

Reframing the Discussion on Finasteride and Neuropsychiatric Safety:

A Call for Balanced Interpretation

To the Editor: In his recent article, Brezis¹ argues that finasteride is causally linked to depression, suicidality, and other neuropsychiatric reactions. While the emphasis on patient safety is commendable, the conclusions may overstate causality and risk misleading clinicians and patients about the safety profile of this medication.

Disproportionality analyses and spontaneous reporting systems are designed to identify potential safety signals rather than to establish causation or quantify risk. These sources are vulnerable to reporting bias, media amplification, and incomplete denominator information. Signals should prompt controlled epidemiologic studies, not population-level extrapolations.

Depression and suicidality are complex outcomes that require careful control of psychiatric history, psychosocial stressors, and indication bias. Individuals with alopecia experience meaningful psychosocial burden. In androgenetic alopecia, meta-analysis shows significant impairment in quality of life and emotional domains even when pooled depression scores do not meet diagnostic thresholds.² In alopecia areata, a nationwide Korean study documents substantial anxiety and depressive symptoms that correlate with disease factors, underscoring baseline vulnerability within alopecia populations.³ Studies that fail to account for these background risks may misattribute outcomes to finasteride exposure.

When confounding is addressed in large population cohorts, there is no observed increase in completed suicide with 5- α reductase inhibitors. Welk and colleagues

reported no increased suicide risk, although transient increases in self-harm and depression were seen early after initiation, which declined over time.⁴ These findings argue for nuanced patient monitoring rather than categorical attribution of suicidality to finasteride.

Dose and population distinctions are also essential. The 1-mg formulation is indicated for androgenetic alopecia in generally younger, healthier men, whereas the 5-mg formulation is used for benign prostatic hyperplasia in older men with different comorbidity profiles. The current US label for finasteride 1 mg lists depression and suicidal ideation and behavior among reported psychiatric adverse events, which supports counseling and vigilance while not proving causality or frequency.⁵ Conflating outcomes across doses and populations can distort risk estimates for hair loss patients.

Fear-based messaging surrounding finasteride may have unintended consequences that extend beyond pharmacovigilance. Individuals from lower socioeconomic backgrounds, who often lack access to specialist dermatologic care or emerging alternatives such as low-dose oral minoxidil or procedural interventions, may avoid treatment altogether because of exaggerated safety concerns. For these patients, progressive hair loss can worsen self-image, social withdrawal, and depressive symptoms, thereby increasing vulnerability to the very mood disturbances the article seeks to prevent. Public communication on drug safety should therefore remain proportionate, evidence-based, and sensitive to disparities in treatment access and psychological burden.

Balanced interpretation of pharmacovigilance data is vital to protect patients without discouraging evidence-based care. Overstated harms may cause patients to avoid effective therapy, which could worsen alopecia-related distress and potentially increase mood symptoms in vulnerable groups. A constructive path forward is rigorous, prospective research that stratifies by dose, indication, age, baseline psychiatric risk, and temporal patterns around treatment initiation.

References

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