

Commentary

Topical Clascoterone: A Potential Treatment for a Hidradenitis Suppurativa Subgroup

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This commentary is a follow-up to the article “Use of topical clascoterone for the treatment of hidradenitis suppurativa [1].” In this article, we described the successful treatment of a 23-year-old female with Hurley stage 1 Hidradenitis Suppurativa (HS) with topical clascoterone 1% cream [1]. With a waxing and waning course, HS is a chronic and debilitating inflammatory skin disease that often requires multiple treatments [2]. Inadequately treated painful nodules and abscesses in intertriginous areas can subsequently develop into fistulae, draining sinus tracts, and scars [2,3]. Thus, while many clinical trials are focused on the population of patients with moderate to severe disease, treatment of HS early in the disease course is of paramount importance, to improve both the characteristic HS lesions and the quality of life of affected patients [2].

HS is a heterogeneous disease with multiple clinical phenotypes that often have varied responses to standard therapies [3,4]. Use of the FDA approved biologic adalimumab, and off-label treatment with topical and systemic antibiotics, systemic hormone modulating therapies like spironolactone and metformin, immunosuppressants, and surgery, does not result in adequate disease control for many patients [2,5,6]. Many studies have suggested different clinical HS phenotypes, taking into account patient age, gender, and relevant comorbidities [4]. These phenotypes have included typical and atypical subtypes [7], axillary-mammary, follicular, and gluteal subtypes [8], axillary-mammary-groin, axillary-gluteal-groin, and axillary-groin subtypes [9], and regular, frictional furuncle, scarring folliculitis, conglobata, syndromic, or ectopic subtypes [10]. Many of these phenotypes

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have overlapping features, however, and none have been validated to be reliable [4]. The existence of an accurate phenotype/phenotype-genotype classification schema based on the results of future studies will be extremely helpful in the management of HS, providing the ability to predict disease progression and treatment responses [4]. This will ultimately improve patient outcomes and the quality of life of affected patients, including those with a hormonal component to their disease process.

Although the role of hormones in the pathogenesis of HS is still unclear, there is evidence suggesting their involvement. Hormone association is suggested by patients who experience perimenstrual disease flares, worsening of disease after pregnancy, peri-pubertal onset, and the occurrence of characteristic lesions in areas high in apocrine glands [11-13]. Additionally, insulin resistance and metabolic syndrome are common comorbidities in HS [12]. The involvement of hormones is further supported by the successful use of anti-androgen therapies such as spironolactone and finasteride, particularly in women, and the oral anti-diabetic drug metformin, which enhances the effect of insulin, in HS patients [12,13]. Although clinical phenotypes are not agreed upon, there may be a phenotype that is more responsive to anti-hormonal therapy.

The pathogenesis of HS is multifactorial, but there is agreement that plugging and ultimately rupture of the follicle within the pilosebaceous apocrine unit drives inflammation in HS [2,3]. Despite studies showing non-elevated levels of systemic testosterone in patients with HS [13,14], one study has reported an increase in androgen receptor (AR) expression in the epidermis, infundibulum, and skin tunnels in HS skin compared to normal controls [15]. Interestingly, AR expression in skin tunnels was continuous in males, yet segmental in females [15]. It has also been reported that increased activity of androgens within infrainfundibular keratinocytes may promote hyperkeratinization and occlusion in acne [16]. This suggests that perhaps the anti-androgen responsiveness of patients with HS may be related to the distribution of AR in HS skin [15]. In particular, localized androgens may be responsible for the initial follicular occlusion in HS, in the terminal conversion of testosterone to dihydrotestosterone (DHT) [14,15].

This provides support for the use of topical clascoterone, which targets DHT at the application site [17]. Since the spectrum of HS consists of inflammatory nodules and abscesses that progress to draining sinus tracts and scars with advanced disease [2], topical clascoterone may have particular success early in the disease course prior to the development of scarring, by targeting an initial step in disease pathogenesis [14]. Additionally, because of the mild adverse event profile and lack of systemic hormonal side effects [17], this therapy may also be particularly useful for men who traditionally are not recommended systemic anti-androgen therapies such as spironolactone, due to adverse events [12]. Thus, topical clascoterone is a promising treatment for a subgroup of HS patients and should be evaluated in future randomized controlled trials.

Conflicts of Interest

Kaitlyne N. Cunningham declares no conflicts of interest. Gabriela A. Cobos has received honoraria as a speaker for Sanofi, Regeneron, and Pfizer; as a consultant for Sanofi, Regeneron, UCB, and Janssen; and as a trial adjudicator for Biogen. David Rosmarin has received honoraria as a consultant for AbbVie, Abcuro, AltruBio, Arena, Boehringer-Ingelheim, Bristol Myers Squibb, Celgene, Concert, CSL Behring, Dermavant, Dermira, Incyte, Janssen, Kyowa Kirin, Lilly, Novartis, Pfizer, Recludix, Regeneron, Revolo Biotherapeutics, Sanofi, Sun Pharmaceuticals, UCB, VielaBio; has received research support from AbbVie, Amgen, Bristol Myers Squibb, Celgene, Dermira, Galderma, Incyte, Janssen, Lilly, Merck, Novartis, Pfizer, and Regeneron Pharmaceuticals Inc; and has served as a paid speaker for AbbVie, Amgen, Bristol Myers Squibb, Celgene, Dermavant, Incyte, Janssen, Lilly, Novartis, Pfizer, Regeneron Pharmaceuticals Inc., and Sanofi.

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