Characteristics of Men Who Report Persistent Sexual Symptoms After Finasteride Use for Hair Loss

John Santmann
Post-Finasteride Syndrome Foundation, Somerset, New Jersey 08873

Basaria et al. (1) attempted to identify the underlying neurobiological abnormalities in brain circuitry implicated in sexual arousal and depression in former finasteride users who developed persistent sexual dysfunction and depression. To fully understand and correctly interpret the implications of this study, additional information and clarification are necessary.

From limited observations that androgen-regulated messenger RNA transcripts in skin biopsy specimens did not differ among the groups, the authors concluded that persistent sexual symptoms cannot be attributed to persistent off-target effects of finasteride. The following observations preclude drawing these conclusions: (1) Upper back skin biopsy samples were taken only from a few subjects in each study group; (2) finasteride is a specific inhibitor of 5α-reductase, type 2 (2); and (3) nongenital skin expresses low levels of 5α-reductase, type 2 (3, 4). Epigenetic changes due to finasteride use cannot be detected by messenger RNA transcript measurements and might possibly have contributed to the persistent sexual dysfunction.

From the granular and limited functional magnetic resonance imaging analyses, the authors reported they had identified abnormalities in the brain regions of symptomatic former finasteride users similar to those identified in patients with psychogenic erectile dysfunction (ED) (5, 6). Although some similarities existed, important and distinct differences were also present. Substantial correlations between blood oxygenation level-dependent findings and International Index of Erectile Function scores during exposure to erotic images in the bilateral thalamus, right precentral gyrus, left caudate, and left putamen (1) were not observed in any of the cited psychogenic ED studies (5, 6). In contrast, psychogenic ED studies have found important differences in the medial prefrontal cortex, precuneus, right middle insular cortex, and claustrum (5, 6) that were not observed in the functional magnetic resonance imaging results of Basaria et al. (1).

These psychogenic ED studies must be interpreted with great caution because psychogenic ED was diagnosed using RigiScan (Timm Obson Medical Technologies, Washington, PA) monitoring of nocturnal penile tumescence (NPT) (5, 6). However, RigiScan (Timm Obson Medical Technologies) monitoring is unreliable at diagnosing psychogenic ED (7). Using NPT and the Minnesota Multiphasic Personality Inventory to diagnose organic versus psychogenic ED yields a 63% rate of misclassification (7). NPT cannot effectively distinguish vasculogenic from psychogenic ED (8). Using NPT, 73.1% of patients with psychogenic ED diagnosed had endothelial dysfunction and 39.8% of patients with organic ED diagnosed had normal endothelial function (8).

Although a causal relationship between previous finasteride use and persistent sexual symptoms cannot be inferred from that study and the depressed mood might have contributed to the persistent sexual symptoms, the absence of a history of sexual dysfunction or depression before finasteride use and the temporal appearance of these symptoms only after finasteride use suggests a possible causal relationship that will likely only be demonstrated in a well-designed epidemiological study. Patients with depression have risk of developing sexual dysfunction that is lower (50% to 70%) than the risk that patients with sexual dysfunction have of developing depression (130% to 210%) (9). It is inappropriate for the authors to conclude that “symptomatic
finasteride users revealed depressed mood and fMRI [functional magnetic resonance imaging] findings consistent with those observed in depression” without mentioning the abnormal function in brain circuitry linked to sexual arousal.

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References


