Biologic Therapies in the Treatment of Hidradenitis Suppurativa

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Hidradenitis suppurativa (HS) is a chronic, recurring disease of the skin often characterized by painful inflammatory lesions in apocrine gland–rich intertriginous regions of the body. It is a frustrating disease for both the patient and clinician because HS is often refractory to multiple medical and surgical treatment regimens. Recently, biologic therapies have emerged as a new treatment option for HS. This article presents a literature review of the use of biologic agents, particularly anti–tumor necrosis factor-α agents in the treatment of recurrent hidradenitis suppurativa.

Hidradenitis suppurativa (HS) is a chronic, debilitating inflammatory disease involving the intertriginous areas of the body where apocrine glands are plentiful. Although the pathogenesis of this cutaneous disorder is still largely unknown, current theories suggest that the initial event is follicular hyperkeratosis and occlusion, with subsequent rupture of follicles initiating an inflammatory response.[1] Local inflammation recruits neutrophils to the area, followed by granulomatous infiltration with multinucleated foreign body giant cells.[1,2,3] Apocrine gland involvement is thought of as a secondary event, resulting only after the rupture of the dilated hair follicles and subsequent spread of inflammation to surrounding tissue.[3] Recurrent inflammation may lead to chronic scarring, fibrosis, and malodorous drainage.

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Evidenced by published data that illustrate its detrimental impact on quality of life, HS is a distressing dermatologic disease with high morbidity.[4,5] Distressing components of the disease were reported by patients in a study by von der Werth and Jemec.[5] These distressing components include soreness, pain, self-consciousness, embarrassment, and limitations in choice of clothing because of foul discharge and abscesses. Thus, there is a need for further investigations into effective alternative treatment options that aim to alleviate this painful, discomforting disease. Existing treatment options have been shown to be of limited value. These include general measures (eg, weight loss, smoking cessation, loose clothing); medical treatments (eg, antibiotics, hormone therapy, retinoids, corticosteroids); radiotherapy; CO_2 laser treatments; and surgical management.[6] This review of the literature will focus on the newer role of biologic agents that have been shown to be promising in the treatment of HS.

OVERVIEW OF BIOLOGIC THERAPIES

The initial experimental use of biologics in the treatment of HS stems from earlier case reports of patients with perianal and genital involvement of HS who had concurrent perianal Crohn disease (CD). Ostlere et al[7] reported clinical similarities between HS and CD. In fact, a positive association between the 2 diseases has been established.[7-15] Therefore, biologic treatments effective in treating CD were also thought to possibly benefit patients with concurrent

HS. Alternatively, HS has recently been included in a group of autoinflammatory disorders characterized by recurrent inflammation without the presence of autoantibodies or antigen-specific T cells, a preponderance of polymorphonuclear cells, and a positive response to anti–tumor necrosis factor (TNF)-α agents.[16] Thus, it is suggested that inhibitors of TNF-α are effective in treating HS because of its similarities with other autoinflammatory disorders.[17]

Immunomodulators currently used in the treatment of HS target TNF-α, a proinflammatory cytokine that can be produced by T cells, keratinocytes, and Langerhans cells.[18] These cytokines are involved in the recruitment of neutrophils during an acute inflammatory response. Neutrophils are responsible for mediating a respiratory burst that generates oxygen radicals and nitric oxide with the release of stored granular contents. These events contribute to host defense, but can also add to local destruction of tissue in sites of pyogenic bacterial infection.[19] In addition, TNF-α promotes the synthesis of other proinflammatory cytokines and increases the expression of intercellular adhesion molecule-1, E-selectin, and vascular cell adhesion molecule-1.[20,21] Thus, the inhibition of TNF-α has been shown to be effective in the treatment of chronic inflammatory skin disorders. Recent biologic therapies have shown promise in the treatment of HS. These include infliximab, etanercept, adalimumab, and efalizumab (Table 1).

INFLIXIMAB

Infliximab is a chimeric IgG1-κ monoclonal antibody against TNF-α. It is currently approved by the US Food and Drug Administration (FDA) for the treatment of rheumatoid arthritis, CD, ankylosing spondylitis, psoriatic arthritis, plaque psoriasis, and ulcerative colitis. Infliximab prevents the binding of TNF-α to its receptor by binding with high affinity to both soluble and transmembrane forms of TNF-α.[28,29] According to the treatment protocol for CD, the conventional dosing regimen for infliximab, administered intravenously, is 5 mg/kg in weeks 0, 2, and 6, followed by 5 mg/kg every 8 weeks thereafter, until signs and symptoms abate.[23]

The first published case of successful infliximab use in the treatment of HS was reported in 2001 by Martinez et al.[14] The subject of the case report was a 30-year-old woman with a history of CD who developed HS that was resistant to treatment with antibiotics (metronidazole and ciprofloxacin) and surgical therapy. This patient had immediate resolution of perianal and axillary lesions after the first dose of infliximab. She also received 2.5 mg/kg per day of azathioprine simultaneously. The third dose of infliximab was never given because of an adverse reaction (erythematous eruption and dyspnea) following the second infusion. A follow-up at 6 months showed continued remission with azathioprine maintenance. Although the patient benefited from the infliximab, the simultaneous use of azathioprine made the efficacy of infliximab as a sole agent unclear.

This case study launched 5 additional case reports between 2001 and 2005.[14,15,17,30-32] These studies included up to 6 months of follow-up and yielded moderate to significant improvement. Each study involved single case reports, with the exception of Sullivan et al,[30] who reported on 5 patients.

In 2006, Thielen et al[33] published the first case report of effective long-term treatment of HS with infliximab. The report described a 48-year-old male with HS involving inguinal, scrotal, and perineal areas who was refractory to antibiotic therapy. Associated CD was ruled out by colonoscopy. He received 3 standard doses of infliximab at

Tumor Necrosis Factor-α Inhibitors

Generic Name	Trade Name	Standard Dosing Regimen for HS	Common Adverse Effects[22]
Infliximab	Enbrel	5 mg/kg (IV) at wk 0, 2, and 6; every 8 wk thereafter 5 mg/kg[23,a]	Injection site reactions, fevers, chills, nausea, headaches, URI symptoms, dizziness, rashes
Etanercept	Remicade	50 mg (SQ) twice wk 72–96 hr apart[24,a]	Injection site reactions, headaches, URI symptoms
Adalimumab	Humira	40 mg (SQ) every other wk[25-27,a]	Injection site reactions, fevers, chills, nausea, headaches, URI symptoms, dizziness, rashes

Abbreviations: HS, hidradenitis suppurativa; IV, intravenous; SQ, subcutaneously; URI, upper respiratory infection.
[a]It is recommended that the patient's purified protein derivative skin test for tuberculosis and chest x-ray be checked prior to use.

5 mg/kg per dose, followed by maintenance infusion every 8 weeks, for a total of 104 weeks and 13 infusions. The maintenance therapy also included 7.5 mg of methotrexate weekly to decrease the risk of developing human antichimeric antibodies. The patient had an 80% improvement in the Dermatology Life Quality Index (DLQI) score in the second year as compared with the score at his first visit. The DLQI is a questionnaire developed by Finlay and Khan that aids the clinician in assessing the extent of impact that a particular skin disorder has on a patient's life. The DLQI assesses disability in the following 6 categories: symptoms and feelings, daily activities, leisure, work and school, personal relationships, and treatment.

Long-term treatment efficacy was revisited in a 2008 publication by Mekkes and Bos.[34] Between 2004 and 2005, eleven patients qualified for the study by having severe HS for at least 2 years, in addition to being refractory to standard treatment regimens. Obese patients with a body mass index greater than 27, or who weighed more than 100 kg were excluded. Ten patients completed the study, and they were given a single course of 5 mg/kg of infliximab at weeks 0, 2, and 6. The progress of these patients was followed for at least one year. Severe HS was defined as patients having more than 5 pus-producing lesions, as well as an acne severity score greater than 100. The acne severity score described by Sartorius et al[35] (Sartorius score) involved counting the number of regions affected by HS, as well as scoring the various types of lesions in each anatomic region. The mean (SD) Sartorius score was 164 (50) before treatment, and 108 (38) one month after the third infusion ($P<.001$). One year after treatment, the mean (SD) Sartorius score was 89 (49) ($P=.002$). Six patients saw improvement, with a reduction in the inflammatory component of HS, and 4 patients had a recurrence of the disease. After 2 years, 3 patients continued to maintain their remission status.

One of the first larger clinical trials involving 7 patients with HS occurred in 2007 and had mixed results. Fardet et al[36] found that 3 of the 7 patients experienced severe adverse effects, including severe abdominal pain from colon cancer, bronchospasm with urticaria, and a grade 3 multifocal motor neuropathy with conduction block without any history of prior neurologic disease. Although 5 patients experienced moderate improvement as evidenced by a 40% decrease in the Sartorius score, these benefits were short lived. At the end of 10 weeks, only 2 patients were found to be still responding.

All studies mentioned thus far involved the conventional dosage regimen of 5 mg/kg of infliximab at 0, 2, and 6 weeks, with 5 mg/kg every 8 weeks thereafter. Fernandez-Vozmediano and Armario-Hita[37] reported on a 6-patient clinical trial, with the initial dose of 5 mg/kg at week 0 increased by 0.5 mg/kg on each subsequent treatment (up to a maximum dose of 10 mg/kg), followed by a maintenance dose every 4 weeks. The 6 patients did not have CD and were administered infliximab as a monotherapy. All 6 patients reported positive subjective results, including decreased pain, itching, and exudation, with overall improvement following the initial dose. At the 6-month follow-up, 3 patients required a combined therapy of infliximab with prednisone (1 mg/kg per day), or with prednisone (1 mg/kg per day) in conjunction with cyclosporine (5 mg/kg per day), after a decline in treatment efficacy.

ETANERCEPT

Etanercept is a dimeric competitive inhibitor at the receptor site that binds to both soluble and membrane-bound forms of TNF-α.[38] In addition, it also has the capability of binding to TNF-β.[38] Unlike infliximab, which is given intravenously, etanercept is available as a subcutaneous (SQ) injection, allowing for the convenience of self-administration at home. It is FDA approved for the treatment of psoriasis (plaque type), psoriatic arthritis, adult and juvenile rheumatoid arthritis, osteoarthritis, and ankylosing spondylitis.[22,38]

The first study on the effectiveness of etanercept for the treatment of HS was reported by Jurgensmayer and Fleischer[39] in 2004. The authors presented a case report of a patient suffering from severe recurrent HS refractory to multiple treatment modalities, including numerous antibiotics (both topical and systemic), dapsone, isotretinoin, and surgical intervention. Etanercept was administered at 25 mg SQ twice a week for 2 months. Dosing frequency was then increased to 25 mg SQ 3 times a week. After 3 months of treatment, the etanercept dose was again increased to 50 mg SQ twice a week. This protocol resulted in a 50% reduction of new lesions in this patient.

An encouraging study involving 6 patients with severe, recalcitrant HS treated with etanercept was reported, with positive results by all subjects.[40] Patients had a significant decrease in self-reported disease activity, along with reductions in both DLQI scores and disease severity. Follow-up evaluation using DLQI questionnaires continued for 24 weeks. Patients were administered 25 mg of etanercept SQ twice a week, with 2 patients increasing their dosage to 50 mg SQ twice a week after 2 months. On physical examination, patients had decreased sinus drainage, tenderness of lesions, and induration. All subjects stated that etanercept was the most beneficial treatment to date compared with previous therapies, which included oral antibiotics, dapsone, isotretinoin, tacrolimus, rifampicin, oral antiandrogenic contraceptives, and radical surgery.

More recently, an open-label phase II study on the efficacy of etanercept for the treatment of HS was conducted

between September 2005 and November 2006.[41] Once a week for 12 weeks, 50 mg of entanercept was administered to 10 patients. Sartorius score and the Visual Analogue Scale (VAS) were used to assess efficacy during a follow-up evaluation that continued up to 24 weeks, and VAS scores were determined by asking patients their impression of the severity of their disease, with 0 indicating no disease activity and 10 indicating very severe disease activity. After 12 weeks, 7 patients reported a statistically significant decrease (P=.024) in their VAS scores; after 24 weeks, 6 patients had reduced VAS scores (P=.042) as compared with their baseline scores. All patients experienced considerable benefits, with decreased pain symptoms after 4 weeks of treatment. However, within 4 to 8 weeks after the last dose of etanercept, 8 patients had recurring pus drainage from involved lesions.

ADALIMUMAB

Positive responses to infliximab have prompted research into the effectiveness of adalimumab for the treatment of HS. Adalimumab is a fully human monoclonal antibody against TNF-α. Its mechanism of action is similar to that of infliximab; therefore, it has the ability to lyse cells expressing TNF-α.[38] Like etanercept, it can be self-administered at home SQ. It is FDA approved for the treatment of rheumatoid arthritis, polyarticular juvenile idiopathic arthritis, psoriatic arthritis, ankylosing spondylitis, and CD.

In 2005, a case study was published of a 54-year-old man with long-standing, severe HS who experienced initial improvement with infliximab infusions after the disease was refractory to multiple drug regimens.[42] The patient had recurrences after 9 infusions of infliximab, which were attributed to antibody-induced resistance to the drug. Subsequently, the patient was switched to adalimumab because antibody formation to that drug was deemed less likely.

This treatment regimen and case scenario were echoed in another case study published in 2007.[25] This study described a 44-year-old female with a 5-year history of HS involving the anogenital region. She was previously treated with prednisone, cyclosporine, and surgical debridement. Infliximab infusions were initiated, with standard dosing of 5 mg/kg at weeks 0, 2, and 6, and 5 mg/kg every 8 weeks thereafter, combined with 10 mg of methotrexate weekly because of the severity of the disease and failed prior treatments. This regimen was successful for 7 months until it became necessary to increase the dosage in order to control the disease. In place of the infliximab/methotrexate combination, 40 mg of adalimumab every other week was then introduced. Sinus drainage was reduced, and methotrexate was then discontinued. Adalimumab was increased to 40 mg weekly for maintenance, along with intralesional steroids and occasional levofloxacin for flare-ups.

Two other case studies illustrated the effectiveness of adalimumab treatment in HS. In the first study, a 41-year-old male with a history of arthritis, HS, and cystic acne began treatment with adalimumab after failed trials of hydroxychloroquine, isotretinoin, and methotrexate.[26] Adalimumab 40 mg SQ every other week was initiated, and the patient experienced improvement of axillary symptoms and an increased ability to ambulate. However, the patient relapsed after the third month, and dosing frequency was subsequently increased to 40 mg weekly.

In the second study, the patient was a 67-year-old male with a 20-year history of severe, persistent HS involving the ears, axillae, buttocks, and groin areas.[27] He also had a history of inflammatory bowel disease postcolectomy. Adalimumab was initiated at 40 mg SQ every other week. After the first injection, the patient had reduced drainage from all lesion sites. After 4 months of treatment, the disease remained under control.

EFALIZUMAB

Efalizumab is a monoclonal antibody that binds to CD11 (a component of LFA1) and inhibits the binding of T cells to ICAM1 on antigen-presenting cells.[38] There have been 2 studies, with mixed results, published about efalizumab for the treatment of HS. The first case report is of a 46-year-old male with a 20-year history of HS involving the groin and perianal regions.[43] Previous failed treatments include surgical intervention, antibiotics, and isotretinoin. Efalizumab was initiated at 1 mg/kg per week. After one month of treatment, the patient had fewer inflammatory nodules and a decrease in pain. Control of inflammation was maintained at the 6-month follow-up.

The second case study reported less promising results.[44] This case study involved 5 patients scheduled to receive 0.7 mg/kg per week of efalizumab for the first 2 doses, then 1 mg/kg per week for 10 subsequent doses. Only 2 patients completed the full treatment regimen schedule. Others were lost to follow-up or withdrew for various reasons, including worsening of symptoms. Both patients who completed the full 12 weeks of treatment experienced no improvement in their symptoms, and one patient reported severe, intractable headaches.

SUMMARY

Hidradenitis suppurativa is a distressing cutaneous disorder that has been shown to be refractory to a multitude of drug and surgical treatments. Because of the debilitating nature of the disease, clinical trials continue to search for alternative treatment options that may bring us closer to understanding the pathogenesis of this disease. Based on prior studies, it is clear that biologics may be a viable

treatment option for patients with severe HS who have exhausted other treatment modalities. However, the small number of patients described in the case reports mentioned previously limits broad conclusions about the efficacy and longevity of biologic agents for the general population suffering from HS. Therefore, current investigations into the use of biologic agents, particularly anti–TNF-α agents, are still ongoing. The first double-blind, placebo-controlled trial with infliximab for patients with moderate to severe HS is underway.[45]

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