**Historical background**

Hidradenitis suppurativa (HS) was first described in 1854 by a French surgeon, Aristide Verneuil [1]. It was linked to sweat glands, although those involved, the apocrine sweat glands were first described only in 1921. In 1955 Shelley [2] experimented with occluding the axillary skin and concluded that the primary event in HS was keratinous plugging of the apocrine sweat duct. Later Yu and Cook MG [3] showed that follicular occlusion was the most constant feature of HS and probably the primary event, confirming the conclusion of Kligman and Plewig [4]. The latter also grouped HS with acne conglobata, dissecting folliculitis of the scalp, and (later) pilonidal cysts, designated this association as follicular tetrad, and renamed HS, calling it acne inversa.
Definition

HS is diagnosed by its clinical features and its chronic nature; no laboratory test or pathology findings identify it. It is defined by the presence of recurrent, painful, deep-seated, rounded nodules that develop into abscesses and sinus tracts, with suppuration and hypertrophic scarring of apocrine gland-bearing skin.

In San Francisco in March 2009, the second international symposium organized by the Hidradenitis Suppurativa Foundation adopted a consensus definition [5]: "HS is a chronic, inflammatory, recurrent, debilitating skin follicular 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.”

Epidemiology

Prevalence

Although HS is considered a rare disease, several studies report a global prevalence of 1% in the general population [6]. A large French survey confirmed this prevalence [7]. The apparent rarity of HS in clinical experience may be explained by the prevalence of mild forms, the discouragement of patients who find no successful treatment, and by the lack of knowledge about HS in the medical community, revealed by the diagnostic delay (usually several years). Moreover HS is a disease often kept secret, even within the family, and some patients are reluctant to seek medical advice. The peak prevalence recorded is 4%, for self-reported cases in a population of young women in Denmark. It is likely due to the higher prevalence in young adults and in women (sex ratio: 1/3.3) [8], confirmed by clinical examination of patients in another study [9]. Although HS is not rare, it is an orphan disease in view of the lack of knowledge about it in the medical community and the poor results of most therapeutic interventions.

Risk factors

Naldi [6] has reviewed the risk factors for HS. A case-control study of factors associated with HS in 302 patients [7] confirmed that smoking and overweight are the two main factors associated with HS. Smoking was significantly more common – more than 70% – in HS patients than in controls, and multivariate analysis showed a strong association with current smoking [odds ratio (OR) = 12.55, 95% confidence interval (CI) (8.58; 18.38)]. Because no temporal relationship or dose effect has been demonstrated between smoking and HS, this strong association cannot be considered a true risk factor. Furthermore, no specific mechanism has been shown to explain this association, and no study has documented the effect of smoking cessation. Nonetheless, the medical community generally accepts that smoking is the most important triggering factor of HS. The same study observed an association with overweight and obesity (risk increased [OR = 1.12, 95% CI (1.08–1.15) for each increase of one unit of body mass index]. Given the dose/effect relationship, overweight and obesity may be true risk factors. A conclusive demonstration, however, requires proof of a temporal relationship and an improvement in HS following weight loss. Antiperspirant, talc, and deodorant use and depilation with a safety razor have been ruled out as pathogenic factors [10]. Tight clothing may contribute to discomfort and is considered a triggering factor by some authors. We consider genetic and hormonal factors below.

Diagnostic criteria

According to the criteria adopted by the international symposium of the Hidradenitis Suppurativa Foundation in March 2009 (5), diagnosis requires the presence of:

- typical lesions, i.e., deep-seated painful nodules: “blind boils” in early lesions; abscesses, draining sinuses, bridged scars, and open “tombstone” comedos in secondary lesions;
- typical topography, i.e., axillae, groin, perineal and perianal regions, buttocks, inframammary folds; and;
- chronicity and recurrences.

All three criteria must be met to establish the diagnosis.

Clinical description

Onset usually occurs after puberty, usually during the second or third decades of life. There is frequently a long delay before diagnosis, particularly in mild cases: an abscess may be surgically treated by incision, and the chronic or relapsing nature of the disease not considered at all. Moreover, general practitioners, surgeons, and even dermatologists frequently know very little about this disease [11].

Primary lesions

The early lesions are solitary, painful nodules that may persist for weeks or months without any change or with occasional episodes of inflammation. Early isolated lesions are not specific and are frequently considered to be boils or common abscesses. The shape of these nodules, round rather than pointed and with no central necrosis, together with their topography should help to differentiate them from common boils. Their location deep in the hypodermis means they are sometimes barely visible. They are extremely painful. Approximately 50% of all patients experience subjective prodromal symptoms, which may include burning, stinging, pain, pruritus, heat, and hyperhidrosis, 12 to 48 hours before the overt nodule appears. The mean duration of a single painful nodule is 7–15 days. The nodules may remain blind, i.e., fail to burst, and either resolve spontaneously or persist as silent nodules with inflammatory recurrences. Most frequently, however, they turn
into abscesses (figure 1)\(^1\) and rupture externally, draining purulent material. Rupture into neighboring structures is rare.

**Secondary lesions**

The repetition of attacks may lead to chronic sinus formation, with intermittent release of serous, purulent, or bloodstained discharges and frequent foul odors from anaerobic colonization (figure 2). These persist for months, sometimes years. Ulceration or pyogenic granulomas sometimes occur. A characteristic secondary lesion is composed of bridged, ropelike hypertrophic scars resulting from a healing process involving dense fibrosis ((figure 3)). Tombstone comedos involving a single or multiple pores are frequently observed, usually as tertiary lesions in burnout regions. Closed comedos are not observed within HS lesions.

Regional lymphadenopathy does not appear, surprisingly in view of the inflammatory and infectious features observed [12]. Pathology specimens are usually not taken from patients with HS, as histopathology is not helpful for diagnostic and/or therapeutic purposes.

**Associated lesions**

Follicular papules and pustules are frequent in areas of HS as well as in other areas; they do not suggest the presence of HS. Epidermal cysts are prominent in some patients: round smooth elastic nodules, 1–6 centimeters in diameter, located on external genital organs, the face, or the thorax.

**Topography**

In decreasing order of frequency, the affected sites are the [8,11,13,14]:

- groin, including inner thighs;
- pubic region;
- scrotum or vulva;
- perianal and perineal regions;
- armpits;
- inter- and submammary folds, and;
- buttocks.

These locations are along the milk lines of apocrine and mammary tissue, which have the same embryonic origin. The groin and submammary region are most commonly affected in women, the buttocks and perianal skin in men; the anal canal is usually spared. Several sites, frequently symmetric, may be affected simultaneously; recurrences occur in and around the original site. Atypical locations are sometimes observed: the waist, abdomen, thorax, and especially the nape of the neck, as in severe acne or in acne keloidalis nuchae. There are frequently epidermal cysts in the retroauricular fold.

---

\(^1\) Pictures (Figures 1, 2, 3, and 4) show severe disease to illustrate typical lesions. Such severe cases are not representative of the patients encountered most frequently.
usually not inflammatory. These atypical locations are more frequent in men [14].

A pilonidal sinus is quite frequent (30%, and still higher in men), either as a true nodular and suppurating lesion that requires excision or as a small midline developmental depression of the upper part of the intergluteal cleft [14].

**Clinical course and severity grading**

Chronicity is the hallmark of HS. The mean age at disease onset is 22.1 years (± 8.2); prepubertal cases are extremely rare. For patients with pre-existing acne, the disease frequently starts after acne has disappeared. A questionnaire survey of English patients found a mean duration of active disease of 18.8 years [13]. Onset is earlier and disease usually milder in patients with a family history of HS. HS tends to become less active in women as they reach their 50s, with complete remission usual after menopause. Premenstrual flares are commonly reported. Pregnancy and breast feeding are usually periods of complete or partial remission. In men HS may continue to be active in old age [14].

Severity is generally assessed by the Hurley classification, which was designed for a surgical approach [15] (table I). Although most practitioners know only the most dramatic picture of severe disease, the mild forms are more frequent. In our personal series of 302 patients, the mildest grade – Hurley I – was diagnosed for 68.2% of the patients; intermediate severity – Hurley II – concerned 27.6%, and the most severe HS – Hurley III – 3.9% [14] (figure 4). Hurley’s grades are helpful for classifying a patient, or rather a localization (i.e., armpit or groin), in a category of severe or mild involvement. The index is not really helpful for managing medical treatment.

HS is polymorphous; its severity and course are vary widely between individuals.

**Mild cases**

This mild, yet chronic and painful, disease may be relentless and progressive or intermittent.

**Intermittent course**

Acute exacerbations alternate with complete spontaneous remissions, lasting several weeks or even months. These patients are very frequently misdiagnosed with furunculosis or considered to have recurring abscesses. Some of them can predict the occurrence of a new attack 24 hours before it occurs, because of such premonitory symptoms as pain, itching, and malaise. Acute exacerbations occur frequently as premenstrual flares (33/65 women experienced premenstrual flares according to Jemec [8]). Severity in these cases may be evaluated by the number, intensity, and duration of painful episodes per year.

**Continuous course**

Cases without remission may correspond to Hurley grade I or II. The disease is continuous; the main factors of severity are the number of locations involved, the extent of lesions in each area, the intensity of pain, number of days per month with pain, and suppuration. Clinical pictures vary widely. In any defined area, a new attack may be the recurrence of inflammation in a pre-existing fistula or the bursting of one or several new nodules/abscesses. Management differs in these two situations.
Severe course

Two different types of severe disease may be observed:
- permanent involvement of an area, with the formation of an infiltrated, painful, suppurating mass with coalescent nodules, fistulas, abscesses, draining sinuses, and fibrous scars. No normal skin exists between the lesions. The patient experiences pain, discomfort, swelling, malodorous discharges, and limited mobility. This is a typical Hurley grade III case, and radical surgery is the only reasonable treatment; alternatively the patient may have a multitude of nodules and abscesses separated by normal skin; each lesion lasts for 10 to 30 days, but they overlap, and new lesions appear in places where there was no previous lesion. Surgery is not helpful in this case; medical treatment is needed.

The following factors must be considered in assessing the global severity of disease:
- severity varies between various locations: the groin may be assessed as grade III, while the armpit is free of disease;
- HS rarely changes grade, e.g. from I to II or III: patients with mild disease remain mild and most severe cases seem to be severe since the start.

Quality of life

Quality of life (QOL) is profoundly impaired even in mild cases. A study of 114 patients with HS using the Dermatology Life Quality Index (DLQI) recorded a very high score, particularly for question 1, which measures the level of pain, itching, and soreness [16]. A study of 61 HS patients using several questionnaires showed that the impairment of QOL in each “dimension” was significantly higher ($p < 0.001$) than for patients with neurofibromatosis, urticaria, psoriasis, and atopic dermatitis [17]. This was particularly dramatic for self-perception, daily living activities, mood state, social functioning and physical discomfort.

Complications

Acute infectious complications such as cellulitis or systemic infection are very unusual. Lymphatic obstruction and lymphedema [18] as well as scrotal elephantiasis may complicate long-standing inflammation. In men, squamous cell carcinoma is observed in buttock areas after several decades of HS. It often starts in deep locations, so diagnosis is delayed and the prognosis usually very poor [19].

Historical complications that are rare today include both the complications of long-standing untreated disease, such as fistula formation into the urethra, bladder, rectum, or peritoneum, and the consequences of chronic suppuration, including anemia, hypoproteinemia, and amyloidosis. Similarly, peripheral or axial arthropathy, a previously standard complication, and SAPHO syndrome are not often encountered anymore. Chronic malaise and depression are frequent.

Associated diseases

Acne and the follicular tetrad

Histopathological studies demonstrate that follicular occlusion is an early feature of HS: it is considered the initial event of HS. However, because biopsies have not been performed in apparently normal skin close to HS lesions, we cannot rule out the possibility that this early follicular occlusion may in fact be a secondary phenomenon. Association of HS with severe nodular acne (acne conglobata), dissecting cellulitis of the scalp, and pilonidal cysts has been reported. Pilonidal cysts are reported especially frequently: $30\%$ if both true sinuses and midline intergluteal dimples are considered [14]. Moreover comedos are frequent in both acne and HS. Some authors have therefore proposed renaming HS “acne inversa” and including all four diseases in an acne tetrad [4]. Concomitant acne is, however, not frequent in HS ($20\%$ in men, $10\%$ in women); a past history of significant acne (long-lasting, leaving scars) is more
frequent, recorded in 44% of men 23% of women [14]. Dissecting folliculitis of the scalp is exceedingly rare (1%) [14]. A prospective study of 70 women with HS and 100 controls failed to find any difference in the prevalence of acne, hirsutism, or irregular periods between the two groups. Contrary to the initial lesions of acne, comedos in HS are never closed; they are secondary tombstone comedos, frequently double-ended open comedos (i.e. blackheads). Elevated sebum excretion, a major pathophysiological feature of acne, is absent in HS [20]. Moreover, effective acne treatments, such as isotretinoin and anti-androgens, are not effective in HS (see below). Hence, while HS may not be the best name for this disease, acne inversa is no better. HS should be retained until better knowledge of the pathophysiology helps to find a better name.

**Dowling-Degos-Kitamura diseases**

Reticulate pigmentation of the flexures is sometimes associated with HS. It is difficult to say if this is a significant association, as usually people do not seek medical advice for these pigment changes, which are discovered during examination of the groin and armpits of HS patients [21].

**Crohn’s disease**

Crohn’s disease [22] is both a differential diagnosis and an associated disease. Cutaneous Crohn’s disease must be differentiated from HS in its perianal location: the clinician must always be aware that the perianal lesions of Crohn’s disease may mimic HS and should perform the necessary workups. The atypical condyloma-like lesions seen in Crohn’s disease are not observed in HS; conversely, specific Crohn’s lesions are unusual outside the perianal region. It is nonetheless impossible to differentiate them on clinical grounds only. The presence of epithelioid granulomas in the dermis, away from the site of active inflammation, is suggestive, but biopsies are not frequently performed in patients with HS. When doubt persists, a colonoscopy must be performed, even in the absence of overt digestive symptoms. True associations do exist, including cases of axillary HS. Several case reports and a few series of this association have been reported [23]. Anti-tumor necrosis factor (TNF-α) drugs may be helpful in both diseases, which may both result from abnormal host-microbial interactions involving pattern recognition receptors (PPRs), such as toll-like receptors.

**Differential diagnoses**

Common abscesses, carbuncles, and furunculosis (recurrent common boils) are the main sources of diagnostic failure. Infected Bartholin’s gland, infected or inflamed epidermal cysts, lymphogranuloma venereum, scrofuloderma, actinomycosis, developmental fistulae, nodular acne with a pilonidal cyst, and Crohn’s disease are other standard sources of error. In fact HS is ignored more frequently than it is mistaken for another disease.

**Etiology**

The causes of HS are unknown; numerous hypothesis concerning tobacco, infection, genetics, and immunity have been raised and discussed [24].

**Pathology and gross morphology**

HS was initially described as a disease of the apocrine gland; histologic studies reveal that the earliest lesion observed in excised tissue is a follicular event with infundibular keratinization, which leads to a histologic image compatible with occlusion; inflammation (early lymphocytic infiltration followed by chronic inflammatory infiltrate containing histiocytes and giant cells related to keratin fragments) and necrosis of the sebaceous or sweat glands are secondary phenomena [25,26]. The similarity of keratin expression in the non-infundibular epithelium of HS lesions, keratinized or not, to that observed in the outer root sheath suggests that it plays a role in the pathogenesis of HS [27]. An ultrasonographic study of hair follicles in healthy skin of HS patients highlighted their wide diameter and distorted shape and their deep location [28]. Magnetic resonance imaging has been used in some case; it may be helpful in differentiating HS from Crohn’s disease or from subcutaneous infection and in preparing for large excisional surgery [29].

**Genetic factors**

Considering genetic factors [30,31], overall, 30–40% of HS patients report a related family history [14]. An autosomal dominant pattern has been reported, and a linkage to a locus at chromosome 1p21.1-1q25.3 [32] has been reported in one family but with no linkage in other families. HS appears to be a genetically heterogeneous disease with several mutations at various locations. No association was found with CARD15, which is linked to Crohn’s disease [33]. Specific genetic studies have yielded conflicting results. No significant HLA association has been reported [34]. Patients with a family history usually have milder disease with an earlier onset; this raises the question of heterogeneity of HS. Good clinical descriptions and the definition of clearly defined subsets of patients are necessary to improve genetic studies.

**Infection**

Traditionally, bacteria have been implicated in HS, and they play an important role in most overt clinical manifestations. They are not, however, the initial causative factor. Furthermore the absence of serious infectious complications (such as cellulitis) and of abnormal lymph nodes [12] rules out a major direct role for bacterial infection. A large variety of bacteria can be found, including streptococci, staphylococci, and *Escherichia coli* in the early stages. But superficial sampling is frequently sterile or finds bacteria from normal flora. Deep sampling is difficult and may be contaminated by superficial flora. It is
therefore impossible to rely on culturing lesions to adapt antibiotic treatment. During the chronic relapsing stages, anaerobic bacteria are present and at least partly responsible for foul odors [35].

**Hormonal factors**

Premenstrual flare-ups, the strongly female sex ratio, the frequent occurrence after menarche, and improvement during pregnancy have drawn attention to hormonal factors and led to the hypothesis of hyperandrogenic syndromes. However, this hypothesis is refuted by the general absence of clinical signs of virilism, the normal circulating androgen levels, the absence of hyperseborrhea, and the limited effect of antiandrogen treatments. Signs of virilization are unusual in HS. Most case series indicate no significant changes in serum hormone levels in HS patients [36]. The free androgen index may be elevated by a low level of sex-hormone-binding globulin, reflecting high body weight. No significant difference in androgen metabolism was noted in apocrine glands isolated from HS patients compared with matched controls [37]. Some facts remain unexplained, however: the high prevalence in women, the frequent improvement during pregnancy, and the usual permanent remission in women after menopause.

**Immunological and other host factors**

Although no gross anomaly of the adaptive immune system is seen, the association with Crohn’s disease, and the success obtained with anti-TNFα biologic agents has focused attention on the possibility that HS is related to impairment of the innate immune system. Minor alterations of NK cells have been found [38]. Enhanced TLR2 expression by infiltrating macrophages and dendritic cells has been shown in the diseased skin of patients with HS [39].

In view of the paradox that both anti-infectious (antibiotics) and pro-inflammatory (anti-TNFα, corticosteroids, and immunosuppressants) therapies may be helpful, HS appears to be an autoinflammatory disease based on a defect of the innate immune system. Future investigations should focus on Toll receptors, the cytokine network, and antimicrobial peptides in the hair and apocrine follicles.

**Drugs**

HS is not usually related to medications. It has been reported to be exacerbated by lithium [40]; some patients receiving sirolimus after transplantation have experienced the onset of de novo HS [41].

**Treatment**

**Severity grading**

Hurley defined the 3 standard clinical stages, as we saw above [15] (Table I). This classification is useful at least as a guide to choose between medical or limited surgical treatment (stage I) and wide-scale excisional surgery (stage III). The Sartorius score [42], a more precise scoring system, has been modified several times [43] and is widely used but not formally validated as an outcome measure for follow-up [44] (Supplementary data, figure 5). It is highly correlated to Hurley’s classification, to intensity and duration of pain and to suppuration, all good markers of inflammation and burden of disease, and to the extent of quality of life impairment. Pain scales and a quality-of-life questionnaire are useful as patient-oriented outcome measures. The frequency of flares also plays a role in the severity assessment of intermittent cases and may be analyzed as number of flares during a 3-month period.

**Therapeutic strategy: medicosurgical management**

Treatment depends on the stage (Hurley’s classification), frequency of exacerbation, and on the patient’s goal. A permanent cure can be obtained by wide surgical excision, but this procedure should be considered only in case of advanced disease, i.e., stage III or severe stage II. Early disease, on the other hand, may benefit from medical or medical and surgical approaches, simultaneously or successively [45].

**Acute stage treatment options**

Some patients suffer from recurrent painful nodules but also experience periods of remission. They may benefit from various options. Topical treatments, including antiseptics and antibiotics, are ineffective due to the depth of the lesions. A short course of systemic antibiotics may be tried for an acute, painful nodule, to shorten the duration of the episode and to abort it, avoiding development into an abscess. Various antibiotics have been used for this purpose. Amoxicillin + clavulanic acid may be the most effective regimen, if it is taken very early – within one hour of the first signs or premonitory symptoms. This means that patients must carry the drug with them at all times. A loading dose should be swallowed immediately (i.e., 3 g for a body weight of 70 kg) and followed by the same dose daily (divided up over the course of the day) for the next few days.

Intralosional corticosteroids (e.g., triamcinolone, 5–10 mg) have been advocated. Rapid involution (12–24 hours) of early lesions may be obtained [46]. High doses of systemic steroids may be used to reduce inflammation and pain. They can be used as an alternative to high-dose antibiotics or together with antibiotics [47].

**Emergency surgical incision**

For an abscess, i.e., when the lesion is fluctuating and full of liquid, medical treatment to abort it is ineffective; the required surgical drainage (incision) procedure should not be delayed. Incision of such an abscess is frequently performed in the emergency department. Usually the diagnostic of HS is missed. The incision is usually followed by packing; nursing in the days that follow and especially the renewal of packing are painful at
the least and often a nightmare for the patient. A limitation on the number of incisions and avoiding packing most of the time should improve patients’ quality of life. Packing is necessary in the case of a very deep cavity, concave area, narrow incision, or incomplete immediate evacuation. In those cases, it should not be changed too often and should be stopped as soon as it is dry [48].

**Chronic continuous intermediate course**

**Drug therapy**

Various drugs have been used on a long-term basis. Their goal is to stop the progress of the disease, reduce the relapse rate, and avoid pain and chronic suppuration. Due to the probable heterogeneity of the disease, some patients may benefit from a drug that is useless for others.

**Antibiotics**

For severely affected patients with a high level of inflammation, pain, and discharge, a ten-week regimen of clindamycin and rifampicin (600 mg/day each) [49–51] is very useful. Patients at Hurley stage I or II may benefit from it. A complete remission, sometimes lasting as long as one year, is obtained in some patients.

In less severely affected patients or after the ten weeks of clindamycin-rifampicin treatment, when inflammation and pain are reduced, maintenance treatment with tetracycline can prevent new attacks. Metronidazole may also be helpful, particularly in case of foul odors.

When surgery is indicated, a course of antibiotics beforehand may be useful to prevent infections, better delineate and identify the lesions to be excised, and effectively downsize the area to be excised.

**Antiangrmons**

Cyproterone acetate at 2 mg/day, combined with estrogens as a contraceptive pill, has been compared with a standard birth-control pill; no difference was observed between the two products [52]. It may therefore be useful at very high doses (e.g., 100 mg/day) in a very limited number of patients. Finasteride has been used in about ten female patients, with good results reported [53,54].

**Retinoids**

Unlike in acne, isotretinoin is ineffective in HS [55,56]. The absence of hyperseborrhea in HS may explain the difference. Some reports of successful treatment with etretinate or acitretin have been published (reviewed in [57]).

**Dapsone**

Dapsone has been used with good results [58]. It must be used cautiously in view of its very serious side effects.

**Anti-TNFα drugs**

Several case reports and short series have reported dramatic efficacy for infliximab, etanercept, and adalimumab in severely affected patients (reviewed in Haslund et al. [59]). Beyond the initial enthusiasm for new “magic pills”, the real risk/benefit ratio remains to be determined, given the usual positive publication bias and the conflicting results: only transient efficacy was observed with infliximab in a series of seven patients, where only two patients had sustained results and three had severe side effects [60]. A case report of lethal squamous cell carcinoma after treatment with infliximab has been published [19]. A prospective open study of 15 patients treated with etanercept 50 mg weekly found only three responders [61]. A small randomized controlled study has shown some results despite the negative findings for the main outcome measure [62]. These findings require us to ask: are TNF-alpha blockers the ultimate alternative [63]? It is obvious that these drugs may be helpful and are sometimes dramatically effective. Today, they are used when the standard very effective antibiotic association of clindamycin and rifampicin fails. A head-to-head comparison of anti-TNFα drugs and this antibiotic combination is highly desirable.

**Radiotherapy**

Several series of patients have been treated with radiotherapy at doses up to 8 gray. Considering the high spontaneous cancer risk, especially for skin cancers in gluteal locations, this potentially carcinogenic treatment should be considered with caution [64].

**Surgical treatment**

Surgery [65] must be performed by an experienced surgeon, aware of the difficulties and failures which may occur in HS. The proper identification of the entire lesion is important as remnant tissue may facilitate recurrences. High-frequency ultrasound examination of the skin or magnetic resonance imaging or both may be helpful. The major element for a good and permanent result is the mapping of sinus tracts and fistulas, usually done during the surgery.

**Minor procedures**

**Local excisions and primary closure**

In patients at Hurley stage I and a permanent or recurring draining sinus, this procedure is extremely helpful and causes a minimum of distress for the patient, as it can be done as an outpatient under local anesthesia. Each time a lesion recurs in the same location, limited excision should be considered. A study of 72 patients treated by excisional surgery of localized lesions reported high patient satisfaction despite recurrences [66]. It is not helpful when many burrowing abscesses are present or when new lesions regularly appear in different locations.

**Exteriorization and deroofing of tracts**

Exteriorization and deroofing of tracts may be an alternative therapy [67].

**Radical excision and healing with secondary intention or graft**

This is the best option – in fact the only option – in stage III. A wide excision should be performed by a surgical team with a
good knowledge of HS. To prevent recurrence, the extent of excision must be wide enough and deep enough to remove all suppurating lesions, tracts, and, if possible, all apocrine gland-bearing skin. Intraoperative mapping of sinus tracts with methyl blue is essential. Spontaneous healing is frequently the best solution except in the axillae, where it may limit arm mobility. Secondary closure is done by graft or flaps. A provisional colon-derived graft may be necessary to allow healing in perianal locations. Recurrences may occur either because of insufficiently wide excision or because of the presence of subclinical predisposing structural or functional abnormalities, i.e., apocrine follicles in aberrant locations. The recurrence rate after wide excision is less than 30%. Wide surgery does not modify the spontaneous disease course. Reappearance in another location not previously involved is not a recurrence!

**Lasers**

CO2 laser excision is used in mild to moderate disease with secondary healing. Superiority in comparison with standard surgery is a matter of debate [68,69]. Laser depilation to prevent new lesions is an investigative procedure in early mild HS.

Nd: YAG laser treatment was used in a controlled right-left comparison of patients with Hurley II disease with good results. The remodeling effect on the dermis may be a more important mechanism of action more than the depilatory effect [70,71]. Botulinum toxin injections have been used with claimed good results [72]. Photodynamic therapy has been disappointing. The deep lesions of HS do not seem to be good targets for this kind of superficial treatment [73].

**Conclusion**

HS is an orphan disease, not because of its rarity – for it is frequent – but because physicians know too little about it. Treatment is not curative, except for wide surgery, appropriate only for very severe cases; good management, using standard well-known treatments, is often more useful than the so-called innovative treatments, whose risk/benefit ratios are unknown today.

Conflicts of interests: None.

**Supplementary material available on La Presse Médicale**


Figure 5: Hidradenitis suppurativa score (Sartorius score) (© Karin Sartorius and Jan Lapins 2008)

**References**


[36] Brunasso AM, Delfino C, Massone C. Hidradenitis suppurativa: are tumour necrosis


