REVIEW ARTICLE

POST-FINASTERIDE SYNDROME: AN OVERVIEW OF ITS MECHANISMS, SYMPTOMS, AND THERAPEUTIC OPTIONS

Zespół pofinasterydowy: przegląd mechanizmów, objawów i opcji terapeutycznych

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Abstract

Post-finasteride syndrome is a complex condition characterized by psychological, sexual, and somatic symptoms observed in patients currently or previously treated with 5α-reductase inhibitors. Although these agents are commonly used in the management of benign prostatic hyperplasia and androgenetic alopecia, an increasing number of cases report persistent adverse effects even after treatment discontinuation. The lack of clearly defined pathophysiological mechanisms and the limited availability of clinical research complicate the diagnosis and management of post-finasteride syndrome. This paper reviews the current understanding of post-finasteride syndrome, including its potential molecular pathways, clinical presentation, and therapeutic approaches. Strong evidence supports the existence of post-finasteride syndrome, and the European Medicines Agency has recognized suicidal ideation as an adverse effect of finasteride administered at doses used to treat androgenetic alopecia. Various therapies can be administered to alleviate the symptoms of post-finasteride syndrome, and effective communication with patients is essential to ensure that adverse events are recognized and addressed. An interdisciplinary framework should be used for the implementation of treatment with 5α-reductase inhibitors, with patients undergoing thorough physical and mental health assessments prior to treatment initiation.

Streszczenie

Zespół pofinasterydowy jest złożonym stanem klinicznym obejmującym objawy psychiczne, seksualne i somatyczne, obserwowanym u pacjentów aktualnie lub uprzednio leczonych inhibitorami 5α-reduktazy. Choć leki te są powszechnie stosowane w terapii łagodnego rozrostu gruczołu krokowego oraz łysienia androgenowego, w coraz większej liczbie przypadków odnotowuje się utrzymywanie działań niepożądanych także po zakończeniu terapii. Brak jednoznacznie określonych mechanizmów patofizjologicznych oraz ograniczona liczba badań klinicznych utrudniają rozpoznawanie i leczenie tego zespołu. Niniejsza praca przedstawia aktualny stan wiedzy na temat zespołu pofinasterydowego, jego potencjalnych mechanizmów molekularnych, obrazu klinicznego oraz możliwych podejść terapeutycznych. Istnieją silne dowody potwierdzające istnienie tego zespołu, a Europejska Agencja Leków uznała myśli samobójcze za działanie niepożądane finasterydu stosowanego w dawkach terapeutycznych w leczeniu łysienia androgenowego. Różne metody terapeutyczne mogą być stosowane w celu łagodzenia objawów zespołu pofinasterydowego, a kluczową rolę odgrywa komunikacja z pacjentem, aby działania niepożądane mogły być wcześnie rozpoznane i odpowiednio leczone. Wdrożenie leczenia inhibitorami 5α-reduktazy powinno odbywać się w ramach podejścia interdyscyplinarnego, z uwzględnieniem szczegółowej oceny stanu somatycznego i psychicznego pacjenta przed rozpoczęciem terapii.

Keywords: 5α-reductase inhibitors; adverse effects; androgenetic alopecia; benign prostatic hyperplasia; post-finasteride syndrome

Słowa kluczowe: inhibitory 5α-reduktazy; działania niepożądane; łysienie androgenowe; łagodny rozrost gruczołu krokowego; zespół pofinasterydowy

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Introduction

Post-finasteride syndrome (PFS) is the term used to describe the occurrence of various adverse psychological, sexual, and somatic symptoms in patients who are currently using, or who used 5α-reductase inhibitors more than three months earlier, regardless of the therapeutic indication [1]. More than 50% of Caucasian men experience androgenetic alopecia, also known as male pattern hair loss. Over 60% of men older than 60 years are diagnosed with benign prostatic hyperplasia, of whom approximately 40% are symptomatic [2, 3]. Consequently, the number of patients eligible for treatment with 5α-reductase inhibitors is substantial, and drugs from this class are widely used in clinical practice.

Since 1992, the United States Food and Drug Administration has approved finasteride at a dose of 5 mg per day for the treatment of lower urinary tract symptoms associated with benign prostatic hyperplasia. Since 1997, the same active ingredient at a dose of 1 mg per day has been approved for treating androgenetic alopecia. Currently, dutasteride at a dose of 0.5 mg per day is another 5α-reductase inhibitor widely used for the treatment of symptomatic benign prostatic hyperplasia.

Finasteride acts as a weak inhibitor of the type I and a potent inhibitor of the type II isoform of the 5α-reductase enzyme. This enzyme, which is responsible for the conversion of testosterone to 5α-dihydrotestosterone, is expressed, among other tissues, in the prostate gland, seminal vesicles, epididymis, hair follicles, brain, and liver. Dutasteride has a strong inhibitory effect on the type II isoform and also exhibits significant affinity for the type I isoform, which is also present in the skin and muscles. The reduction in 5α-dihydrotestosterone concentrations achieved during therapy with 5α-reductase inhibitors results in decreased androgenic effects on prostate tissue and hair follicles.

Safety of therapy and controversies surrounding PFS

For many years, the use of finasteride and dutasteride for approved indications was considered safe and well tolerated. Recently, however, concerns have arisen regarding the safety of these drugs, partly due to the influence of patient organizations, particularly in the United States, that bring together individuals who have reported sexual, psychological, and somatic disorders during and after treatment with 5α-reductase inhibitors.

This has led to the initiation of research and systematic collection of data on potential adverse effects associated with the use of 5α-reductase inhibitors. To date, only a limited number of scientific studies dedicated to PFS have been published, most of which are based on subjective patient reports. Difficulties in analyzing PFS arise, among other reasons, from the lack of well-established molecular and genetic mechanisms underlying the syndrome, the absence of clear and widely accepted diagnostic criteria, and insufficient awareness among physicians prescribing 5α-reductase inhibitors. These physicians rarely report adverse effects.

In recent years, PFS has been listed as a potential consequence of 5α-reductase inhibitor use in the United States National Institutes of Health's rare disease registry (The Rare Diseases Clinical Research Network, funded by the NIH); however, PFS is still not recognized as a distinct clinical entity.

Use and mechanism of action of 5α-reductase inhibitors

The widespread use of 5α-reductase inhibitors in clinical practice results from the large target population and the proven beneficial effects of these drugs on lower urinary tract symptoms. These effects are due to the ability of the inhibitors to reduce prostate volume by inhibiting the conversion of testosterone to dihydrotestosterone, the key androgen that stimulates stromal and epithelial cell proliferation in the prostate [4, 5]. Lowering 5α-dihydrotestosterone levels leads to a reduction in prostate mass, decreased urethral pressure, and improved urinary flow.

Clinical trials, such as the Proscar Long-Term Efficacy and Safety Study, have demonstrated that treatment with finasteride reduces prostate volume by approximately 20% to 30% after 6 to 12 months of therapy, and significantly decreases the risk of acute urinary retention and the need for surgical intervention in patients with benign prostatic hyperplasia [6]. Moreover, dutasteride, which inhibits both type I and type II 5α-reductase isoforms, produces a greater reduction in 5α-dihydrotestosterone levels in serum and prostate tissue (over 90%) compared with finasteride (approximately 70%) [7].

The mechanism of action of 5α-reductase inhibitors also includes the reduction of inflammation and edema within the prostate, which further contributes to the alleviation of lower urinary tract symptoms. However, the therapeutic effect becomes apparent only after several months of use. This distinguishes 5α-reductase inhibitors from α-adrenolytic agents, which act more rapidly by relaxing the smooth muscles of the bladder neck and prostate.

The effect of 5α-reductase inhibitors on androgenetic alopecia results from their ability to reduce the local concentration of 5α-dihydrotestosterone in the scalp, specifically within the hair follicles, particularly in the frontoparietal region [8]. Excessive 5α-dihydrotestosterone activity leads to the miniaturization of hair follicles, shortening of the anagen phase, and prolongation of the telogen phase. Ultimately, this process causes hair thinning, reduced hair shaft diameter, and progressive hair loss.

Finasteride, a selective type II 5α-reductase inhibitor, reduces 5α-dihydrotestosterone levels in the scalp by approximately 60% and in plasma by approximately 70%. This helps halt follicular miniaturization, improve follicular blood supply and, consequently, nourishment, and prolong the anagen phase. Randomized clinical trials have shown that treatment with finasteride at a dose of 1 mg per day for 12 months increased hair density, halted hair loss in 83% of men, and led to significant aesthetic improvement in 66% of patients [9].

Dutasteride, by inhibiting both type I and II 5α-reductase isoforms, produces an even greater reduction in 5α-dihydrotestosterone levels (more than 90%), which may translate into greater clinical efficacy compared with finasteride. However, its use for androgenetic alopecia is approved in only some countries and remains off-label in the United States and Europe [9].

That 5α-reductase inhibitors are lipophilic and can cross the blood–brain barrier, thereby exerting effects on the central nervous system, is not widely recognized [10]. The enzyme 5α-reductase plays a crucial role in the metabolism of neuroactive steroids within the central nervous system. Three isoforms of this enzyme have been identified in the brain, where they participate in the conversion of testosterone and progesterone to their active metabolites of dihydrotestosterone and dihydroprogesterone, respectively.

These products are further metabolized into neuroactive derivatives such as allopregnanolone, isopregnanolone, 5α-androstan-3α,17β-diol, and 5α-androstan-3β, 17β-diol. These compounds modulate the activity of neurosteroid receptors, including the GABA-A receptor complex, thereby playing an important role in regulating emotional, cognitive, and behavioral processes [11].

As an inhibitor of type II 5α-reductase and, to a lesser extent, type I, finasteride disrupts this metabolic pathway. Such disruption may lead to significant alterations in the concentrations of neuroactive steroids in both plasma and cerebrospinal fluid.

Neurobiological and hormonal mechanisms of PFS

Studies in patients with PFS have demonstrated decreased concentrations of pregnenolone, progesterone, and dihydroprogesterone in the cerebrospinal fluid, accompanied by increased levels of dehydroepiandrosterone and testosterone. Concurrently, reductions in 5α-dihydrotestosterone and 17β-estradiol levels were observed, indicating extensive disturbances of the neurosteroid axis in this group of patients [12].

Experimental studies have also documented changes in the expression of androgen receptors, estrogen receptors, and GABA-A receptor subunits in the brain, which may contribute to the persistence of neuropsychiatric symptoms [12]. These mechanisms may be further modulated by epigenetic phenomena [13, 14]. The frequency of methylation of the *SRD5A2* gene promoter, which encodes type II 5α-reductase, has been shown to be higher in the cerebrospinal fluid of patients with PFS than in that of the control group [13, 14].

Studies using animal models have demonstrated that chronic inhibition of 5α-reductase activates inflammatory mechanisms within the central nervous system, including an increased number of astrocytes and elevated levels of pro-inflammatory cytokines, such as tumor necrosis factor-α and interleukin-1β, in the hippocampus. These changes are accompanied by reduced neurogenesis in the hippocampus: This may contribute to the depressive symptoms and cognitive deficits observed in patients.

Clinical presentation of PFS

At the clinical level, symptoms in patients with PFS can be classified into three main categories: sexual, psychological, and somatic.

The most frequently reported sexual dysfunctions include decreased or absent libido; erectile dysfunction, affecting both the initiation and maintenance of an erection; orgasmic disorders involving both the ability to achieve orgasm and the perception of orgasmic sensations; reduced ejaculate volume; and complete absence of ejaculation [15, 16]. Less common complications include testicular atrophy, prostatitis, subjective perception of penile shortening, and infertility. The literature also describes an unusual symptom – a subjective feeling of incoherence between central nervous system activity and the functioning of genital organs – described by patients as "penis disconnected from the body," and referred to in the English-language literature as *brain–penis disconnect*.

Among the psychological symptoms, affective disorders, including depressive episodes and dysthymia, are predominant. Anxiety disorders and cognitive impairment involving difficulties with comprehension, problem-solving, decision-making, and slowed thought processes – often referred to as *brain fog* – have been reported [17]. Emotional disturbances (such as blunted affect and anhedonia), memory deficits, sleep disorders, as well as suicidal thoughts and self-harming behaviors [18], have also been described.

Somatic manifestations include skin changes (rash), muscle atrophy and weakness, gynecomastia, chronic fatigue, and hearing disturbances (including hearing loss and tinnitus).

Criteria for the diagnosis and diagnostic evaluation of PFS

The diagnostic criteria for PFS were proposed in a consensus statement published in the *International Journal of*

Risk & Safety in Medicine by David Healy and colleagues under the auspices of the European Society for Sexual Medicine.

The primary criterion for the diagnosis of PFS is prior use of a 5α-reductase inhibitor, followed by the onset of sexual dysfunction during therapy with the inhibitor or after its discontinuation. These symptoms must persist continuously for at least 3 months (Fig. 1).

The key sexual symptoms recognized in the criteria and necessary for establishing the diagnosis include decreased libido, erectile dysfunction, reduced genital and orgasmic sensations, and decreased ejaculate volume. An important diagnostic element is the absence of pre-existing sexual dysfunction and the exclusion of other diseases, medications, or factors that could explain the current complaints.

In addition to sexual dysfunction, some patients present with the aforementioned cognitive and neuropsychiatric symptoms, which – although not essential for the diagnosis of PFS – frequently co-occur. These include brain fog, memory and concentration difficulties, slowed thinking, and emotional disturbances such as depression (including suicidal ideation), anxiety, panic attacks, irritability, anhedonia, and insomnia.

Notably, patients using 5α-reductase inhibitors may experience symptoms such as gynecomastia and changes in semen quality or volume; however, these symptoms are not pathognomonic for PFS and are not required for diagnosis.

Risk factors and predisposed groups

Importantly, most data on PFS are based on retrospective observations and patient reports, whereas available clinical studies have significant methodological limitations. The pathophysiological mechanisms underlying PFS remain a matter of debate and, in some cases, the contribution of the nocebo effect to the reported symptoms cannot be entirely ruled out.

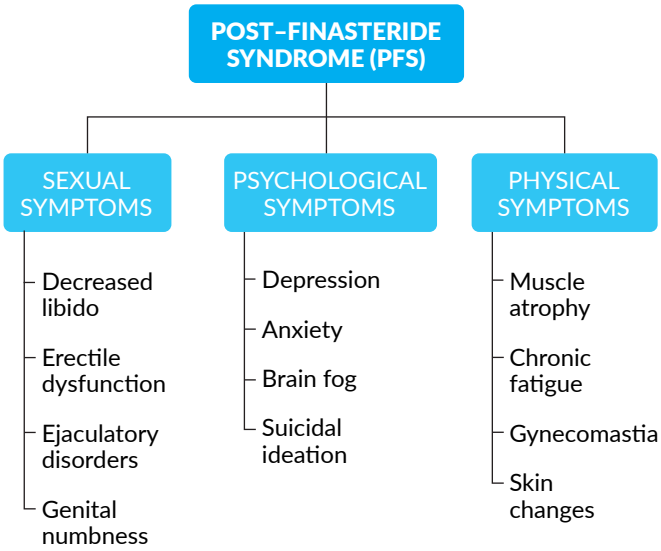

Figure 1. Post-finasteride syndrome – symptoms

Evidence suggests that PFS – although still controversial as a distinct nosological entity – may occur in predisposed individuals. An increasing number of reports indicate that even short-term exposure to 5α-reductase inhibitors can lead to the development of persistent neuropsychiatric disorders in susceptible individuals. Groups at particular risk of developing PFS include men with a history of depression, anxiety disorders, other psychiatric conditions, or sexual dysfunction, who – as shown in studies – are more likely to experience persistent neuropsychiatric and sexual adverse effects after discontinuing finasteride. Additional risk factors include a history of psychiatric disorders in first-degree relatives, a history of infertility or reduced fertility, and long-term use of finasteride. A treatment duration exceeding 7 months has been identified as an independent predictor of persistent symptoms.

Analysis of data from the United States Food and Drug Administration Adverse Event Reporting System indicates that reports of PFS are more frequent among younger men taking the 1 mg dose used for androgenetic alopecia than among older patients taking the 5 mg dose commonly prescribed for benign prostatic hyperplasia [19, 20]. Based on the available evidence, particular caution should also be exercised in patients older than 35 years with risk factors for metabolic syndrome; preliminary screening for insulin resistance is recommended in these patients. Attention should also be paid to potential molecular underpinnings, such as genetic variability (e.g., polymorphisms in the androgen receptor gene) and epigenetic mechanisms (including methylation of the *SRD5A2* gene promoter) [21], which may modulate the body's response to therapy and predispose certain individuals to developing PFS.

Therapeutic options and clinical approach

Given these observations, special caution is advised when considering treatment with 5α-reductase inhibitors in patients belonging to high-risk groups, and alternative therapeutic strategies should be explored. Topical preparations such as minoxidil can be used to treat androgenetic alopecia; this agent acts independently of the testosterone-to-dihydrotestosterone conversion pathway and has a different adverse effect profile. Furthermore, interest in low-level laser therapy devices is growing. This approach may stimulate hair growth through photobiomodulation.

Conclusions

For lower urinary tract symptoms in patients at risk of developing PFS, phosphodiesterase type 5 inhibitors, which can improve sexual function and urinary flow without directly affecting the androgen hormonal axis, may be a treatment option. Drugs from this class, along with α-adrenergic antagonists, may serve as pharmacologic alternatives for symptomatic benign prostatic hyperplasia. If pharmacotherapy is not effective in alleviating symptoms, patients can be offered a wide range of procedural interventions tailored to their individual needs and clinical condition.

Because the pathophysiological mechanisms underlying PFS remain unknown, causal treatment is currently

not possible, and interventions are symptomatic and supportive. Current recommendations emphasize the need for an integrated biopsychosocial approach that considers biological, psychological, and social aspects. Psychological and psychiatric support plays a key role and includes cognitive behavioral therapy, treatment of coexisting depressive or anxiety disorders, with a preference for antidepressants with a minimal impact on sexual function (e.g., bupropion, mirtazapine), and sex therapy incorporating sensate focus techniques and motivational interviewing.

PDE5 inhibitors or intracavernosal injections are used to treat erectile dysfunction, and supportive measures may include supplementation with, for example, vitamin D, vitamin E, L-arginine, or L-carnitine. These supplements may positively influence nitric oxide metabolism and vascular function.

Cilio et al. [22] describe PFS as a relatively rare syndrome, with symptoms that may persist for more than a year after finasteride discontinuation. The authors also discuss several potential factors that may contribute to PFS, including androgen receptor gene polymorphisms and changes in the gut microbiota. In another study, authored by Thaibah and published in 2025 [23], an extensive dataset involving more than 9,400 participants with PFS was analyzed. The results revealed that nearly 18% of participants reported suicidal ideation and 7% had attempted suicide. Based on these findings, the European Medicines Agency formally recognized suicidal ideation as an adverse effect of finasteride at a dose of 1 mg, the dose used to treat androgenetic alopecia.

Cilio et al. [22] discussed PFS and its core side effects, which require a complex diagnostic process. The authors emphasized that the absence of clearly established pathophysiological mechanisms cannot be grounds for denying the existence of the syndrome, especially given the increasing number of consistent clinical observations.

A promising, although still experimental, area of research is the development of therapies targeting neuroactive steroids and modulation of androgen receptor expression. However, none of the available therapeutic regimens guarantees complete remission of symptoms. The goal of treatment is the gradual alleviation of symptoms and improvement of patients' quality of life. Given the possible influence of the nocebo effect, effective communication with the patient is crucial, including ensuring that reported complaints are taken seriously and fostering a trust-based therapeutic relationship. Treatment should be implemented within an interdisciplinary framework involving physicians, psychologists, and sexologists. In every case, the decision to initiate therapy with a 5α-reductase inhibitor should be preceded by a thorough assessment of the patient's physical and mental health and by obtaining informed consent after discussion of the potential benefits and risks.

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