

Beyond the surface: A closer look at hirsutism

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Abstract

Hirsutism is defined as excessive hair growth in females that resembles male-pattern hair distribution. This condition often causes significant emotional distress and affects a considerable number of women seeking dermatological care. Hirsutism can arise from various etiologies, most commonly linked to androgen excess originating from the ovaries or adrenal glands, and is frequently associated with metabolic disorders such as polycystic ovary syndrome (PCOS). In some cases, hirsutism occurs idiopathically, as a side effect of medication, or in rare instances, as a manifestation of underlying serious medical conditions. This narrative review explores the multifaceted nature of hirsutism, including its prevalence, etiological factors, diagnostic approaches, treatment options, and psychological impact, aiming to enhance understanding and improve clinical care for affected women.

Key words

Hirsutism; Hyperandrogenism; Polycystic Ovary Syndrome; Hypertrichosis; Congenital adrenal hyperplasia.

Introduction

Hirsutism, characterized by excessive growth of terminal coarse hair in females, following a male-pattern distribution, is a global concern, impacting approximately 5-10% of women worldwide.¹ Hirsutism is specifically linked to elevated levels of androgens, particularly testosterone, which drives the transformation of fine vellus hair into thicker terminal hair in androgen sensitive regions including the upper lip, chin, chest, abdomen, and back and is often accompanied by signs such as acne.^{2,3} The etiology of hirsutism is multifactorial, including

endocrine and nonendocrine causes, certain medications and idiopathic hirsutism.⁴⁻⁶

Racial and ethnic variations play an important role in the prevalence of hirsutism, for instance, women from Hispanic, Middle Eastern, and African American backgrounds exhibit higher rates compared to Caucasian and East/Southeast Asian women.⁵ The psychosocial impact of hirsutism in these communities cannot be understated, as it significantly affects quality of life, often leading to emotional distress, anxiety, and diminished self-esteem.⁷ Therefore, a comprehensive approach is essential for addressing the multifaceted nature of hirsutism and its impact on women's health.

By accurately identifying the underlying causes and assessing disease severity, clinicians can offer tailored therapeutic approaches that combine pharmacological interventions with

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cosmetic treatments to improve both physical symptoms and the quality of life of patients. But the severity of hirsutism, as assessed by tools like the Ferriman-Gallwey scoring system, does not always correlate with the psychological burden experienced by affected individuals.⁸ The overlap between hirsutism and hypertrichosis (non-androgen-dependent excessive hair growth) further complicates diagnosis, underscoring the need for more precise frameworks to differentiate between these conditions.^{9,10} Moreover, despite advances in treatment options such as anti-androgen medications, lifestyle interventions, and cosmetic procedures, gaps persist in the understanding of optimal long-term management.¹¹ This review aims to address the gaps in diagnosis and management of hirsutism with latest advancements in diagnostic tools and therapeutic strategies while identifying areas where further research is required.

Methods

This narrative review was conducted with the aim of exploring the etiologies, prevalence, and treatment strategies associated with hirsutism. We searched across multiple high-impact medical databases, including Medline, Embase, PubMed, Scopus, and the NIH repository, ensuring that the most recent and relevant literature from 2018 to 2024 was reviewed to update the existing knowledge. The search was carried out using a predefined set of keywords such as "hirsutism," "hyperandrogenism," "polycystic ovary syndrome," "congenital adrenal hyperplasia," and other associated medical conditions. The strategy was designed to maximize sensitivity and specificity, with Boolean operators and controlled vocabulary (MeSH terms) incorporated to ensure appropriate literature coverage. The review process employed a two-tiered screening approach. First, two independent reviewers conducted an initial screen of titles and abstracts

to identify studies that met the predefined inclusion criteria. These criteria focused on studies that examined the pathophysiology, diagnosis, epidemiology, and management of hirsutism. Full-text articles were then subjected to a secondary in-depth evaluation. Where discrepancies arose between the reviewers, they were resolved by through consensus discussions with the principal investigator.

Quality appraisal of the included studies was performed using validated tools such as the Newcastle-Ottawa Scale for observational studies and Cochrane Risk of Bias tools for randomized controlled trials. We also utilized data extraction sheets to collect key information systematically, including patient demographics, study design, hormonal profiles, diagnostic criteria and treatment options. Only studies meeting stringent methodological and clinical relevance thresholds were included in the final synthesis. Approximately 40% of the screened articles were selected for inclusion based on their ability to contribute substantive, evidence-based insights into the underlying mechanisms and management of hirsutism.

Epidemiology and demography

The global prevalence of hirsutism is challenging to determine precisely; however, estimates suggest that it affects approximately 5-10% of women, with significant variability influenced by geographic, ethnic, and genetic factors.^{1,12} Hirsutism is more prevalent among certain populations, particularly those of Mediterranean, South Asian, and Middle Eastern descent.⁴ These groups often exhibit heightened sensitivity to androgens, even when serum androgen levels are comparable to those in other populations⁴. Social and cultural norms further shape the perception and acceptability of hirsutism, impacting its clinical presentation and the psychological well-being of affected individuals.¹³



Figure 1 Hirsutism in a woman with PCOS a) before and b) after treatment.

Causes and their prevalence

Investigating the underlying cause of hirsutism is essential for effective treatment. Current literature identifies three main causes of hirsutism:

1. Ovarian Factors (Approximately 60%). Ovarian factors encompass conditions like polycystic ovarian syndrome (PCOS) and ovarian tumours.

- Polycystic Ovary Syndrome (PCOS): The most common cause, which is a hormonal disorder that affects the ovaries and causes irregular periods, obesity, and hirsutism.¹⁴

- Ovarian Tumors: Less common, but androgen secreting tumors manifest by symptoms of androgen excess.

2. Adrenal Factors (Approximately 10%)

- Cushing's Disease: It is caused by a benign tumor located in your pituitary gland that releases ACTH. Excess ACTH causes your adrenal glands to release excess androgens

- Adrenal Tumors: Some adrenal tumors can produce androgens.

- Congenital Adrenal Hyperplasia (CAH): A genetic disorder that affects the adrenal glands, often due to 21-hydroxylase deficiency.¹⁵

3. Other Causes—Less than 10%

- Hyperandrogenic-Insulin Resistant-Acanthosis Nigricans Syndrome (HAIRAN): A rare condition associated with insulin resistance and skin darkening.

- Hyperprolactinemia: Excessive prolactin production.

- Androgenic Drug Use: Certain medications can increase androgen levels.

Idiopathic Hirsutism (IH)-20% Idiopathic hirsutism, means the Hirsutism without androgen excess.¹⁶ IH is characterized by normal androgen levels and ovarian function. Some



Figure 2 Paediatric patient suffering from pemphigus vulgaris with glucocorticoid-induced cushing syndrome. Because pure glucocorticoids have no androgenic activity, the treatment rarely produces hirsutism. Instead, glucocorticoid therapy is one of the causes of hypertrichosis.



Figure 3 Topical corticosteroid-induced hypertrichosis and skin atrophy in a patient with psoriasis.

Table 1 Mechanisms contributing to hirsutism.

| <i>Producing organ</i> | <i>Action that triggers hirsutism</i> |
|-------------------------------------|---|
| Ovaries and suprarenal glands | Increased production of hormone in either one or both. |
| Receptors in target organ | Increased sensitivity to 5 α -reductase activity. |
| Sex hormone binding globulin (SHBG) | SHBG increase decreases active hormone in circulation. SHBG decrease increases the action of the active circulating hormone. |

studies suggest that many women with idiopathic hirsutism actually have PCOS, but it is not detected by conventional tests. Idiopathic hirsutism, characterized by normal androgen levels, may be associated with androgen receptor polymorphisms, or altered androgen sensitivity in XY females (patients have actually males genotype with female external genitalia) as in peripheral 5-alpha reductase deficiency.^{17,18} IH typically manifests shortly after puberty and then progresses gradually.

Pathogenesis

The development and distribution of hair in hirsutism are influenced by a combination of growth factors, cytokines, and sex steroids, with androgen excess-particularly testosterone and its potent derivative, dihydrotestosterone (DHT)-playing a central role.^{19,20} During puberty, androgens stimulate the transformation of fine, non-pigmented vellus hairs into thicker, pigmented terminal hairs in androgen-sensitive areas such as the face, chest, and back. This process is mediated by the enzyme 5-alpha-reductase, which converts testosterone into DHT, promoting the prolonged growth (anagen) phase of hair follicles.²¹ The severity of hirsutism is

modulated not only by circulating androgen levels but also by the sensitivity of individual hair follicles to the androgens. This variable sensitivity, influenced by genetic and racial factors, explains why some women develop significant hirsutism despite only modest elevations in androgen levels.^{19,20} Additionally, factors such as insulin resistance and obesity can exacerbate hirsutism by lowering levels of sex hormone-binding globulin (SHBG), thereby increasing the proportion of free, biologically active testosterone.²² Beyond hormonal influences, certain medications, including anabolic steroids and oral contraceptives, can contribute to hirsutism. Lifestyle factors, such as smoking, have also been implicated in worsening the condition.²³ In rare cases, postmenopausal hirsutism may indicate more serious underlying conditions, such as androgen-secreting ovarian or adrenal tumours, necessitating thorough investigation when accompanied by rapid-onset hirsutism and signs of virilization.²⁴ Another presentation of excessive hair growth in androgen dependent area is in males with XY genotype but due to congenital deficiency in enzyme 5 alpha reductase, the male external genitalia fail to develop giving a female phenotypical appearance.

Table 2 Medications associated with Hirsutism across clinical categories.

| <i>Category</i> | <i>Medications causing Hirsutism (excessive hair growth on androgen-dependent areas in females)</i> |
|--|---|
| Alzheimer's Disease and Cognitive Function | Donepezil |
| Epilepsy and Seizure Disorders | Carbamazepine, Diazoxide, Lamotrigine, Phenytoin, Tiagabine, Zonisamide |
| Endocrine and Hormonal Disorders | Leuprolide, Progestins |
| Hormonal Therapy | Estrogens, Testosterones |
| Immunosuppressant Medications | Interferon alfa, Mycophenolate, Tacrolimus |
| Mental Health and Mood Disorders | Aripiprazole, Bupropion, Clonazepam, Fluoxetine, Olanzapine, Paroxetine, Trazodone, Venlafaxine |
| Neurological Disorders (Muscle Relaxants and Spasticity) | Dantrolene, Selegiline |
| Ophthalmic Medications | Bimatoprost |
| Sleep Disorders | Eszopiclone |

Table 3 Medications associated with hypertrichosis across clinical categories.

| Category | Medications causing Hypertrichosis (Excessive Hair Growth) |
|--|--|
| Acne and Hyperpigmentation | Azelaic acid |
| Antidepressant (SSRI) | Citalopram |
| Antiepileptic Medications | Phenytoin |
| Antihistamines | Cetirizine |
| Antihypertensive Medications (Sympatholytic) | Methyldopa |
| Antibiotic | Streptomycin |
| Antiemetic Medications | Metoclopramide |
| Diuretics | Acetazolamide, Dioxazide |
| Glucocorticoid | Hydrocortisone |
| Hair Loss | Minoxidil |
| Heavy Metal Chelating Agents | Penicillamine |
| Hypertensive Emergencies | Diazoxide |
| Immunosuppressive Medication | Cyclosporine |
| Light Sensitization Therapy (Phototherapy) | Psoralens |
| Psoriasis and Severe Skin Conditions | Acitretin, Cyclosporine |
| Topical Skin Conditions | Corticosteroids (topical) |
| Antipsychotic Medications | Phenothiazines |

Table 4 Common sites of hirsutism.

- Face (including moustache, beard, and temple areas)
- Chest
- Areolae
- Linea alba
- Upper back
- Lower back
- Buttocks
- Inner thighs
- External genitalia

Clinical features and physical examination

The clinical evaluation of hirsutism begins with a comprehensive assessment of the distribution and density of terminal hair.²⁵ **Table 4** enumerates Important sites for assessment of excess terminal hair growth.

The Ferriman-Gallwey scoring system is the most widely utilized tool for quantifying the severity of hirsutism, assessing hair growth in nine androgen-sensitive areas, including the upper lip, chin, chest, abdomen, and back. A score of 8 or higher indicates clinically significant hirsutism, which can be further categorized into mild (8-16), moderate (17-25), and severe (greater than 25).²⁵ In primary care settings, a simplified version of this scoring system, focusing on three key areas- chin, lower abdomen, and upper thigh- is often employed to facilitate quicker assessments.²⁶

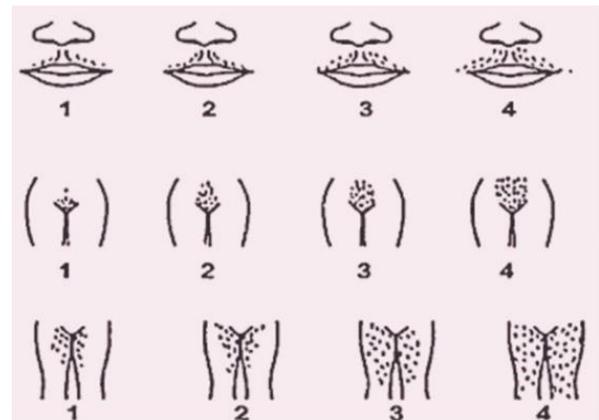


Figure 4 The simplified Ferriman-Gallwey scoring system.

During the physical examination, it is crucial to identify signs of virilization, such as deepening of the voice, clitoromegaly, and increased muscle mass, as these may indicate significant androgen excess from sources such as adrenal or ovarian tumours.²⁵ Additional clinical signs, including obesity and acanthosis nigricans, can suggest underlying insulin resistance, which is commonly associated with conditions like polycystic ovary syndrome (PCOS).²⁷ The presence of hirsutism can have profound psychological effects on affected individuals, often leading to anxiety and depression due to societal stigma and personal distress.⁸ Therefore, a thorough clinical evaluation not only focuses on the physical manifestations of hirsutism

but also considers the psychological impact on the patient. This holistic approach is essential for developing effective management strategies tailored to the individual needs of women suffering from hirsutism.

In brief, the clinical features of hirsutism are assessed through a systematic approach that includes the Ferriman-Gallwey scoring system and a detailed physical examination to identify signs of virilization and associated conditions. Understanding the multifaceted nature of hirsutism, including its psychological implications, is vital for effective diagnosis and management.

A thorough diagnostic workup for hirsutism begins with a comprehensive medical history that focuses on the onset and progression of symptoms, menstrual irregularities, signs of hyperandrogenism, family history, and current medications. Gradual-onset hirsutism is often associated with conditions such as polycystic ovary syndrome (PCOS) and idiopathic hirsutism, while rapid-onset hirsutism raises suspicion for more serious etiologies, including androgen-secreting neoplasms.¹ Laboratory evaluation typically includes the measurement of serum androgens, specifically testosterone, free testosterone, and dehydroepiandrosterone sulfate (DHEA-S), to assess adrenal and ovarian androgen production.²⁸ An elevated luteinizing hormone (LH) to follicle-stimulating hormone (FSH) ratio (>2:1) is

commonly seen in women with PCOS but is not diagnostic on its own.²⁹ Additional hormonal assays, such as 17 alpha-hydroxyprogesterone for congenital adrenal hyperplasia (CAH) and cortisol levels for suspected Cushing's syndrome, may be indicated depending on the clinical context.^{28,30} The integration of clinical findings with laboratory results is crucial for accurate diagnosis. For instance, the presence of signs such as virilization, obesity, and acanthosis nigricans can indicate underlying conditions like insulin resistance, which is frequently associated with PCOS.³¹ Furthermore, genetic testing may be warranted in cases of suspected non-classic CAH, as early recognition and diagnosis are essential to initiate appropriate treatment and prevent long-term complications associated with androgen excess, such as infertility and metabolic disturbances.^{32,33} In summary, the diagnostic approach for hirsutism is multifaceted, requiring a detailed medical history, physical examination, and targeted laboratory evaluations. By synthesizing clinical insights with recent research findings, healthcare providers can enhance their understanding of hirsutism and improve diagnostic accuracy, ultimately leading to more effective management strategies for affected individuals. androgen production and action, alongside non-pharmacological approaches aimed at hair removal. Treatment plans are individualized based on the severity of symptoms, underlying etiology, and patient preferences.⁵

Table 5 Accompanying signs and symptoms in patients with hirsutism.

| <i>Accompanying Signs and Symptoms</i> | <i>Possible Diagnostic Insights</i> |
|--|---|
| Acanthosis nigricans (dark, velvety skin patches) | May indicate insulin resistance and potential association with polycystic ovary syndrome (PCOS). |
| Obesity | Obesity can be related to metabolic syndrome and contribute to hirsutism, especially in PCOS. |
| Presence of a pelvic mass | Suggests the presence of an ovarian or uterine tumour, which may produce androgens or cause hirsutism. |
| Signs or symptoms of virilization (e.g., acne, deepening of voice, irregular menstruation, Breast tissue loss, Clitoromegaly, Increased libido, Increased muscle mass) | Strongly indicative of excess androgen production, possibly due to various underlying conditions such as PCOS, ovarian or adrenal tumours, or HAIR AN syndrome. |
| Signs or symptoms of Cushing syndrome (e.g., moon face, buffalo hump, central obesity, purple striae) | Indicates possible hypercortisolism, which can be caused by adrenal tumours or other adrenal issues. |
| Acne | May suggest elevated androgen levels, which could be due to PCOS or other hormonal imbalances. |
| Alopecia (hair loss) | Hair loss, especially in a male pattern, can be associated with androgen excess, as seen in PCOS or adrenal tumours. |

Management of hirsutism

The management of hirsutism is multifaceted, integrating pharmacological interventions that target

Pharmacological Management

Pharmacological management remains the cornerstone of therapy, particularly for cases linked to androgen excess. Oral contraceptives (OCPs) are considered the first-line treatment for hirsutism, as they help regulate menstrual cycles, increase sex hormone-binding globulin (SHBG), and decrease free testosterone levels.³⁴ OCPs are especially beneficial for women seeking contraception, with certain formulations also providing advantages such as acne control.³⁵ Antiandrogens, including spironolactone, cyproterone acetate, and finasteride, block androgen receptors and inhibit the conversion of testosterone to dihydrotestosterone (DHT).³⁶ These agents are effective for treating moderate to severe hirsutism but require caution in pregnant individuals due to potential teratogenic effects. Close monitoring of potassium levels is recommended when using spironolactone.³⁷ Gonadotropin-releasing hormone (GnRH) agonists reduce ovarian androgen production by suppressing gonadotropin secretion. While potent, these agents are reserved for severe cases due to side effects, including hypoestrogenism and bone density loss.³⁸ Glucocorticoids are primarily used in cases of hirsutism associated with congenital adrenal hyperplasia (CAH), as they reduce adrenal androgen production. However, long-term use necessitates careful monitoring for adverse effects such as weight gain and osteoporosis.³⁹

Cosmetic and procedural interventions

Cosmetic and procedural interventions are critical components of hirsutism management, particularly for individuals seeking immediate and visible results. Depilation methods such as shaving, waxing, and chemical depilatories provide temporary solutions but require frequent repetition. Photoepilation techniques, including laser hair removal and intense pulsed light (IPL), offer more durable hair reduction by targeting melanin in hair

| Approach | Treatment | Mechanism of Action | Considerations |
|---------------------|---|---|---|
| Pharmacological | Oral Contraceptive Pills (OCP) | Regulates menstrual cycle, reduces androgen levels, increases SHBG, decreases free testosterone availability. | First-line treatment, suitable for contraception. Different formulations may have varying effects and side effects. Potential benefits for acne control. |
| | Antiandrogen Medications (Spironolactone, Cyproterone Acetate, Finasteride) | Blocks androgen receptors, inhibits DHT conversion, reduces androgen effects | Effective for moderate to severe hirsutism. Requires caution in pregnant individuals; Monitoring for potassium levels and signs of feminization in males. |
| | Gonadotropin-Releasing Hormone (GnRH) Agonists | Decreases gonadotropin secretion, reduces ovarian stimulation, lowers testosterone production | Reserved for severe cases; Can induce a hypoestrogenic state, necessitating add-back therapy; May impact bone health and fertility. |
| | Glucocorticoids | Suppresses adrenal androgen production, lowers androgen levels | Used in hirsutism associated with CAH; Short-term use may be considered due to long-term adverse effects; Requires careful dosing and monitoring. |
| Cosmetic Methods | Depilation (Shaving, chemical depilatory agents) | Physically removes hair shaft from the skin surface | Temporary solution, creates illusion of thicker hair; Quick and easy but needs to be repeated frequently. |
| | Bleaching | Lightens hair color, making it less noticeable | Masks appearance of undesired hair; Not effective for very dark hair; Regular use may cause skin irritation. |
| | Eflornithine | Inhibits an enzyme involved in hair growth, reducing the rate of hair growth | Particularly effective for facial hair; Takes time to show visible results; Requires continuous use for ongoing effects. |
| | Electrolysis | Destroys hair follicle using electrical current, preventing future hair growth | Time-consuming, treats each hair individually; Potential for pain, redness, and scarring; Requires skilled practitioners. |
| | Photoepilation (Laser, IPL) | Uses light sources to target and damage hair follicles, reducing hair growth | Widely used for permanent hair reduction; Multiple sessions needed for optimal results; Not effective for light or gray hair. |
| Direct Hair Removal | Laser Hair Bleaching | Weakens fine, thin, or downy hair, making it less visible | Utilizes Q-switch ND-YAG lasers; May cause temporary skin color changes; Requires multiple sessions for desired outcomes. |
| | Eflornithine (combined with other therapies) | Reduces hair growth rate by inhibiting an enzyme involved in hair growth | Enhances results when combined with lasers or IPL; Requires continuous use for prolonged effects. |

follicles to disrupt hair growth.⁴⁰ Laser treatments, particularly Nd:YAG and Alexandrite lasers, are preferred for permanent hair reduction, though multiple sessions are typically required. Nd:YAG lasers are effective for darker skin types, while Alexandrite lasers work best for lighter skin with dark hair.⁴¹ Eflornithine, a topical agent, inhibits ornithine decarboxylase and reduces the rate of hair



Figure 5 Hirsutism (excessive amount of terminal hairs in androgen-dependent sites in females).

growth, particularly effective for facial hirsutism, but must be used continuously for sustained results. When combined with laser therapy, it enhances the efficacy of hair removal.⁴²

Emerging therapies

Recent advances in the treatment of hirsutism include novel therapeutic agents. Selective androgen receptor modulators (SARMs) offer tissue-selective inhibition of androgen activity, potentially minimizing systemic side effects seen with traditional antiandrogens.⁴³ Topical antiandrogens, such as furidil and mirtazapine gel, are being explored for their ability to reduce hair growth, although further studies are needed to confirm their long-term efficacy and safety.⁴⁴ Additionally, some antibiotics, such as roxithromycin, have shown potential antiandrogenic effects, though their use remains investigational.⁴⁵ Ketoconazole, an antifungal with antiandrogenic properties, has been studied for its ability to reduce androgen production; however, its systemic side effects limit its long-term use.⁴⁶

Addressing psychosocial impact

The psychosocial burden of hirsutism, including emotional distress, anxiety, and reduced quality of life, must not be overlooked. Counseling, social support, and mental health interventions are crucial components of a holistic treatment approach.⁴⁷ Recognizing and addressing the psychological

implications of hirsutism can improve adherence to treatment and overall patient outcomes.⁴⁸

Limitations

This study has several limitations that warrant acknowledgment. Firstly, the literature review focused exclusively on studies from 2018 to 2024, potentially overlooking valuable insights from earlier research. Additionally, non-English studies not included, which may have restricted the diversity and depth of perspectives analyzed. The reliance on the Ferriman-Gallwey scoring system, which is based primarily on data from a predominantly white population, may not fully account for racial and ethnic variations in hair growth patterns, limiting the applicability of findings across diverse groups. While the study recognizes the psychosocial impact of hirsutism, the absence of quantitative measures of psychological distress restricts our understanding of the relationship between physical severity and emotional burden. Furthermore, inconsistencies in diagnostic criteria across the included studies introduce variability, and there is limited focus on patient-reported outcomes, which are crucial for assessing patient satisfaction and treatment effectiveness.

Finally, the study's scope is further constrained by potential reviewer and publication biases, an insufficient examination of cultural differences in the psychosocial impact, and minimal guidance on preventive strategies. Addressing these limitations in future research could pave the way for a more inclusive, culturally sensitive, and holistic approach to understanding and managing hirsutism.

Conclusion

The management of hirsutism requires a personalized and multidisciplinary approach that addresses both physical symptoms and psychosocial impacts. While established therapies such as oral contraceptives and antiandrogens remain central to treatment, novel therapies are emerging, offering potential for improved outcomes with fewer side effects. Further research is needed to refine these strategies and optimize care for affected women.

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Author's contribution

YS,SS,DS,NS,AS,NAA: Have made substantial contributions to the design, data acquisition, analysis, and interpretation of data; participated in drafting, critical review for intellectual content; approved the final version for publication.

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