Medical therapy is the optimal treatment for hidradenitis suppurativa



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This article is part of a point-counterpoint series of controversies.

ur ultimate treatment goal in hidradenitis suppurativa (HS) is to eliminate disease activity, which will prevent progression of disease. Although our understanding of the pathophysiology of HS is not complete, we know that inflammation in the skin drives the development of scarring and sinus tracts. Therefore, medical therapies that suppress inflammation have the potential to prevent the development of these lesions and obviate the need for surgery. Indeed, the measure of success for therapies in HS is a reduction in the number of existing lesions and prevention of new lesion development.² The only surgical therapy capable of preventing the development of new lesions is wide local excision, which removes large amounts of tissue from apocrine gland-bearing areas of the body. Owing to the risk of complications and postoperative recurrence, wide local excision does not have a role as a prophylactic therapy and is reserved for advanced-stage disease with extensive involvement.³ Additionally, medical therapy is recommended before, during, and after surgery for HS with proven benefit in improving surgical outcomes and prevention of disease progression.⁴ Even after scarring and sinus tracts have developed, medical therapies can eliminate inflammation, pain, and drainage, which results in asymptomatic scars and sinus tracts. The goal with surgery in HS is to remove lesions, replacing them with asymptomatic scars. Therefore, medical therapy can achieve a comparable outcome to surgery, although we cannot consistently provide this outcome to the majority of patients with our current medical options.

Abbreviation used:

HS: hidradenitis suppurativa

The reason that surgery still has a role in the treatment is because we are not yet able to provide medical therapy with optimal results. To prevent progression of disease with treatment, we must first increase disease awareness and recognition. There is currently an average delay between disease onset and diagnosis of longer than 7 years.⁵ During this time, patients progress from intermittent nodules and abscesses to develop persistent scars and sinus tracts, many of which will require surgical intervention. The elimination of diagnostic delay is paramount to optimizing the efficacy of medical therapies, particularly with respect to prevention of progression. Diagnostic delay is not the sole hurdle for medical treatment to dominate HS therapy. Our medical treatment of HS is still in its infancy. Although it holds great potential to change the course of disease, we do not yet have the tools to bring that outcome to fruition. As we develop a better understanding of the inflammatory pathways and cytokines involved in HS, we will become able to develop treatment options capable of providing disease control for patients across the full spectrum of disease severity and phenotypes. This scenario is similar to psoriasis, where it is only recently that the development of a wide treatment landscape with numerous very effective options has allowed us to make our treatment goal 1% of body surface area involvement or less. I am confident that, in time, medical therapy will provide us with the ability to control HS without surgical intervention.

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