherman), polyps, synechiae; therapeutic if present.

Endometrial histology/ biopsy — to rule out chronic endometritis (microbial culture, plasma cells on histology), to assess secretory transformation if indicated.

Laboratory endocrine tests — serum estradiol, progesterone, TSH, prolactin, AMH (ovarian reserve), as clinically indicated. Low E2 suggests poor estrogenic stimulation.

**Scoring system** — practical, evidence-based composite score for “Influence of Thin Endometrium on Infertility”.<sup>[6,7]</sup>

### **Endometrial thickness (EMT) — 0–4 points**

- EMT < 6.0 mm → 0 points
- EMT 6.0–6.9 mm → 1 point
- EMT 7.0–8.9 mm → 2 points
- EMT 9.0–11.9 mm → 3 points
- EMT ≥ 12.0 mm → 4 points

(Rationale: many studies use ≤7 mm as threshold for thin; pregnancy rates increase as EMT rises).

### **Endometrial pattern on TVS (echo pattern) — 0–2 points**

- Homogeneous hyperechoic / trilaminar absent → 0 points
- Intermediate / unclear pattern → 1 point
- Trilaminar (triple line) pre-ovulation or appropriate secretory pattern → 2 points

(Echo pattern linked to receptivity in ultrasound-based scoring literature).

### **Endometrial/subendometrial blood flow (color Doppler) — 0–2 points**

- No subendometrial flow / high uterine artery RI (>0.8) → 0 points
- Minimal flow / moderate RI (0.6–0.8) → 1 point
- Good subendometrial flow / low RI (<0.6) → 2 points

### **Endometrial volume / 3-D assessment or other markers — 0–2 points**

- If 3-D available: Volume below institutional cutoff (example < 2 mL) or absent peristalsis → 0 points
- Moderate volume/peristalsis present → 1 point
- Adequate volume and normal peristalsis → 2 points

**Interpretation (ERS-10):**

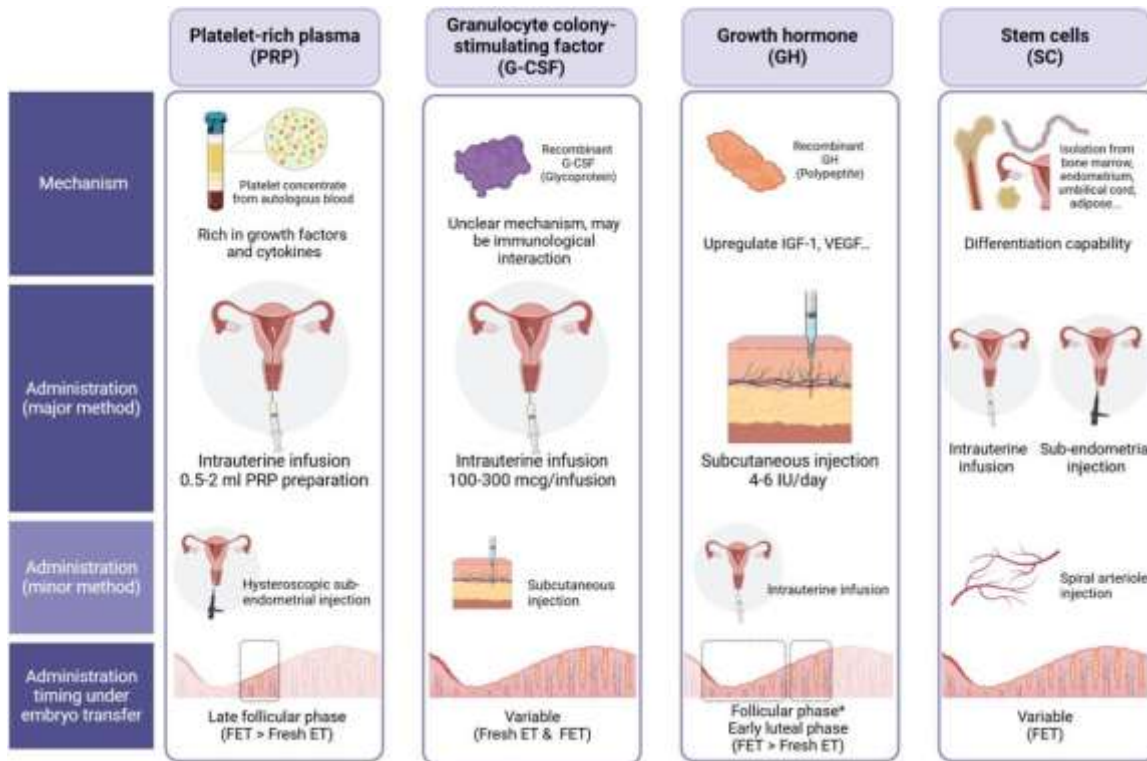
**0–3 (High negative influence)** — Very poor endometrial receptivity; consider cancelling transfer, treat underlying cause (e.g., hysteroscopy for synechiae, estrogen priming, PRP/G-CSF in selected centres), or consider deferring embryo transfer.

**4–6 (Moderate influence)** — Reduced receptivity; consider optimization before transfer (medical/regenerative strategies), discuss prognosis with patient.

**7–10 (Low negative influence / favorable)** — Endometrium likely adequate for transfer; expected better implantation probability.

**Treatment**

Treatment Category	Method / Examples	Mechanism of Action	Clinical Use
Hormonal Therapy	High-dose estrogen (oral/patch), Estradiol valerate	Stimulates endometrial proliferation	First-line, especially in hypoestrogenic states
Vasodilators	Sildenafil citrate (oral/vaginal), Pentoxifylline	Improves uterine blood flow and angiogenesis	Useful when Doppler shows high RI/low flow
Anti-inflammatory Therapy	Antibiotics for chronic endometritis	Treats infection, restores normal endometrial function	Requires confirmation via hysteroscopy or biopsy
Uterine Regenerative Therapies	Platelet-Rich Plasma (PRP) intrauterine infusion	Enhances tissue repair, stem cell activation	Used in recurrent thin endometrium; promising
Growth Factor Therapy	G-CSF intrauterine infusion	Improves endometrial growth and vascularity	Particularly used in ART cycles with persistent thin lining
Endometrial Scratch / Local Injury	Pipelle biopsy scratch	Promotes local inflammatory response improving receptivity	May help in repeated implantation failure
Surgical Correction	Hysteroscopic adhesiolysis	Removes intrauterine adhesions or fibrosis	Essential for Asherman’s syndrome-related thin endometrium
Nutritional & Adjunct Options	Vitamin E, L-arginine	Antioxidant & nitric oxide-mediated vasodilation	Adjunct therapy; modest benefit
Adjuvant Medical Options	Low-dose aspirin	Improves uterine perfusion	Adjuvant Medical Options Low-dose aspirin Improves uterine perfusion Used as supportive therapy in ART



### unani treatment<sup>[13,14,15]</sup>

Many Unani single and compound formulations possess actions that support uterine health and may help in improving the environment of a thin endometrium according to Unani principles. Asl-us-Soos (*Glycyrrhiza glabra*) is considered mulattif and muqawwi, Badiyan/Fennel (*Foeniculum vulgare*) as mufatteḥ and muqawwi-e-raḥim, Anar (*Punica granatum*) as qabiz and muqawwi, while Asgand (*Withania somnifera*) is listed as muqawwi-e-badan and muqawwi-e-aaza, Shatavari (*Asparagus racemosus*) as mughazzi, muwallid-e-manī and muqawwi-e-raḥim, and Tukhm-e-Konch (*Mucuna pruriens*) as muwallid-e-manī, muqawwi-e-asab and muqawwi-e-quwwat-e-jinsiyyah. Compound formulations such as Majoon Supari Pak (muqawwi-e-raḥim), Majoon Hamal (for taqwiyat-e-raḥim), and Jawarish Zaruni (quwwat-e-ma'ida wa raḥim) contain phytoestrogen-related herbs like licorice, fennel, and pomegranate. Together, these documented actions—muqawwi, mulattif, muwallid-e-manī, and taqwiyat-e-raḥim—support uterine tonicity, nourishment, and hormonal balance, and thus may help improve the uterine environment in cases of thin endometrium within the Unani framework.

### Data and Sources of Data

This study is based on secondary data collected from published and authentic sources. Biomedical information related to endometrial thinness and infertility was obtained from peer-reviewed journals, review articles, clinical guidelines, and standard textbooks accessed through databases such as PubMed, Google Scholar, and WHO publications. Data related to the Unani perspective were collected from classical Unani texts and published articles in reputed AYUSH and Unani journals. All relevant literature was systematically reviewed to provide a comparative analysis of biomedical and Unani therapeutic concepts.

### Results

Thin endometrium ( $\leq 7$  mm) is consistently associated with impaired endometrial receptivity and reduced fertility outcomes, as evidenced by poor ultrasonographic patterns, decreased subendometrial blood flow with high uterine

artery resistance, reduced endometrial volume, pale mucosa, and intrauterine adhesions, along with lower implantation and clinical pregnancy rates despite good-quality embryos. In Unani medicine, these findings correspond to Sue-Mizāj-e-Raḥim, particularly Barid wa Yābis Mizāj, resulting from reduced Harārat-e-Gharīziyya, Qillat-e-Dam, and dominance of Balgham or Sawdā, which impair uterine tissue proliferation and receptivity. Decreased uterine perfusion and high vascular resistance reflect Sudda-e-Raḥim and weakened Quwwat-e-Ghādhīyah, while pale endometrium, adhesions, and fibrosis correspond to Yubūsāt wa Tasallub-e-Raḥim. The frequent association of thin endometrium with chronic endometritis, low estradiol levels, and prior uterine instrumentation further aligns with Iltehāb-e-Raḥim Muzmin and Ḍaʿf-e-Raḥim described in classical Unani texts. Thus, both modern evidence and Unani principles recognize thin endometrium as an independent negative factor for implantation, arising from combined disturbances in uterine temperament, vascularity, nutrition, and Quwwat-e-Qābilah-e-Raḥim.

## Discussion

The available literature demonstrates that thin endometrium is a multifactorial condition leading to compromised endometrial receptivity and poor fertility outcomes; this concept closely aligns with the Unani understanding of Ilal-e-Raḥim arising from combined disturbances in Mizāj, Akhlāt, and uterine Quwā. Impaired estrogen responsiveness and reduced angiogenesis correspond to Barūdat-e-Raḥim, Qillat-e-Dam, and weakening of Harārat-e-Gharīziyya, resulting in deficient tissue proliferation and nourishment. Chronic inflammation and structural damage due to curettage or adhesions are analogous to Iltehāb-e-Raḥim Muzmin, Sudda, and Tasallub-e-Raḥim, which impair uterine vascularity and receptivity. Reduced expression of implantation mediators such as VEGF, LIF, integrins, and growth factors reflects weakening of Quwwat-e-Ghādhīyah and Quwwat-e-Qābilah-e-Raḥim, while ultrasonographic findings of poor echo pattern, low vascularity, and reduced endometrial volume signify Sue-Mizāj Barid wa Yābis with dominance of Balgham or Sawdā. Although various therapeutic approaches have been explored in modern medicine, their variable efficacy supports the Unani principle that management should be individualized, focusing on correction of uterine temperament, removal of obstruction and inflammation, improvement of circulation, restoration of humoral balance, and strengthening of uterine faculties, which together remain the most rational strategy for improving reproductive outcomes in women with thin endometrium.

## Conclusion

Thin endometrium is a clinically significant and independent factor contributing to infertility by compromising endometrial receptivity, uterine vascular perfusion, and the biological capacity for embryo implantation. Evidence consistently shows that an endometrial thickness of  $\leq 7$  mm, particularly when associated with poor vascular flow or abnormal echogenic pattern, is linked to markedly reduced implantation and pregnancy rates in both natural and assisted reproductive cycles. In the Unani system of medicine, this condition corresponds to Sue-Mizāj-e-Raḥim, especially Barid wa Yābis Mizāj, along with Qillat-e-Dam, Ḍaʿf-e-Quwwat-e-Ghādhīyah, and impairment of Quwwat-e-Qābilah-e-Raḥim, leading to inadequate uterine nourishment and receptivity. The underlying pathologies—such as hormonal imbalance, chronic inflammation, intrauterine adhesions, and impaired regenerative capacity—are analogous to Iltehāb-e-Raḥim Muzmin, Sudda, and Tasallub-e-Raḥim described in classical Unani texts. Comprehensive evaluation of endometrial thickness, ultrasonographic pattern, Doppler blood flow, and uterine structure aligns with the Unani emphasis on assessing uterine temperament, circulation, and tissue vitality rather than a single parameter alone. While various therapeutic measures are described in both systems, their variable outcomes reinforce the need for early detection, individualized management, correction of underlying causes, and restoration of uterine temperament and faculties to improve reproductive outcomes.

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