Hidradenitis Suppurativa

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Key Points

- Hidradenitis suppurativa/acne inversa (HS) is a chronic, multifactorial, debilitating, inflammatory skin appendage condition of the hair follicle.
- HS is a common disorder with a point prevalence of 1.0 % and a female preponderance.
- Management of HS should encompass the education of the patient regarding the nature of the disease, general measures, pharmacological topical or systemic treatment and surgical therapy.
- Antibiotic, immunosuppressive, anti-inflammatory, antiandrogenic and surgical treatments, as well as laser and light sources, have been used for the management of HS, although few randomised, controlled trials exist to provide evidence-based data on their efficacy for this condition.
- Topical clindamycin 1 % is the only studied topical antibiotic for HS.
- Systemic treatments that have been used for HS include antibiotics, retinoids (acitretin), biologic agents, antiandrogens, zinc gluconate, dapsone, colchicine, corticosteroids, ciclosporin and metformin.
- The combination of clindamycin and rifampicin is indicated for active inflammatory HS.
- The treatment choice will depend on the severity and extent (localised or widespread disease) of HS.
- The combination of different treatment modalities may be required to achieve improvement.

Definition and Epidemiology

The definition of hidradenitis suppurativa (HS) relies on clinical criteria. According to the 'Dessau definition' of HS, it is a chronic, inflammatory, recurrent, debilitating skin disease that usually presents after puberty with painful, deep-seated, inflamed lesions in the apocrine gland-bearing areas of the body, most commonly the axillary, inguinal and anogenital regions (First International Conference on Hidradenitis suppurativa, March 30–April 1, 2006, Dessau, Germany).

Although HS was considered a rare disease in the past, its prevalence has been estimated to be 1–4 %. It presents as a sporadic or familial form.

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A.D. Katsambas et al. (eds.), *European Handbook of Dermatological Treatments*,
DOI 10.1007/978-3-662-45139-7_42, © Springer-Verlag Berlin Heidelberg 2015

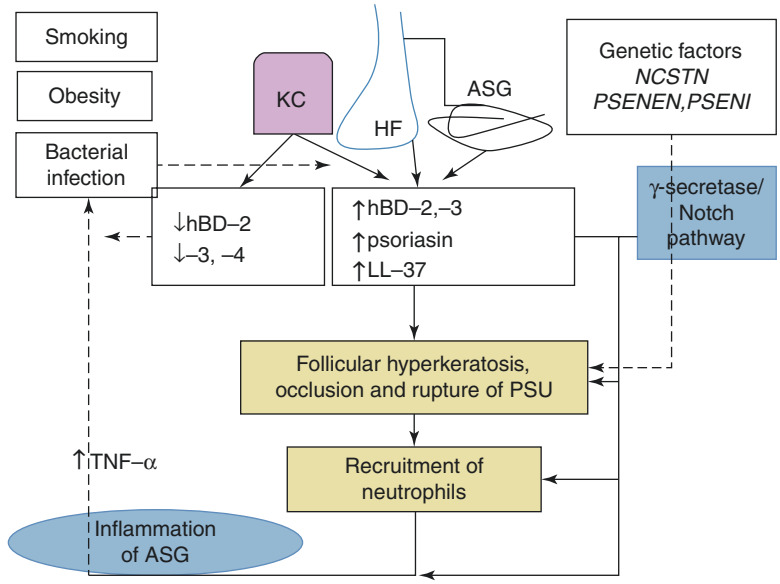

Fig. 42.1 Schematic pathophysiology of HS (Dessinioti et al. 2013)

HS affects more women than men, with a female-to-male predominance as high as 4:1. The disease occurs almost always after puberty and before the age of 40 years.

The chronicity, relapses and sequelae of the disease, including dermal contraction, scarring, restricted limb mobility, lymphoedema and fistula formation, contribute to the long-term burden of the disease and have a considerable negative impact on the patients' psychology, sex health and quality of life.

Basic Concepts of Pathogenesis

The pathogenesis of HS has not yet been elucidated, but it is considered to be a multifactorial disease. Current theories implicate the hyperkeratosis of the follicular epithelium as the morphological hallmark of HS pathogenesis, leading to occlusion of the apocrine glands with subsequent follicular rupture, inflammation and possible secondary infection. Genetic factors, hormones, smoking, obesity, bacterial infection and alteration of antimicrobial peptides (AMPs) that regulate cutaneous innate immunity have been implicated in the multifactorial pathogenesis of HS (Fig. 42.1).

It is clear that bacterial infection is not the primary cause of HS. However, bacteria, their products and immune responses raised against bacterial antigens may influence the clinical phenotype of HS lesions as well as the course of HS in any given patient (Zouboulis et al. 2012). In terms of natural immunity, patients with HS may respond abnormally to these bacteria (e.g. *Propionibacterium acnes*), whose products stimulate Toll-like receptors, producing pro-inflammatory cytokines such as IL-1α and giving rise to abnormal keratinisation (Zouboulis et al. 2012).

Current smoking has been associated with HS, with a highly significant association in multivariate analysis (OR = 12.55) (Hana et al. 2007). It has been suggested that smoking affects polymorphonuclear cell chemotaxis. Obesity and being overweight are frequent in HS and could represent a risk factor. Two case-control studies by Revuz et al. showed an association of HS with body mass index in medically assessed patients ($n = 302$) (odds ratio = 1.12 [1.08–1.15]) for each increase of 1 U of BMI. However, HS may also develop in low or normal body mass index.

Clinical Presentation

The clinical lesions of HS consist of primary lesions including painful nodules and abscesses and secondary lesions including sinus tracts,

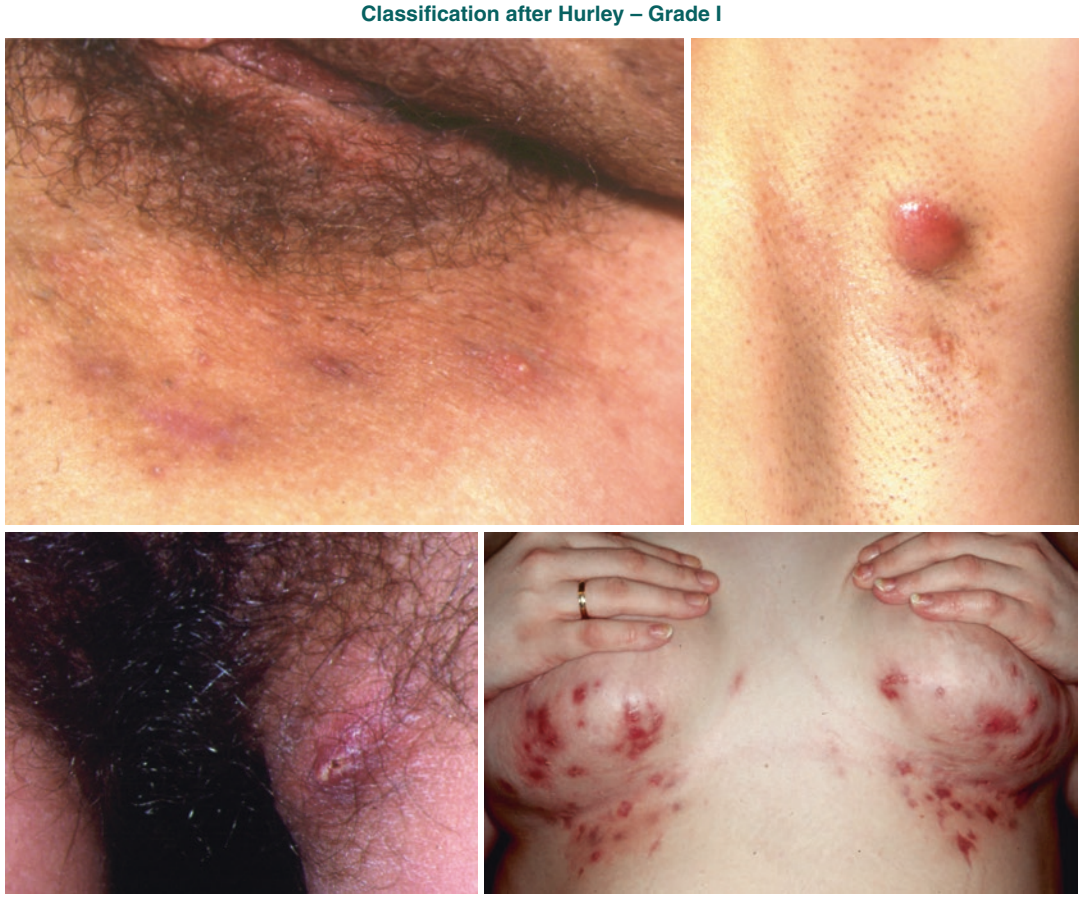

Fig. 42.2 Hidradenitis suppurativa Hurley grade I

hypertrophic scars and multiparous or uniporous 'tombstone' open pseudo-comedones. In contrast to acne, there is no hyperseborrhoea or closed comedones in HS.

The topography of involvement may be explained by the distribution of apocrine glands and shearing forces, which originate in large skin folds, especially of overweight patients. The location of HS lesions is along the 'milk line' of apocrine and mammary tissues that have the same embryonic origin.

For each affected body site, the severity of the disease can be classified in three grades according to the Hurley classification (Figs. 42.2, 42.3 and 42.4) that provides a simple but static assessment (Table 42.1). The Sartorius score is also used as a more complex, dynamic score to assess severity of HS, mainly in the context of clinical studies, grading the number, size and extent of individual lesions.

Recently, Canoui-Pointrine et al. reported three distinct HS phenotypes in 618 patients, including the 'axillary-mammary' class (48 %) that had a high probability of breast and armpit lesions and hypertrophic scars; the 'follicular' class (26 %) that were more often male, current smokers and with more severe disease and had a high probability of breast, armpit, ear, chest, back or leg lesions and follicular lesions and a family history of HS; and the 'gluteal class' that were less often obese and had less severe, with gluteal involvement, papules and folliculitis.

Complications of HS include lymphatic obstruction and lymphoedema of the anogenital area; the development of squamous cell carcinoma in 2–4 % of patients, especially in men and in the buttock area; and serious infections, anaemia or hypoproteinaemia.

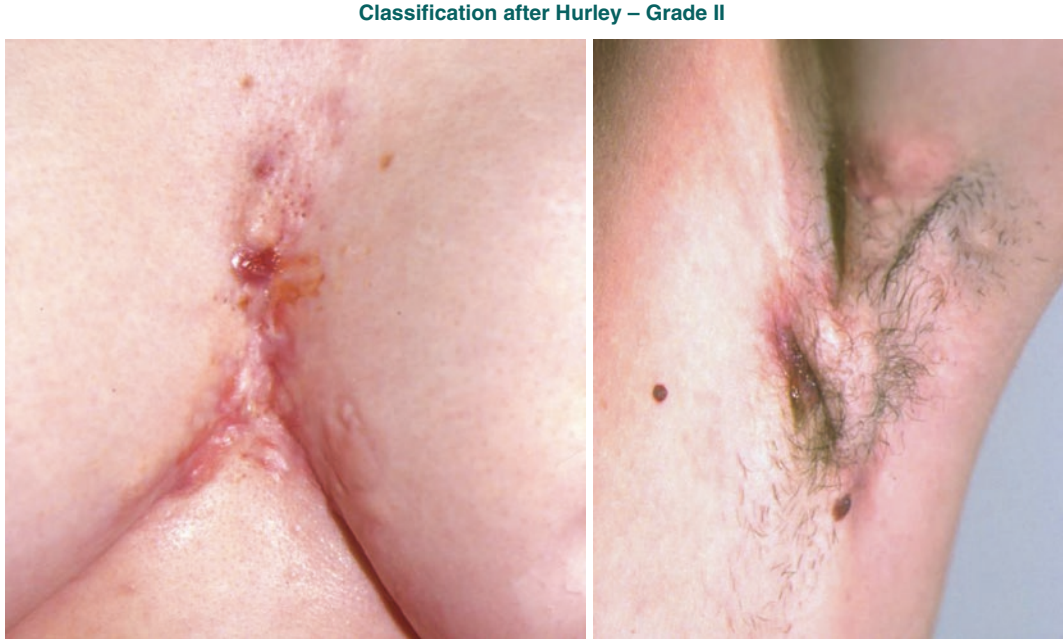

Fig. 42.3 Hidradenitis suppurativa Hurley grade II

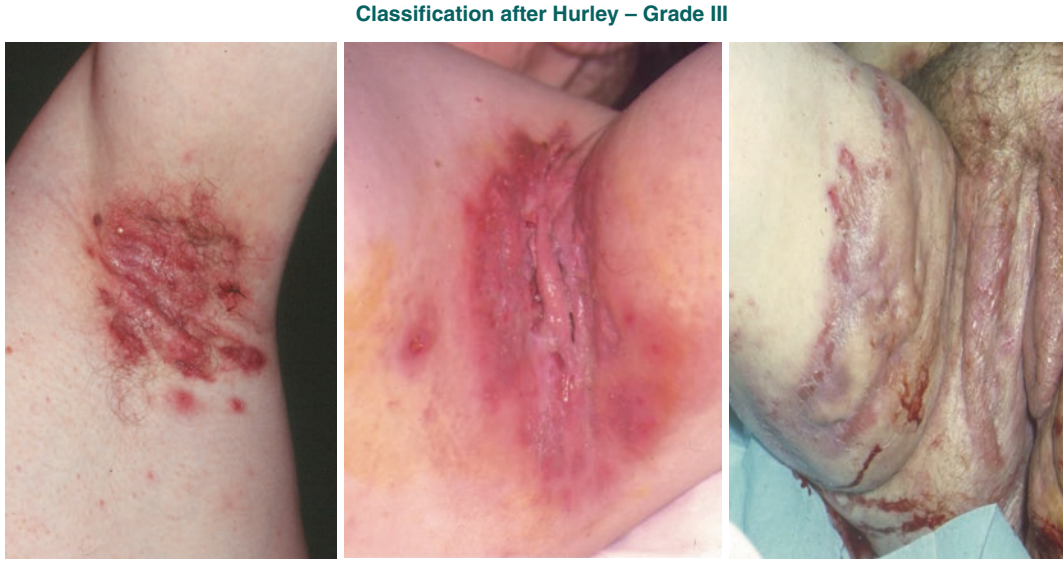

Fig. 42.4 Hidradenitis suppurativa Hurley grade III

Several diseases have been reported in association with HS, including Crohn's disease, the synovitis-acne-pustulosis-hyperostosis-osteitis (SAPHO) syndrome, pyoderma gangrenosum, acne and suppurative hidradenitis (PASH) syndrome, pyoderma gangrenosum, Adamantiades-Behçet's disease, spondyloarthritis without or with follicular occlusion triad signs, genetic keratin disorders associated with follicular occlusion (pachyonychia congenita, steatocystoma multiplex, Dowling-Degos disease without and with arthritis), keratitis-ichthyosis-deafness (KID) syndrome and Down syndrome.

The follicular occlusion tetrad includes HS, acne conglobata, pilonidal sinus and dissecting cellulitis of the scalp; these conditions are characterised by follicular occlusion as the initial event.

Table 42.1 Severity classification of hidradenitis suppurativa according to Hurley staging

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

Diagnosis and Differential Diagnosis

The diagnosis of HS is clinical and a biopsy is not necessary for diagnosis. The following three diagnostic criteria must all be met:

1. Chronicity and recurrences
2. Typical lesions, i.e. deep-seated painful nodules: 'blind boils' in early lesions; abscesses, draining sinus, bridged scars and 'tombstone' double-ended pseudo-comedones in secondary lesions
3. Typical topography, i.e. axillae, groins, perineal and perianal region, buttocks and infra- and inter-mammary folds.

Closed comedones in HS are never present. The apparently open comedones are never closed; they are double-ended 'pseudo-comedones', i.e. literally scars.

Histologically, HS is a disease of the hair follicle, with follicular occlusion leading to the occlusion of the apocrine gland and subsequent follicular rupture, associated with lympho-histiocytic inflammation, inflammation, granulomatous reactions, sinus tracts and scarring. The early lesions of HS demonstrate follicular hyperkeratosis. The deep part of the follicle appears to be involved. Dermal features include perifolliculitis, active folliculitis or abscess, sinus tract formation, fibrosis and granuloma formation.

HS should be differentiated from:

- Folliculitis: it presents with follicular pustules. Comedones are absent.
- Furuncles (boils): may be confused with the early solitary, painful nodules of HS. In HS, nodules are more deeply seated, they are located on the characteristic anatomic sites affected by HS and there are recurrences and progression to abscesses and scars over time.
- Crohn's disease: cutaneous CD mimicking HS has to be taken into consideration in case of sole perianal lesions. Metastatic CD cannot be easily differentiated from HS. There is associated intestinal Crohn's disease.
- Acne vulgaris: there are always closed comedones and different topography of the lesions.

General Principles of Treatment

Management of HS includes the education of the patient regarding the nature of the disease, general measures, pharmacological topical or systemic treatment and surgical therapy.

The education of the patient by the treating dermatologist is pivotal, so that he/she understands that HS is usually a chronic disease, with exacerbations and remissions, and that it may necessitate long-term therapies. Also, the patient should be reassured that HS is not contagious and that it is not caused by an infection of the skin or by the lack of hygiene.

General measures and lifestyle changes may contribute to the better management of HS. Friction should be avoided by wearing loose-fitting clothes. Although no data exist for the improvement of HS lesions after reduction of weight and cessation of tobacco smoking, the general expert opinion (EDF) is that cigarette smoking and overweight have to be avoided.

Antibiotic, immunosuppressive, anti-inflammatory, antiandrogenic and surgical treatments, as well as laser and light sources, have been used for the management of HS, although few randomised, controlled trials exist to provide evidence-based data on their efficacy for this condition. A systematic review on the use of immunosuppressive treatments and systemic retinoids for HS evaluated 87 papers including 518 HS patients treated with biologics, colchicine, ciclosporin, methotrexate, dapsone, acitretin or isotretinoin and graded the level of evidence. The authors reported that infliximab, adalimumab and acitretin were the most effective systemic agents, with 89, 79 and 95 % of patients, respectively, responding to treatment (however, the quality of

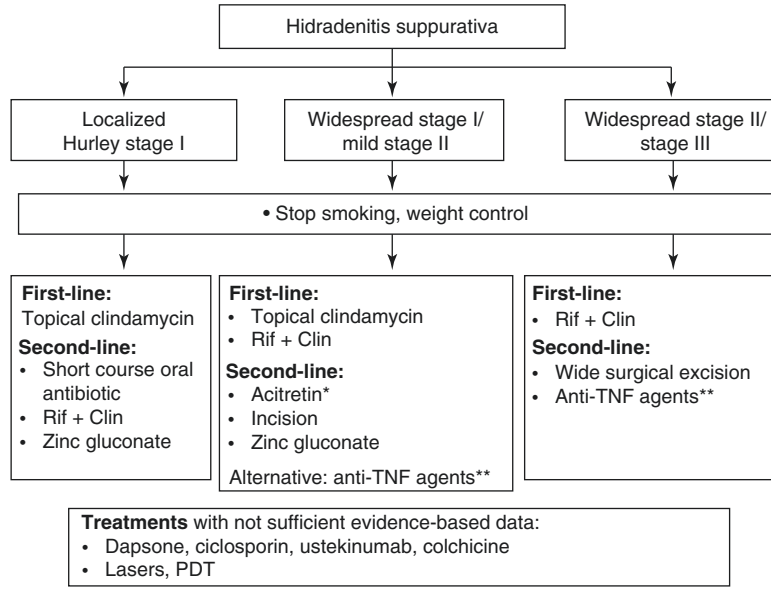

Fig. 42.5 Algorithm of treatment for hidradenitis suppurativa. *Rif+Clin* rifampicin plus clindamycin, * strictly contraindicated for women of childbearing potential, ** off-label therapies

evidence was lower for acitretin) (Matusiak et al. 2009). Nonsteroidal anti-inflammatory drugs (NSAIDs) may be proposed as analgesics for the symptomatic therapy of pain.

Based on the severity of HS, for Hurley stage I, topical or systemic drugs are proposed; stage II may benefit from medical treatment and from limited excisions of locally recurring lesions; and stage III requires radical surgery (Zouboulis et al.). The severity of HS, and whether it is localised or more widespread, should be taken into account before determining optimal treatment. The combination of different treatment modalities may be required to achieve improvement (Matusiak et al. 2009). A proposed algorithm of treatments is presented in Fig. 42.5.

Topical Treatments

Topical Antibiotics: Clindamycin

Topical clindamycin is the only antibiotic that has been studied in HS. A randomised, placebo-controlled study by Clemmensen et al. in 27 HS patients showed that topical clindamycin 0.1 % was superior according to patients' assessments and improved lesion counts. Another randomised trial by Jemec et al. compared topical clindamycin 1 % solution twice daily with oral tetracycline 500 mg twice daily, for 3 months, with response and no significant difference in efficacy between topical and oral treatment (Brocard et al. 2007). Topical clindamycin may be applied twice daily for 3 months or longer if clinically indicated.

Resorcinol 15 %

Resorcinol 15 % cream twice daily has been described for flares of lesions in patients with Hurley stage I or II HS.

Intralesional Steroids

Intralesional injection of triamcinolone acetonide may be used for individual early HS lesions.

Topical Treatments at a Glance

- Topical antibiotic treatment with clindamycin is indicated for localised Hurley stage I or mild stage II disease.

Systemic Treatments

The use of systemic treatments is indicated for more severe or widely spread lesions. Systemic treatments that have been used for HS include antibiotics, retinoids, biologic agents, antiandrogens, zinc gluconate, dapsone, colchicine, corticosteroids, ciclosporin and metformin.

Antibiotics

Antibiotics may be used as a short-term treatment to provide a rapid control of an exacerbation of the disease or as a long-term therapy with the aim to provide improvement as well as sustained remission.

For an acute flare of HS, a short course of an oral antibiotic such as amoxicillin/clavulanic acid, cephalosporin or clindamycin may be proposed.

For long-term continuous therapy, the combination of clindamycin and rifampicin is useful.

Clindamycin and Rifampicin

Although HS is not primarily an infectious disease, the combination of two antibiotics, rifampicin and clindamycin, is one of the most useful regimens in cases of inflammatory lesions. The combination of clindamycin and rifampicin for the treatment of HS has been evaluated in three retrospective studies, including a total of 118 patients. A response was reported in 96 patients (81.36 %), with 32 (27.11 %) achieving complete remission (Van der Zee et al. 2009, 2010; Mendonca and Griffiths 2006). Most reported side effects were gastrointestinal and affected 23 of 104 (22.11 %) patients (Gener et al. 2009).

Another prospective study of Bettoli et al. in 23 patients with HS treated with oral clindamycin (600 mg daily) and rifampicin (600 mg daily) for 10 weeks reported improvement (a Sartorius score improvement higher than 25 %) in 85 % of patients. Side effects were reported in three patients (13 %) and included nausea and vomiting.

Oral clindamycin should not be used in patients with intestinal inflammatory disease. The development of marked diarrhoea should prompt its immediate discontinuation, as it may progress to pseudomembranous colitis caused by *Clostridium difficile*. Liver function evaluation and complete blood count should be carried out during prolonged treatment.

Oral rifampicin at very high doses has been shown to have teratogenic effects in animals. There are no well-controlled studies with rifampicin in pregnant women (FDA pregnancy category C). Rifampicin causes an orange coloration of urine, swat, sputum and tears. Monitoring of hepatic function is necessary during treatment with oral rifampicin, and liver function evaluation, bilirubin, serum creatinine and complete blood count should be carried out.

The EDF guidelines recommend this combination for any stage active inflammatory HS.

Retinoids

Isotretinoin

Systemic isotretinoin (13-*cis* retinoic acid), a vitamin A derivative, has been used as an off-label treatment for HS, but it is ineffective in the treatment of HS, as this agent primarily works on sebaceous glands, which are not involved in the pathogenesis of HS (Zouboulis et al.; Matusiak et al. 2009).

The systematic review of Blok et al. reported seven papers evaluating the use of isotretinoin for HS in a total of 174 patients. Daily dosages were 0.5–1.2 mg/kg and treatment duration was 4–12 months. There was no response in the majority of patients (112 patients, 64 %), while 30 patients (17 %) had moderate improvement and 32 patients (18 %) had significant improvement.

Oral isotretinoin is a known teratogen for women (FDA pregnancy category X). Before treatment initiation, women of childbearing age should be informed about teratogenicity of isotretinoin and the absolute need of avoidance of pregnancy and effective contraceptive measures throughout treatment and for 1 month after treatment completion.

Acitretin and Etretinate

Retinoids have antiproliferative and immunomodulatory properties. Etretinate is a prodrug of acitretin. Acitretin is converted in vivo to etretinate

by re-esterification. Acitretin also has anti-inflammatory actions and may target the process of hyperkeratosis of the infundibular follicular epithelium, normalising epithelial cell proliferation and differentiation (Matusiak et al. 2009).

In the study of Boer et al., 12 patients with HS Hurley stage II/III were treated with acitretin (mean dose 0.59 mg kg once daily) as monotherapy for 9–12 months (mean 10.8). Overall, an improvement was noted in all 12 patients. Nine patients achieved marked or complete remission after one course of acitretin therapy, and the other three patients showed mild or moderate improvement. Follow-up showed that nine patients had a long-lasting improvement, with remissions ranging from 6 to 45 months (mean 24.9). A systematic review evaluated six available studies on the use of acitretin and etretinate for HS that included 22 patients. Used doses were 0.35–1.1 mg/kg for etretinate and 0.25–0.88 mg/kg for acitretin for a period of 2–29 months. Significant improvement was reported in 16 of 22 patients (73 %), moderate improvement in 5 (13 %) and no response in 1 (5 %). Within a follow-up of 6 months, there was recurrence in 6 of 17 patients (35 %), while 8 patients (47 %) relapsed later than 1 year after discontinuation of treatment.

The EDF guidelines recommend acitretin for early HS stages (Hurley I or mild II) and for the chronic stages of HS with recurrent abscesses with sinus tracts and/or scarring.

Retinoids, including etretinate and acitretin, are potent teratogens, leading to strict requirements for pregnancy prevention during their use and after their discontinuation. In the case of acitretin (FDA pregnancy category X), women of childbearing potential have to use adequate contraception measures during therapy and for 24–36 months after discontinuation, depending on local regulations.

Biologic Agents

Anti-TNF-α Agents: Adalimumab, Etanercept, Infliximab

Tumour necrosis factor alpha (TNF-α) is a pro-inflammatory cytokine produced by many cell typesı, including macrophages, monocytes and T cells. It is expressed in the basal layer of the epidermis, sweat glands and hair follicles. The rationale for the use of anti-TNF agents for the treatment of hidradenitis was based on the clinical observation of improvement of HS in patients with concomitant Crohn's disease, as well as on experimental evidence that TNF-α may play a key role in HS. TNF-α serum concentration in 54 HS patients was significantly higher than healthy controls ($p = 0.006$), although it was not associated with the severity of the disease. Van der Zee et al. showed elevated TNF-α production after culture of biopsies of lesional and perilesional skin of 20 HS patients compared to skin of six healthy controls and similar TNF-α levels with that of the skin of seven psoriasis patients. Also, the −238 G⁄A SNP at the promoter region of the TNF gene was significantly more frequent in HS patients than in healthy controls ($p = 0.027$), and a certain haplotype of the TNF gene seems to be associated with a greater reduction of disease severity after treatment with TNF agents.

Adalimumab is a fully anti-TNF monoclonal antibody that binds with high affinity and specificity to soluble and membrane-bound TNF-α. It is administered subcutaneously. In the systematic review of Blok et al., adalimumab was evaluated in 15 papers in 68 HS patients, with only one being a randomised, double-blind, placebo-controlled design (evidence level A). Dosing regimens varied from 40 to 80 mg, in a frequency ranging from weekly to every other week. In total, 30⁄68 patients (44 %) showed a significant response to adalimumab, 24 patients (35 %) had a moderate response and 14 patients (21 %) did not respond. Relapse of HS was reported in 23 out of 35 responders (66 %) within 3–10 months after discontinuation of treatment, while 7 patients (20 %) relapsed during treatment but improved with an increase in the dose of adalimumab.

Etanercept is a soluble fusion protein comprising a TNF-α receptor and the Fc component of human immunoglobulin G1. It is administered subcutaneously. Etanercept was evaluated in nine papers in 54 patients, with only one being a randomised, double-blind, placebo-controlled design (evidence level A). Dosing schedules varied from

25 to 50 mg once or twice weekly to 100 mg weekly. In total, 21⁄54 patients (39 %) showed a significant response to etanercept, nine patients (17 %) had moderate improvement and 24 patients (44 %) did not respond. Relapse of HS was reported in 18 out of 30 responders (60 %) within immediately up to 8 months after discontinuation of treatment.

Infliximab is a chimeric (mouse/human) anti-TNF monoclonal antibody that binds both soluble and transmembrane TNF-α. It is administered intravenously. Infliximab was evaluated in 42 papers in 147 patients, with only one being a randomised, double-blind, placebo-controlled design (evidence level A). Dosing schedule was 5 mg kg^{-1} at weeks 0, 2 and 6 and every 6–8 weeks thereafter. In total, 74⁄147 patients (50 %) showed a significant response to infliximab, 57 patients (39 %) had moderate improvement and 16 patients (11 %) did not respond. Relapse of HS was reported in 26 out of 131 responders (20 %) 2 weeks to 3 years after discontinuation of treatment.

Anti-IL12/23 Agents: Ustekinumab

Ustekinumab is a human monoclonal antibody against interleukin 12/23 (IL 12/23). It is administered subcutaneously. The systematic review of Blok et al. reported two papers in four patients evaluating ustekinumab for HS (both evidence level C). Dosing schedule was 45 mg at weeks 0, 4 and 12 and every 3 months thereafter. In total, two out of four patients showed a significant response to ustekinumab, one patient had a moderate response and one patient did not respond. Relapse of HS was reported in two out of three responders.

Antiandrogens

The rationale for the use of antiandrogens for HS was based on the female preponderance of HS, its development after puberty and its association with PCOS.

On the other hand, the usual absence of clinical signs of hyperandrogenism and the normal levels of serum androgens do not support a role for androgens. This may explain the limited effect of anti-androgen treatments for HS.

Zinc Gluconate

Zinc acts via inhibition of polymorphonuclear cell chemotaxis. Its anti-inflammatory activity could be related to a decrease in TNF-α production and the modulation of the expression of integrins and the inhibition of Toll-like receptor 2 (TLR2) surface expression by keratinocytes.

In a pilot study of Brocard et al., 22 patients with Hurley grades I and II were treated with 90 mg/day zinc gluconate. Complete remission was noted in eight patients and partial remission in 14 patients. Four out of 22 patients experienced side effects of gastrointestinal upset.

The EDF guidelines recommend zinc gluconate as maintenance therapy for HS Hurley stages I and II.

Dapsone

A systemic review of Blok et al. reported three studies in 34 patients treated with dapsone at dosages of 25–100 mg daily during 0.5–48 months. There was a significant improvement seen in 12 of 34 patients (35 %), moderate improvement in 7 patients (21 %) and no response in 15 patients (44 %). It was reported that there was a rapid recurrence after treatment discontinuation in all patients.

The EDF guidelines recommend dapsone as third-line treatment for mild to moderate HS (Hurley stage I or II).

Colchicine

Colchicine is a natural product that can be extracted from plants of the lily family. It inhibits neutrophil expression of cell adhesion molecules and decreases neutrophil degranulation, chemotaxis and phagocytosis.

There is a report by van der Zee et al. of eight patients with refractory HS treated with

colchicine 0.5 mg twice daily for up to 4 months that reported that six patients (75 %) did not respond, while there was a moderate response in the remaining two patients. Adverse events were nausea and diarrhoea. The authors suggested that higher doses may be needed for clinical efficacy and that colchicine treatment should start with 1 mg followed by 0.5 mg every 2 h with a cumulative maximum of 5 mg, followed by 1 mg daily (Boer et al. 2011).

The EDF guidelines recommend that colchicine should not be used for the treatment of HS.

Corticosteroids

Systemic steroids may provide relief from pain and inflammation and be used as an alternative or in combination with oral antibiotics. Steroids may be used for a short period of time and tapered rapidly.

Ciclosporin

Ciclosporin is a calcineurin inhibitor with potent immunosuppressive activity. It targets T lymphocytes and it inhibits the production of TNF-α and IL-2.

Ciclosporin at a dose ranging from 2 to 6 mg/kg/day has been studied in four patients for 4–16 months, with a significant response in three patients and a moderate response in the remaining two patients.

Metformin

Metformin improves insulin insensitivity and has antiandrogenic properties. Verdolini et al. studied metformin in 25 patients with HS, at a starting dose of 500 mg once daily for the first week, increased to 500 mg twice daily for the second week, with a maximum dose of 500 mg three times daily for a total of 24 weeks. There was improvement in 18 patients, while seven patients (28 %) showed no response. Only minor gastrointestinal side effects were reported.

Systemic Treatments at a Glance

- The combination of clindamycin and rifampicin is indicated for any stage active inflammatory HS.
- A systematic review evaluating immunosuppressive treatments and oral retinoids for hidradenitis suppurativa reported that infliximab, adalimumab and acitretin were the most effective systemic agents, with 89, 79 and 95 % of patients, respectively, responding to treatment (however, the quality of evidence was lower for acitretin).
- Oral acitretin should be reserved for male patients and sterilised or postmenopausal women due to its long-term teratogenic side effects.
- Oral isotretinoin was shown to be ineffective for hidradenitis suppurativa.
- There are a limited number of studies in a small number of patients on the efficacy of ustekinumab, colchicine, dapsone, ciclosporin and zinc gluconate.
- There is limited data on long-term results and relapse rates with systemic treatments for HS.

Surgical Therapy

The surgical approach for HS Hurley stage I is incision and drainage; for stage II, deroofing and secondary healing; and for HS stage II/III, wide surgical incision and healing with secondary intention or skin graft.

Surgical incision and drainage is indicated as means of symptomatic relief of pain, e.g. as an analgesic for a fluctuating abscess. Incision does not affect the course of the disease nor does it provide clearance of the incised lesion.

A simple technique consists of unroofing under local anaesthesia and debridement of the scars, abscesses, cysts and sinuses. In the study of van der Zee et al., 44 patients with mild to moderate HS were treated with the surgical deroofing technique, with no recurrence in 83 % of treated lesions after a median follow-up of 34 months.

Traditional surgery for hidradenitis suppurativa (HS) consists of en bloc wide excision followed by primary closure or healing by secondary

intent. Mapping of sinus tracts with methyl blue intra-operatively is crucial. In one series of 106 patients, there was a 70 % recurrence rate requiring subsequent operation in the primary closure cohort and no recurrence in the split-thickness graft and flap groups. In general, the recurrence rate with wide excision has been reported to be less than 30 %. However, poor surgical outcomes (scarring) have been associated with surgery (van der Zee and Prens 2011).

Other Treatment Options

Lasers and Light Sources

There is scarce data on the use of laser and light sources for the treatment of HS.

Long-pulsed neodymium:yttrium-aluminium-garnet (Nd:YAG) laser is a laser hair reduction device. The rational for treating HS was based on the role of follicular occlusion as the initiating event in the pathogenesis of HS. The proposed mechanism of action included the release of obstruction of the hair follicle and photothermolysis.

A prospective, randomised study of Mahmoud et al. in 22 patients with HS evaluated the efficacy of Nd:YAG laser, clinically and histopathologically. Topical benzoyl peroxide wash 10 % and clindamycin 1 % lotion were used on both sides of the face. Laser treatment consisted of four monthly sessions. The reported fluence was 40–50 J/cm^2, pulse duration was 20 ms and spot size was10 mm, whereas for skin types IV–VI, the fluence used was 25–35 J/cm^2, pulse duration was 35 ms and spot size was 10 mm. The percent improvement was 72.7 % on the laser treated side and 22.9 % on the control side ($p<0.05$). Similar results were reported by Xu et al. in 19 patients with HS Hurley stage II, treated with two monthly sessions of long-pulsed 1,064-nm Nd:YAG laser. The percentage change in HS severity after two sessions of laser treatment was −31.6 over all anatomic sites ($p<0.005$).

Photodynamic therapy (PDT) as treatment for HS has been recently described in three small case series, but it is neither established nor has it been standardised.

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