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Hidradenitis Suppurativa: Recognition and Treatment

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About the Expert

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This publication is intended as an educational resource for healthcare professionals, particularly those who may encounter hidradenitis suppurativa/acne inversa (HS) in an acute setting. It aims to assist with recognition of the disease, and reviews the available treatments, both pharmacological and surgical. Particular emphasis is given to adalimumab, the only pharmacological treatment with registered approval for HS, both in New Zealand and worldwide.

Introduction

HS is a painful, chronic inflammatory skin disease characterised by multifocal, recurrent nodules, abscesses and fistulae, predominantly affecting the axillary, inguinal, breast-fold and anogenital regions.[1] HS ranges from localised lesions (mild disease) to multiple areas of widely dispersed lesions, including interconnected sinus tracts and hypertrophic scars (severe disease).[2] The disease has profound physical and psychological consequences that affect quality of life.[3] Early disease-modifying intervention is often hampered by poor recognition of the disease, with an average time interval of 7.2 years from symptom onset to diagnosis.[4] A range of treatments are currently used in HS, but high-quality evidence is lacking for many of them.[5-9]

The anti-TNF-α monoclonal antibody adalimumab is the only pharmacological treatment with registered approval for the treatment of HS, both in New Zealand and worldwide.[10,11] In October 2019, adalimumab was approved for reimbursement by PHARMAC for patients with moderate to severe disease and an inadequate response to antibiotics, when prescribed by a dermatologist.[12]

Pathophysiology, risk factors and comorbid conditions

The central pathogenic event in HS is believed to be follicular occlusion. Infundibular hyperkeratosis of the terminal follicles and hyperplasia of the follicular epithelium result in the collection of debris, cyst formation, rupture, sinus tract formation and ultimately, scarring.[13] Disruption of the hair follicle produces an inflammatory response,[13] and increased levels of interleukin-1β, tumour necrosis factor-α and interleukin-10 have been found in HS lesions.[14] Factors contributing to this inflammation include the patient's genotype and smoking status, obesity, adipokine dysregulation, insulin or glucose dysregulation, the microbiome and environmental factors:[15]

- A positive family history is found in approximately 33% of patients, and an autosomal dominant inheritance pattern has been suggested[16]
- Smoking prevalence has been estimated at 42-90% and is associated with increased disease severity.[17-19] Nicotine may lead to follicular plugging, or contribute to inflammation via induction of neutrophil chemotaxis[20]
- Obesity is present in 60-88% of patients.[19-22] Obesity is associated with increased disease severity, likely as a result of effects on the skin and microbiome, as well as mechanical friction.[15,23,24] Weight loss can lead to clinical improvement[23]
- Metabolic syndrome prevalence is increased compared with the general population, likely due to the proinflammatory state.[15] Vekic et al. report high rates of dyslipidaemia (44%), insulin resistance (42%), diabetes (17%) and hypertension (16%) in patients with HS treated at the Liverpool Dermatology Clinic in New South Wales, Australia[15]
- Polycystic ovary syndrome prevalence in female patients is also increased compared with the general population (30% at the Liverpool Dermatology Clinic), and suggests an association with the endocrine system.[15]

HS is also clearly associated with autoinflammatory diseases such as Crohn disease and spondyloarthropathies, raising the possibility of a shared pathogenesis.[15,25] Follicular occlusion diseases such as nodulocystic acne, pilonidal sinus and keratosis pilaris are also closely associated with HS.[15,25]

Epidemiology

Estimates of global prevalence of HS range from 1-4%, similar to the prevalence of psoriasis, meaning HS cannot be considered a rare disease.[26,27] While there are no specific prevalence data for New Zealand, prevalence of HS in Australia has been estimated at 0.67%.[28] Females are three times more likely to develop the disease than males.[26,27]

Onset of HS most commonly occurs in patients aged in their early 20s, and is typically active during the third and fourth decades of life.[26,27] However, the disease can occur at any age, including prepubertal children.[5,29] Disease onset before the age of 13 years has been reported in 7.7% of patients with HS, and is associated with stronger genetic susceptibility and more widespread disease.[5,30]

Consequences of disease

Pain is reported by almost all patients with HS, and is the most significant factor contributing to impaired quality of life.[3,26] Quality of life in patients with HS has been reported to be lower than that of patients with other burdensome skin diseases such as psoriasis and atopic dermatitis.[31,32]

Depression is significantly associated with HS, with a prevalence of up to 39% reported in cross-sectional studies.[32-36] Patients with HS are also at increased risk of anxiety, social isolation, poverty, family deterioration and suicide.[3,37,38] A European multicentre, cross-sectional study reported impairment of sex life in 67% of patients with HS.[39]

Patients with HS have a higher unemployment rate than the general population.[38,40] Among employed patients, approximately half have taken sick days because of HS, on average 14-33.6 days per year.[33,35] A large UK study of Hospital Episode Statistics data found a high burden of hospital attendances for patients with HS, who were predominantly of working age.[41] In a large US study of MarketScan medical claims, rates of hospitalisation and emergency department use were higher in patients with HS compared to those with psoriasis.[42]

EXPERT COMMENTARY

The New Zealand experience of HS mirrors the published reports from elsewhere. It is one of the most distressing skin conditions seen in the skin clinic, resulting in high patient Dermatology Life Quality index (DLQI) scores, psychological distress, and unemployment.

Diagnosis

HS is often not recognised and/or may be misdiagnosed by healthcare professionals.[4,43] Patients may present to a variety of healthcare providers and be subjected to repeat and unnecessary investigations and procedures.[43] The diagnosis of HS should therefore be made by a dermatologist or other healthcare professional with expert knowledge of the disease.[5]

Diagnosis requires 3 criteria to be fulfilled:

- Typical lesions (painful nodules, sinus tracts, abscesses and/or scarring) are present
- Typical distribution of lesions (axillae, groins, perineal and perianal regions, buttocks, infra-mammary and inter-mammary folds)
- Chronicity and recurrence of lesions (≥2 episodes in a 6-month period).[5,44]

Examples of typical HS lesions in the axillar, breast and buttocks are shown in **Figures 1a, 1b** and **1c**.

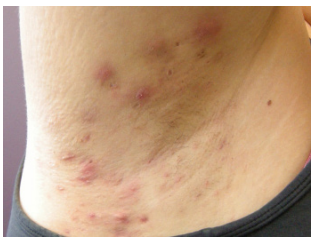

Figure 1a. Axillary HS lesions (reproduced with permission from DermNet New Zealand: hs-axilla-041.jpg).

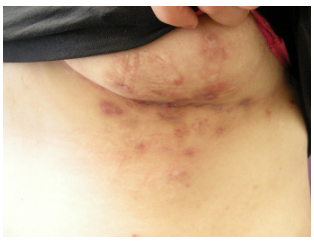

Figure 1b. Breast HS lesions (reproduced with permission from DermNet New Zealand: hs-breast-077.jpg).

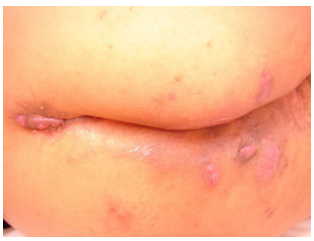

Figure 1c.: Buttocks HS lesions (reproduced with permission from DermNet New Zealand: hs-buttocks-121.jpg).

Secondary diagnostic criteria for HS include a family history of the disease and an absence of pathogens at lesional sites.[44]

Differential diagnoses include staphylococcal infection, cutaneous Crohn disease, simple abscesses, neoplasms, lymphogranuloma venereum, cutaneous actinomycosis and the scrofuloderma type of cutaneous tuberculosis.[44]

Assessment

Clinical assessment of HS severity can be achieved using Hurley staging, the HS-Physicians Global Assessment (HS-PGA) and the modified Sartorius score.[15,44] The Hurley classification is the most widely used of these scores and is useful for the determination of 3 disease severity groups (see **Table 1**), but as a static score it is not suitable for monitoring disease changes with treatment, particularly the inflammatory component of HS.[5,44]

Hurley stage	Description
I	Abscess formation, single or multiple, without sinus tracts and cicatrisation
II	Recurrent abscesses with tract formation and cicatrisation, single or multiple, widely separated lesions
III	Diffuse or near-diffuse involvement, or multiple interconnected tracts and abscesses across the entire area

Table 1. Hurley classification of HS disease severity.[5,44]

The HS-PGA is a dynamic, 6-stage tool which can be used to assess both disease severity and clinical improvement with pharmacological treatment (see **Table 2**).[44,45] However, marked heterogeneity can exist amongst patients in the most severe category, meaning that some patients may experience clinically important improvement without a meaningful reduction in HS-PGA score.[45]

HS-PGA	Description
Clear (score = 0)	No inflammatory or noninflammatory nodules
Minimal (score = 1)	Only the presence of noninflammatory nodules
Mild (score = 2)	<5 inflammatory nodules *OR* 1 abscess or draining fistula and no inflammatory nodules
Moderate (score = 3)	<5 inflammatory nodules *OR* 1 abscess or draining fistula and ≥1 inflammatory nodule *OR* 2-5 abscesses or draining fistulae and <10 inflammatory nodules
Severe (score = 4)	2-5 abscesses or draining fistulae and ≥10 inflammatory nodules
Very severe (score = 5)	≥5 abscesses or draining fistulae

Table 2. HS-PGA classification of HS disease severity.[44,45]

The modified Sartorius score involves counting individual nodules and fistulae, but the fact it includes lesions insensitive to pharmacological treatment, such as scars, is a limitation when assessing treatment effectiveness.[44]

A newly developed, dynamic assessment tool called the Hidradenitis Suppurativa Clinical Response (HiSCR) has been developed and validated in randomised controlled trials of adalimumab.[10,46,47] The HiSCR considers the status of 3 types of lesions: abscesses (fluctuant, with or without drainage, tender or painful); inflammatory nodules (tender, erythematous, pyogenic granuloma lesion); and draining fistulae (sinus tracts, with communication to skin surface, draining purulent fluid).[46] Response to treatment using the HiSCR is defined as a ≥ 50% reduction from baseline in the total abscess and inflammatory nodule count, with no increase in the abscess or draining fistula count.[46] HiSCR responders also show clinically meaningful improvement in DLQI score, Pain Numeric Rating Scale, and measures of the Work Productivity and Activity Impairment questionnaire.[48]

EXPERT COMMENTARY

The diagnosis of HS is clinical, and is based on the presence of chronic and recurrent typical lesions, such as nodules, abscesses, bridged scars, draining sinuses and double-ended comedones, in typical locations. As described, the extent and severity scoring for HS is problematic and there isn't a perfect system. The Special Authority application to PHARMAC for funding of adalimumab (October 2019) uses a combination of Hurley stage and modified HiSCR: Hurley Stage II or III lesions and ≥3 active lesions (e.g. inflammatory nodules, abscesses, draining fistulae) are required for initial application. A 25% reduction in the number of active lesions from baseline is required for renewal.

Treatment

Guidelines for the treatment of HS were published by the European Dermatology Forum in 2015.[44] Based on these guidelines and considering the strength of evidence for various treatment strategies, researchers from the European Hidradenitis Suppurativa Foundation developed a treatment algorithm for HS in 2016.[5] In 2018, the international HS ALLIANCE published consensus-based recommendations for the management of HS, which largely reflect the 2016 European treatment algorithm.[8] Guidelines from the United States and Canadian Hidradenitis Suppurativa Foundations were published in 2019.[9] As these guidelines do not necessarily reflect access to medications or treatment practice in New Zealand and Australia, An Australasian consensus statement was released in 2018.[49]

All guidelines recommend that treatment for HS should be based on disease severity, and should take account of inflammatory components of the disease as well as scarring.[5,8,9,44] Modalities should include surgery as well as pharmacological therapy.[5,8,9,44] All patients should be offered adjuvant therapy for pain, weight loss, tobacco cessation, treatment of super infections and application of appropriate dressings.[5,8,9,44]

The most common comorbidities and complications of HS are:

- Smoking
- Inappropriate diet
- Obesity
- Scarring
- Obstruction of lymph drainage
- Psychological impact.[50]

Metabolic disorders associated with HS can be addressed in primary care with appropriate intervention and referral.[27]

Treatment of HS in the emergency department should be limited to management of acute deterioration of symptoms or side effects of management.[27,51] Patients seen in emergency departments are often treated with simple incision and drainage and a short course of antibiotics.[27,51] Other complications that may present acutely include cellulitis and sepsis, as well as complications from chronic inflammatory disease such as anaemia, hypoproteinaemia, reactive arthritis, ophthalmic complications such as keratitis and corneal ulcerations, and mental health crises, and finally complications of treatment, including potentially serious adverse effects of antibiotics.[27,51]

To ensure adherence with treatment, patients should be educated at the time of diagnosis about the complex nature of HS and its comorbidities.[37] Poor adherence to treatment leads to a larger component of disease-specific cost allotted to inpatient and emergency department care than other chronic skin conditions such as psoriasis.[52] Patients should be referred to support groups, and may also require specialist psychology and psychiatry services.[33,37] A peer support group for New Zealand and Australian patients with HS can be found on Facebook (https://www.facebook.com/groups/101870120150148/).

Pharmacological treatment

First-line pharmacological therapies for HS in New Zealand include topical antiseptics and bleach baths, oral doxycycline, oral clindamycin/rifampicin and subcutaneous adalimumab, and these are discussed in more detail below.[5,8,9,49]

While European and international guidelines recommend the use of topical clindamycin as first-line treatment for patients with PGA mild HS or localised Hurley stage I/mild Hurley stage II HS,[5,8] this treatment is not reimbursed by PHARMAC in New Zealand. It should be noted that use of topical clindamycin increases rates of *Staphylococcus aureus* resistance in patients with HS,[53] and a Ministry of Health Action Plan discourages the use of topical antibiotics.[54]

Second-line, off-label therapies for HS include oral zinc gluconate, topical resorcinol, intralesional corticosteroids, systemic corticosteroids, acitretin, intravenous ertapenem for superinfected flares, and infliximab.[5,8,9] Third-line therapies include a metronidazole + moxifloxacin + rifampicin combination regimen, colchicine, botulinum toxin, isotretinoin, dapsone, ciclosporin, and anakinra.[5,8,9]

Several small studies have evaluated a variety of other biologic agents for the treatment of HS, but high-quality evidence is lacking.[55] International and North American guidelines state that etanercept is not effective in patients with HS, but ustekinumab may be beneficial.[8,9] Neither drug has regulatory approval for HS.

North American guidelines place more emphasis on hormonal therapies for HS than European guidelines, despite limited evidence,[9] and they are also discussed in Australasian guidelines.[49] Hormonal therapies include estrogen-containing combined oral contraceptives, spironolactone, cyproterone acetate, metformin and finasteride for appropriate female patients.[9]

Topical antiseptics and bleach baths

Topical antiseptics such as chlorhexidine, and bleach baths, may be effective in patients with mild HS where there are no deep inflammatory lesions, and they are commonly used in New Zealand as adjunctive therapy in more severe disease.[49] These treatments aim to maintain skin hygiene, reduce bacterial colonisation and potentially suppress a proinflammatory response, but there is no high-level evidence for their use.[49,56]

Systemic antibiotics

Oral tetracycline 500 mg twice daily was as effective as topical clindamycin 1% in a randomised controlled trial of 46 patients with Hurley stage I or II HS.[57] The anti-inflammatory properties of tetracycline are likely to be responsible for its efficacy in this patient population.[58] Oral tetracycline is recommended by European and international guidelines as first-line therapy for patients with PGA moderate HS or more widespread Hurley stage I/mild Hurley stage II HS, particularly when no deep inflammatory lesions are present.[5,8] The treatment is also recommended by North American guidelines.[9]

In New Zealand, as tetracycline is not available, doxycycline 100 mg once daily for 3 months is the preferred systemic antibiotic option.[49] The dose may be increased to 100 mg twice daily if necessary, and reduced to ≤50 mg daily for maintenance treatment.[49] Minocycline is a second-line option due to its less favourable adverse event profile,[59] and is not reimbursed by PHARMAC. There is no high-level evidence for the efficacy of either doxycycline or minocycline in HS.[8]

Three case series found that oral clindamycin 300 mg twice daily combined with oral rifampicin 600 mg once daily or 300 mg twice daily was effective in patients with HS.[60-62] The combination is thought to have immunomodulatory and

anti-inflammatory effects in this patient population.[63] Clindamycin + rifampicin is recommended by European and international guidelines as first-line therapy for patients with PGA moderate to severe HS or Hurley stage II HS for a period of 10 weeks.[5,8] Other treatments must be considered if clinical responses are not achieved within this time.[5] North American guidelines also recommend clindamycin + rifampicin as a first-line or adjunct treatment for severe HS.[9] In New Zealand, PHARMAC reimbursement of clindamycin + rifampicin requires endorsement by a specialist physician.[64]

Adalimumab

The efficacy and tolerability of subcutaneous adalimumab 40 mg/week for the treatment of patients with moderate to severe HS has been demonstrated in randomised controlled trials involving a total of 787 patients.[10,45] A phase 2 dose-ranging trial[45] was followed by the phase 3 PIONEER I and II trials,[10] leading to regulatory approval of adalimumab for moderate to severe HS.[10,11] A long-term extension study showed that the benefit of adalimumab was maintained over a 3-year period, with no additional safety issues.[65] A recent analysis of data from PIONEER I and II has also confirmed the rapid effectiveness of adalimumab in alleviating skin pain.[66]

An updated summary of a 2016 Cochrane Review concluded that there is high-quality evidence of benefit with weekly adalimumab for patients with HS.[7] Adalimumab is recommended in European and international guidelines as first-line therapy for patients with moderate to severe HS who are unresponsive or intolerant to oral antibiotics.[5,8] North American guidelines assign the highest level of evidence and strength of recommendations for adalimumab compared with other treatments for HS.[9]

For adults, the initial dose of adalimumab should be 160 mg, given as two 80 mg injections in one day, one 80 mg injection for two consecutive days, four 40 mg injections in one day or two 40 mg injections for two consecutive days.[11] Two weeks later, adalimumab 80 mg should be given as either one 80 mg injection or two 40 mg injections.[11] At week 4, adalimumab should be continued at a dose of 40 mg per week.[11] For adolescents (from 12 years of age and weighing >30kg), the initial dose of adalimumab should be 80 mg, given as one 80 mg injection or two 40 mg injections, followed by 40 mg fortnightly, starting 1 week later.[11] An increase in dose frequency to 40 mg every week can be considered in adolescents with an inadequate response to fortnightly adalimumab.[11]

Patients demonstrating a partial response after 12 weeks on adalimumab are likely to show a full response if treatment is continued.[67] If no benefit is seen after 12 weeks, adalimumab should be discontinued,[11,67] and second-line treatment options must be considered.[5]

PHARMAC funding criteria for adalimumab

Initial application must be made by a dermatologist. Approvals are valid for 4 months for applications meeting all the following criteria:[12]

- Hurley Stage II or III lesions in distinct anatomic areas
- Inadequate response to ≥90 days of systemic antibiotics or intolerance/contraindications to systemic antibiotics
- At least 3 active lesions (e.g. inflammatory nodules, abscesses, draining fistulae)
- DLQI ≥10 within 1 month of application
- Following the initial loading doses, adalimumab is to be administered at doses ≤40mg every 7 days.[12]

Applications for renewal can be made by a dermatologist or a general practitioner on the recommendation of a dermatologist. Approvals are valid for 6 months for applications meeting all of the following criteria:[12]

- Reduction in active lesions (e.g. inflammatory nodules, abscesses, draining fistulae) of ≥25% vs baseline
- DLQI improvement ≥4 vs baseline
- Adalimumab is to be administered at doses ≤40mg every 7 days. Fortnightly dosing has been considered.[12]

PIONEER I and II trials

Patients enrolled in PIONEER I (n = 307) and II (n = 326) had moderate to severe HS with a total abscess and inflammatory nodule count ≥3 at baseline, an inadequate response to oral antibiotics, and had not previously received anti-TNF-α treatment.[10] Patients in PIONEER I stopped oral antibiotic treatment ≥28 days before study entry, while 19% of patients in PIONEER II continued to receive tetracycline at stable doses.[10] Patients in PIONEER I had a higher mean bodyweight and a greater disease burden than those in PIONEER II.[10] All patients used a daily antiseptic wash on their lesions.[10]

Both PIONEER I and II were multicentre trials with two double-blind, placebo-controlled periods.[10] In period 1, patients were randomly assigned in a 1:1 ratio to receive adalimumab 40 mg weekly or placebo for 12 weeks.[10] In period 2, patients were reassigned to adalimumab 40 mg weekly or every other week, or placebo, for 24 weeks.[10] The primary endpoint was the proportion of patients with a clinical response at week 12, defined according to the HiSCR measure as a >50% reduction from baseline in the total abscess and inflammatory nodule count, with no increase in the abscess or draining fistula count.[10]

The proportion of patients with a clinical response at week 12 of period 1 was significantly higher in the adalimumab vs placebo groups: 41.8% vs 26.0% in PIONEER 1 (p=0.003) and 58.9% vs 27.6% in PIONEER II (p<0.001) (see **Figure 3a** and **Figure 3b**).[10] In the PIONEER II trial, patients who received adalimumab had significantly greater improvement in rank-ordered secondary outcomes (p=0.01 for total abscess and inflammatory nodule count of 0-2 for patients with Hurley stage II disease at baseline, p<0.001 for 30% reduction in skin pain score vs baseline, and p<0.001 for mean improvement in the modified Sartorius score) at week 12 compared with placebo recipients.[10]

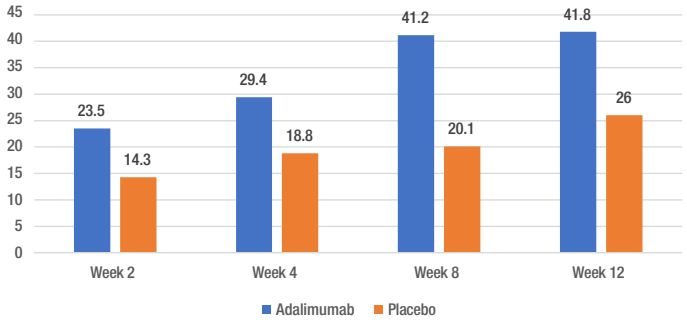

Figure 3a. Patients with clinical response according to HiSCR in period 1 of the PIONEER I trial.[10]

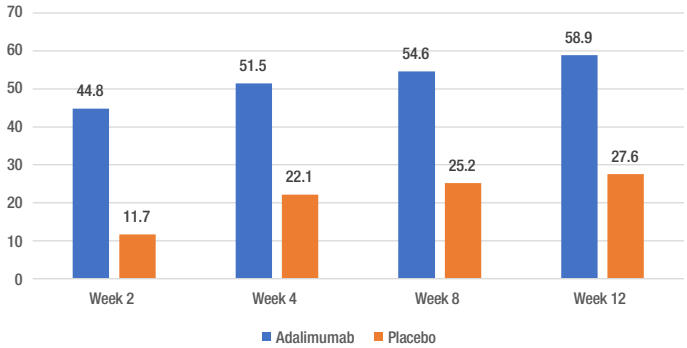

Figure 3b. Patients with clinical response according to HiSCR in period 1 of the PIONEER II trial.[10]

Efficacy outcomes in PIONEER I and II generally favoured weekly dosing with adalimumab rather than every other week.[67]

Serious adverse events in period 1 occurred in 1.3% of patients receiving adalimumab and 1.3% of patients receiving placebo in PIONEER I, and 1.8% and 3.7% of patients, respectively, in PIONEER II.[10] Serious adverse events occurred in ≤4.6% of patients in all groups in both studies during period 2, with no significant between-group differences.[10]

Infliximab

Infliximab 5 mg/kg intravenously has been evaluated in a randomised, double-blind, placebo-controlled trial of 33 patients with moderate to severe HS.[68] There was no statistically significant difference between the infliximab and placebo groups for the primary endpoint (>50% improvement in HS Severity Index score) after 8 weeks of therapy.[68] However, 27% of infliximab recipients had a 25-50% improvement in HS Severity Index score compared with only 5% of placebo recipients ($p<0.001$).[68] DLQI and Visual Analogue Scale pain scores were also improved with infliximab vs placebo.[68] An updated summary of a Cochrane Review concluded there is moderate-quality evidence of benefit with infliximab for patients with HS.[7]

Treatment with intravenous infliximab 5 mg/kg at week 0, 2, 6 and every 2 months thereafter for 12 weeks is recommended by European and international guidelines for patients with moderate to severe HS as a second-line option, after failure of adalimumab.[5,8] Other treatments must be considered if clinical response is not achieved after 12 weeks.[5] North American guidelines also recommend infliximab for moderate to severe HS, but state that dose-ranging studies are needed to determine the optimal dosage, with expert experience suggesting titration to a dosage of 10 mg/kg every 4-8 weeks may be needed for optimal control.[9] Indeed, a recently published retrospective study of 52 patients found that infliximab 10 mg/kg every 6 or 8 weeks was a reasonable starting dose for most patients.[69] Infliximab does not have regulatory approval for the treatment of HS, and is therefore not reimbursed by PHARMAC for this indication.

Surgery

Surgery is a common treatment modality for medically non-responsive HS lesions,[5,44] although there is no universal agreement about the stage at which surgical intervention should take place.[37] Evidence-based studies of surgical techniques are sparse, with most literature comprising case series and retrospective reports.[6,70] The type of surgery chosen depends on the body region and severity of disease.[44] Options include wide excision, local excision, deroofing, carbon dioxide laser therapy, Nd:YAG laser therapy and intense pulsed light.[5,44] In a meta-analysis of surgical techniques for HS, recurrence rates were 13% for wide excisions, 22% for local excisions and 27% for deroofing.[71]

Lasers and intense pulsed light

The use of lasers and intense pulsed light for the treatment of HS has increased over recent years.[72] Carbon dioxide laser is used for cutting or vaporisation of stationary disease elements,[72] however recurrence rates have varied across studies.[5] Nd:YAG laser and intense pulsed light destroy hair follicles and are thus able to reduce disease activity in the treated area.[72] A randomised controlled study of Nd:YAG laser in 22 patients with Hurley stage II-III HS found disease severity significantly improved after 3 one-monthly treatment sessions ($p<0.05$ vs baseline).[73] A significant improvement in mean examination score occurred in 18 patients with HS randomised to twice-weekly treatment with intense pulsed light for 4 weeks on one side of bilaterally affected region, and this was maintained at 12 months ($p<0.001$).[74]

EXPERT COMMENTARY

Although mild HS often responds to lifestyle changes and intermittent or continuous courses of doxycycline, treatment of moderate to severe HS remains challenging. Dermatologists in New Zealand are excited to have the option to use adalimumab in this group of patients. Surgical management of HS is difficult to access in many regions.

TAKE-HOME MESSAGES

- HS is a painful, chronic inflammatory skin disease occurring in 1-4% of the population[1,26,27]
- HS is poorly recognised, with an average diagnostic delay of 7.2 years[4]
- HS has profound physical and psychological consequences that affect quality of life[3]
- A number of pharmacological and surgical treatments are currently used in HS, but high-quality evidence is lacking for many of them[5-9]
- The anti-TNF-α monoclonal antibody adalimumab is the only pharmacological treatment with registered approval for HS, both in New Zealand and worldwide[10,11]
- Adalimumab is now reimbursed by PHARMAC for patients with moderate to severe disease and an inadequate response to antibiotics, when prescribed by a dermatologist.[12]

EXPERT CONCLUSIONS

Once health professionals are aware of it, chronic inflammatory nodules in the axilla, submammary area and groin are easy to diagnose as HS. Advice regarding smoking cessation and obesity management, and a 3-month course of doxycycline, can be commenced in primary care. When sinus tracts are present, patients are best referred to a dermatologist for assessment and treatment with a longer course of antibiotics, adalimumab, or perhaps other agents. The referral should include a description of the affected areas and lesion morphology (and photographs), and report the results of any treatment.

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