Hidradenitis suppurativa: pathogenesis and management

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Abstract

Hidradenitis suppurativa (HS) is a chronic disease manifested by recurrent abscesses, sinus tracts and scarring. It arises most commonly, however, not exclusive from apocrine gland bearing skin. This review article sets out to clarify the importance of early diagnosis, the pathogenesis and aetiology of HS, and evidence for medical and surgical therapies for this debilitating disease.

HS is caused primarily by follicular occlusion with secondary involvement of the apocrine glands. The aetiology is still poorly understood. There is a genetic component with probable hormonal influence on gene expression. Shearing forces from obesity and tight clothing contribute to its development.

Management should be appropriately tailored for the severity and distribution of HS as well as quality of life of the patient. Medical management with appropriate antibiotics, if initiated early, can be successful in mild to moderate severity HS as well as improving disease control prior to attempted curative surgery in severe HS. Other helpful measures include advice on
lifestyle changes, intralesional steroids, systemic retinoids, hormonal manipulation, and a revival of interest in the use of radiotherapy for HS.

While there is a place for 'conservative' surgical procedures (including CO₂ laser) in selected cases of mild to moderate HS, radical excision of all apocrine-bearing tissue is the definitive treatment. We advocate close interdisciplinary collaboration as well as a cautionary approach to timing and planning of surgery to minimise recurrence rates.

**Author Keywords:** Hidradenitis suppurativa; Apocrine gland; Pathogenesis

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The term hidradenitis suppurativa (HS) is a misnomer. While there is no denying its suppurative nature, HS is almost certainly not a disease initiated by infection and is probably not a condition primarily affecting (apocrine) sweat glands.[1] There are many myths
associated with HS, such as its postulated association with diabetes mellitus, poor hygiene, deodorant and chemical depilation. HS is, however, associated with obesity. HS is highly debilitating for sufferers, both physically and psychologically. Patients experience recurrent painful abscesses and malodorous discharge necessitating regular dressings. This can lead to social isolation, failed relationships and depression. The condition is thought to be under-reported, as patients feel embarrassed to seek medical advice. Web-based organisations such as H.I.D.E (http://members.ncbi.com/capiii/indexfyl.htm) provide a useful forum for patients to update and share information on medical advances, lifestyle tips as well as mutual support.

Because of the varying clinical manifestations and sites involved by the disease, patients with HS present to, or are referred to many different specialities, including gynaecology, surgery, medicine, dermatology, plastic surgery, immunology and infection control. Unfortunately, HS is commonly mismanaged owing to a failure of early diagnosis and once established, chronicity and progression ensue. No specific test for diagnosis exists. [1] Inflammatory abscess-like swelling(s) in apocrine gland bearing skin should be regarded as possible HS. The clinician should also bear in mind the other possible sites of HS, which may have no, or little apocrine component. In order of frequency of involvement, the following sites are recognised: axillary, inguinal, perianal and perineal, mammary and inframammary, buttock, pubic region, chest, scalp, retroauricular, eyelid skin.

There is little evidence for treatment efficacy from randomised controlled trials or from long-term follow up. Three prospective randomised controlled trials of medical management exist. First, topical clindamycin significantly improved the symptoms of HS patients when compared with topical placebo. [2] Secondly, there was no symptom improvement in HS patients treated with either systemic tetracycline or topical clindamycin. [3] The third showed that antiandrogen therapy using cyproterone acetate significantly improved the symptoms in female HS patients as well as decreasing their free androgen index. [4] In the surgical literature, a controlled prospective trial of bilateral axillary HS excision randomised to either split skin graft (SSG) or silastic foam dressings (healing by secondary intention) showed that the latter achieved good cosmesis, healing and patient acceptability. [5]

Early diagnosis and close collaboration between the dermatologist and the surgeon can lead to effective control and prevention of development of advanced disease. Where the disruption of normal skin architecture is severe, complete resolution is impossible. In these cases, surgery in the form of radical excision (with or without skin replacement) offers the only effective treatment. [6] Surgery of this magnitude is associated with significant morbidity and the possibility of local (inadequate primary excision), or distant, recurrence. This review will discuss:

- Diagnosis and clinical features
- Epidemiology
- Pathology and pathogenesis
- Aetiology and bacteriology
- Medical management

- General
- Antibiotics
- Hormonal therapy including finasteride
- Retinoids
- Immunosuppression
- Radiotherapy
Surgery

Simple drainage
Limited excision and primary closure
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Radical excision and cover with local and distant flaps
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Complications
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1. Diagnosis and clinical features

Tender subcutaneous nodules (0.5–1.5 cm diameter) frequently develop first around the time of puberty. In women, the condition frequently flares premenstrually and following pregnancy but may ease during pregnancy and after the menopause. Obesity, old or active acne, comedones and hirsutism are associations of significance.[7]

Characteristics of hidradenitis suppurativa

- **Age of onset:** puberty
- **Sex:** female to male ratio of 3:1
- **Associations:** obesity, acne, comedones, and hirsutism
- **Sites affected in order of frequency:** axillary, inguinal, perianal and perineal, mammary and inframammary, buttock, pubic region, chest, scalp, retroauricular, eyelid

The abscesses are deep and round without pointing or central 'necrosis' (unlike staphylococcal furunculosis). In the beginning, axillae and/or anogenital regions are involved usually with remarkable symmetry [Fig. 1]. These lesions may either resolve, in milder cases or generally, progress within hours to days to yield purulent or seropurulent drainage on to the skin. Some cases involve only one site and remain mild or even undergo spontaneous remission. New lesions in adjacent skin may develop and if uncontrolled, will lead to chronic inflammation, sinus formation, 'bridged' scarring and 'tombstone' comedones involving complete apocrine areas. With more severe disease lesions develop in other sites, particularly sites of shearing such as submammary regions (in women) (Fig. 2 and Fig. 3), nape of neck (Fig. 4), waistband and inner thighs.[1] These are the areas not commonly thought of as being associated with HS (Fig. 5 and Fig. 6).

Fig. 1. Right axillary hidradenitis showing sinus formation, acute inflamed nodule and scarring.
Anogenital involvement most commonly affects the groins with extension to the inguinal
regions, mons pubis, vulva, inner thighs, and sides of the scrotum. The perineum, buttocks and perianal folds are often involved (Fig. 7). These sinuses can dissect into tissues involving muscle, fascia, bowel, and urethra forming a labyrinth of tracts in advanced cases. Perianal HS, in particular, may present with pain, swelling, purulent discharge, pruritus or bleeding thus mimicking the presentation of Crohn's, anal fistula, pilonidal sinus or perianal abscess.[8] A group in the Cleveland Clinic reviewed 61 patients with perianal HS and noted that Crohn's disease coexisted in 24 (39%) of the cases. This coexistence is not really understood but it does have implications for the management of perianal sepsis. First, each condition may mask the other and inappropriate treatment may be given, and secondly, the clinical course seems to be more severe in combined cases. Proctectomy was required in 70% and faecal diversion in 91% of patients suffering from coexistent disease in the Cleveland series.

Fig. 7. Severe perineal hidradenitis.

Differential diagnoses include carbuncles, lymphadenitis, infected Bartholin's or sebaceous cysts. Uninflamed cysts, alone or as part of the steatocystoma multiplex syndrome, should be looked for. Tuberculosis, actinomycosis, cat-scratch disease, lymphogranuloma venereum, granuloma inguinale, and ulcerative colitis are sinus and fistula-producing diseases, which should be excluded in apocrine regions in which they may occur.

A series of conditions which bear an aetiological resemblance to each other have previously been known as the follicular occlusion triad comprising (a) acne conglobata, (b) dissecting cellulitis of the scalp (perifolliculitis capitis) and (c) hidradenitis suppurativa.[9] Acne conglobata is a severe manifestation of acne that involves the chest, back and buttocks. Comedones, together with multiple areas of small purulent nodules, are prominent features. Perifolliculitis capitis is a similar process affecting the scalp. Fistulous tracts and scarring in discrete areas lead to patches of alopecia. Most recently pilonidal sinus was included to form a tetrad. [10] Patients manifesting any combination of this follicular occlusion tetrad may be encountered.

2. Epidemiology

A point prevalence of 4.1% was found.[11] However, this has been suggested to be falsely elevated as a select (at-risk) age range was studied. [12] A prevalence estimate of 1:300 was found by another group. [13]

Women are affected three times as often as men. The reason for this preponderance is unknown.[11, 14, 15 and 16] The condition has a peak incidence in the second and third decades, coinciding with the postpubertal increase in androgen levels, declining rapidly by the fifth decade. In women, HS may persist into the climacteric, but onset after menopause is rare. The belief that axillary disease is more common in women and ano-genital disease is more common in men has recently been refuted. [11] The disorder rarely occurs before puberty. However, the rare onset in neonates and infants may reflect the relatively hyperandrogenic state in the first year of life. [15 and 17]
Some authors believe that HS is more common in patients of Afro-Carribean origins[18 and 19] and others do not believe there is any racial predilection. [20] A tropical environment favours the development of HS but it has been seen in many different parts of the world under different climatic conditions. [21, 22 and 23]

3. Pathology and pathogenesis

Apocrine glands are compound sweat glands, which extend down through the dermis into the subcutaneous tissue. Each gland consists of a deep-coiled secretory component, which drains via a long straight excretory duct, into a hair follicle. Secretion from these glands is usually opalescent and malodorous due to the effects of surface bacteria.[24] Apocrine glands are present in the axilla (most commonly), anogenital region, periareolar, submammary, periumbilical, scalp, zygomatic and malar areas of the face, the buttocks, thighs and popliteal fossa. [19] (See below: Glandular elements of skin.)

Glandular elements of skin

- **Eccrine glands:** present all over skin but concentrated on palms, soles and axilla. Small, superficial, tubular structures that drain directly on to skin surface. Primary function is temperature regulation.
- **Apocrine glands:** mainly concentrated in axilla, groin and smaller nests in perineum, perianal, mammary, pubis, scrotum, labiae, periumbilical, ear canal (ceruminial glands) and eyelids (Moll's glands). Compound sweat glands with secretory coil that extend deep through dermis into subcutaneous tissue and drain via a duct into a hair follicle. Probable vestigial origin and responsible for axillary and inguinal odour.
- **Apoeccrine glands:** present in axilla and combines features of apocrine and eccrine glands with a deep secretory coil and a straight intradermal duct which opens directly on to skin.

HS was first described as a distinct entity by Velpeau in 1839. [25] He reported a patient with inflammatory disease affecting the skin of the axilla, mammary and perianal areas. Verneuil, in 1854, described a series of patients with similar lesions and assigned them to diseases of the sweat glands. [26] However, it was not until 1922 that there was an association made between HS and apocrine glands. [27] The anatomical distribution of the disease suggested that it is a primary disorder of the apocrine glands, and the term 'apocrinitis' had been employed as a synonym for the condition. Indeed the presence of distended apocrine glands containing polymorph neutrophils in the subcutis, was demonstrated in an early description of the disease by Brunsting in 1939.[9] Shelley and Cahn in 1955[28] applied perforated belladonna adhesive tape to depilated axillary skin and produced lesions at the tape site which clinically resembled HS in three out of 12 healthy males. Although it does not represent an accurate model of the disease, [29] it lent some support to the initiating event in the disease being poral occlusion in the apocrine ducts. More recent publications have shown that the primary event is follicular occlusion by keratinised stratified squamous epithelium. [29] HS would therefore appear to be a disorder of follicular rather than apocrine occlusion. The work by Attanoos and coworkers, [17] in particular, showed that regardless of disease duration (1 month to 18 years), follicular occlusion was an early and important feature in the pathogenesis of the disease. Comedones are a conspicuous feature of the disease underlining the follicular occlusion. It was also demonstrated that in the majority of cases active folliculitis was associated with apocrinitis and apocrine destruction, whereas apoeccrine glands (present in axillae), which drain directly on to the
epidermal surface, appeared intact and noninflamed. Therefore, HS is better considered as a disorder of terminal follicular epithelium within apocrine gland bearing skin. Follicular occlusion leads to dilatation followed by rupture, spilling contents, including keratin and bacteria into the surrounding dermis. This induces a vigorous chemotactic response with an inflammatory cellular infiltrate consisting of neutrophils, lymphocytes and histiocytes. Abscess formation develops, leading to the destruction of the pilosebaceous unit and eventually the other adnexal structures in the vicinity. Epithelial strands are generated possibly from ruptured follicular epithelium to form sinus tracts. Histologically, chronic disease shows a dermis that contains inflammatory cells, giant cells, sinus tracts, subcutaneous abscesses, and later, extensive fibrosis. Sinus tracts are the hallmark of established disease and the one feature most resistant to treatment.

4. Aetiology and bacteriology

The exact aetiology of HS is unclear. There were no significant differences in the size and density of the apocrine glands in HS compared with normal controls.[30] Several factors including use of deodorants and depilatory products, as well as shaving are often discussed to be causal factors. However, a retrospective comparison of 40 HS patients with 40 age-matched controls found no significant difference in the use of these items. [31] Obesity is unlikely to be causal but may be an exacerbating factor. [32] It is considered important in HS, possibly by increasing shearing forces of skin and possibly androgen effects. Weight loss can therefore help control the disease. [1] Keratin hydration is thought to be enhanced in sweat gland-rich regions of the body, and this has been shown to favour occlusion. [33] Genetics and endocrine factors may be contributory. Patients frequently report cases among their relatives. In one study 18 out of 70 patients (26%) with HS had a positive family history, whereas 96 control subjects matched for age and sex, who did not have HS, only two of their relatives suffered from HS. [11] There appears to be autosomal dominant inheritance with single gene transmission. [13, 34, 35 and 36] A recent study supports the concept of a familial form of HS with autosomal dominant inheritance. [36] A variable degree of gene penetrance and possibly hormonal influence on gene expression may explain the reduced risk to first-degree relatives, which falls short of the expected 50% found in that study. There is an apparent strong influence of sex hormones on HS (association with acne vulgaris comedones and hirsutism, development post puberty, general decline in disease activity seen at the climacteric, and improvement seen during pregnancy [7, 37 and 38] However, other authors have disagreed with these findings. [14] Most HS patients have normal androgen profiles [1 and 16] and apocrine glands, unlike sebaceous glands, are not androgen sensitive. [39] Nevertheless, there have been reports of symptomatic improvement with the use of antiandrogen therapy. [4, 40, 41, 42 and 43] Smoking in patients with HS was found to be significantly higher (88.9%) than a matched-pair control group (46%) of nonHS sufferers.[44] Smoking induces altered chemotaxis of polymorphic neutrophils which possibly plays a role in aetiology, as already postulated for palmoplantar pustulosis. [45 and 46] There was an association between cigarette smoking and perianal HS in 70% of patients in one series. [20] It seems reasonable that smoking cessation should therefore be strongly encouraged in patients with HS.

4.1. Microbiology

The significance of bacterial findings in HS is controversial.[47] While bacteria are likely to be involved in the pathogenesis, it is probably in a role similar to acne. Sepsis is unlikely to be
causal and pus is usually sterile in the early stages. In the later stages of the disease, bacterial infection seems to be a risk factor for the destructive scarring and extension of the lesions. Negative cultures from superficial HS lesions are common. In most studies of HS, samples for bacteriological culture are collected from the surface of the lesions. In one study, *Streptococcus milleri* was the most frequently isolated bacterium in HS and *Bacteroides fragilis* and *B. meleninogenicus* were anaerobic bacteria frequently isolated by another group. Chlamydia trachomatis has been reported in active ano-genital HS. Surface bacteriology has obvious drawbacks, as contamination from resident bacterial populations obscure the interpretation of the culture result. This problem was partially circumvented by an aspiration technique aiming to sample deeper parts of HS. Cultures taken from early HS lesions (to increase the likelihood of identifying causative organisms) were negative in half the cases. The authors concluded that HS was primarily a disease of follicular epithelium secondarily colonised and infected by bacteria. The carbon dioxide laser method used to treat HS yielded a sufficient bacterial count for culture in deeper parts of HS, apparently minimising resident skin bacteria interference. They showed that bacterial growth is frequent in the deeper parts of HS but sepsis was uncommon. The most commonly isolated bacteria were coagulase negative staphylococcus (64%) and *Staphylococcus aureus* (24%). The next most commonly isolated species were *Peptostreptococcus* species and *Propionibacterium acnes*.

### 5. Medical management

Evidence proving efficacy for medical treatment is very limited and current practice aims to control early or milder forms of the disease. Parallels with acne have resulted in the development of similar approaches, with limited success.

#### 5.1. General

As already discussed, advice for weight loss in obese patients as well as cessation of smoking may be useful. General measures include antiseptic soaps, tea tree oil, cotton underwear, loose clothing (including avoiding tight jeans), soft/friction free brassieres (or no brassieres at all) and hold-up hosiery in preference to tights. Patients may have their own supply of dressings.

#### 5.2. Antibiotics

Anti-acne antibiotics, if used early and aggressively, e.g. oral clindamycin 300 mg bd, can be effective and result in remission but relapse is common on discontinuation. This however, may be an important adjunct to surgery, reducing the possible risk of infection or skin graft failure. Clindamycin as a topical preparation has been used with success. This study had 13 patients who received the active treatment and the authors admitted that the material might not be comparable to HS in general (disease activity was low and the disease location was predominantly in the groins alone). Clindamycin has also been shown to be effective in anaerobic organisms isolated in HS. Topical clindamycin was compared with systemic tetracycline in stage 1 or 2 HS in a double blind, double-dummy controlled trial. There was no difference in the results between the two treatments. When specific pathogens are isolated, e.g. anaerobes, then it make sense to target treatment with the appropriate drug, but the duration of this treatment is unknown and relapse rates are high.
5.3. Hormonal therapy

Hormonal therapy using antiandrogen cyproterone acetate and ethinyloestradiol proved to be useful in a randomised controlled trial[4] but continued use raises concerns over safety due to the high doses required. Finasteride (Proscar, Merck Sharp and Dohme, Hoddesdon, UK) is a competitive inhibitor of 5-alpha reductase which converts testosterone to the potent dihydrotestosterone. It was initially manufactured for the treatment of benign prostatic hypertrophy but patients noticed an effect on hair growth. Finasteride was approved by the FDA (1997) for the treatment of androgenic alopecia in men. The main side effect is impaired androgenisation of the male foetus and is therefore, contraindicated in women of childbearing age. It has recently been used to treat two patients with HS inducing short term (1 month) remission.[54] The authors are investigating the expression of 5-alpha reductase type I and II in the skin of HS sufferers.

5.4. Retinoids

Isotretinoin has been successful in the treatment of acne[55] and therefore, considered for the treatment of HS. The results in HS have, however been less encouraging. There has been no randomised controlled trial to our knowledge and studies show that oral isotretinoin has little effect on HS. [56] However, acitretin 25 mg bd does seem to be effective. [57] This is thought to be because isotretinoin has maximal effect on sebaceous gland activity but as sebum production is normal in HS, [58] it is unsurprising that the treatment is ineffective. Acitretin acts more on keratinisation and therefore may be more appropriate for HS. Use of oral isotretinoin during the weeks or months before and after surgery has been recommended, as it has anti-inflammatory effects. [56]

There is early, unpublished work studying the effects of cimetidine on HS. Histamine receptors are found in the skin and the drug has well documented anti-androgenic effects.

5.5. Immunosuppression

Systemic steroids are often used with initial benefit due to the nonspecific anti-inflammatory effects but relapse may be more difficult to control when steroid dosage is reduced particularly if infection is present. Intralesional steroids (e.g. Triamcinolone acetonide suspension) can be useful for localised disease but administration can be difficult when it escapes via the sinuses. There is an anecdotal report of benefit in HS using cyclosporin.[59]

5.6. Radiotherapy

A recent German publication retrospectively reviewed 231 patients who had undergone radiotherapy for the treatment of HS with ‘complete relief of symptoms’ in 89 (38%) of patients and ‘clear improvement of symptoms’ in 92 (40%) patients.[60] Further research is required.

6. Surgical management

There is no evidence that treatment other than surgery has any effect on the natural history of
severe HS. However, surgical management should be in a multidisciplinary setting allowing optimisation of the condition pre- as well as postoperatively. This collaboration, we feel, is essential in the successful management of HS.

Plastic surgeons are guilty of the fact that too much emphasis has been placed on the technique used to cover the defect rather than adequate excision margins and good long-term outcome (i.e. recurrence rate). Good reports on relative cure rates of different surgical options are sadly lacking and controlled trials almost nonexistent. The largest surgical survey of HS reports the following rates of recurrence in 82 patients treated with 118 radical excisions: axillary, 3%; perianal, 0%; inguinoperineal, 37%; and submammary, 50%. Recurrence occurred 3–72 months following surgery. The conclusions were that recurrence occurred from either inadequate excision or (as in the case of submammary recurrence) an ‘unusually wide distribution of apocrine glands’. Other studies have showed that extent of surgical excision influenced recurrence rates more significantly than the method of wound management. [62, 63 and 64] The attempt to obtain primary skin cover may lead to inadvertent compromise of the excision margin and an increased risk of recurrence. [61] However, the use of intraoperative marking may be a helpful tool. [64] Recurrence can occur in distant apocrine gland bearing sites, and patients should be warned of this possibility. The surgical options and their pro and cons will be discussed.

6.1. Simple drainage

Incision and drainage of individual lesions may lead to temporary relief but recurrence is inevitable. [65]

6.2. Limited excision and primary closure

Where the extent of skin involvement is limited, local excision and primary closure is a method advocated by some authors, giving lower morbidity but a higher recurrence. [6 and 62] A specific technique of excision and primary closure in the management of axillary disease has been described. [66] Minimal undermining of the wound edges was undertaken. Skin closure incorporated the use of subcuticular and interrupted vertical mattress sutures in addition to nylon retention sutures tied over a bolus dressing.

6.3. Laying open of sinus tracts

Deroofing and marsupialisation of the sinus tracts may be of benefit but recurrences do occur. [6 and 67] Careful laying open of all tracts, possibly as a staged procedure according to geographical distribution is an option which clinicians and patients alike see as an appropriate compromise between extremes of surgical intervention. [1]

6.4. Radical excision and healing by secondary intention

Complete excision of axillary and perineal skin is often needed to prevent recurrence. [21] The extent of excision is variable in the literature, accounting for the differences in recurrence rate as already discussed. The block of tissue, however, should not only be adequately excised in width but also in depth. Excision of subcutaneous tissue down to deep fascia, or at least excision of a minimum of 5 mm of subcutaneous fat ensures that deep coils of apocrine glands are removed. [24]
A good method of obtaining complete clearance is to visualise the apocrine glands completely.[30 and 64] Using the iodine/starch/oxytocin method excision of 11 × 8 cm of axillary tissue was required in order to eradicate the main bulk of the apocrine glands. This measurement, in the contracted skin specimen, represents a much larger skin defect, which cannot be repaired primarily. [5] These authors are proponents of healing by secondary intention using silastic foam (Dow Corning, Reading, UK). [30] Although healing is quicker following split skin grafting, patients are more comfortable with the silastic method allowing early discharge back to the community, very little limitation in movement, and apparently, a surgically acceptable scar, superior to that obtained by split skin grafting. Patients (7/10) preferred the silastic dressing method to SSG. Most importantly adequate clearance was achieved which was reflected in their low recurrence rates.

6.5. Radical excision and SSG (+/- negative pressure dressings)

Large defects can be covered by immediate or delayed SSG.[68 and 69] The proponents for delayed SSG believe that haemostasis is better achieved hence, less chance of graft failure. On the other hand, with the advent of meshing, many surgeons believe that immediate grafting decreases the chances of bacterial contamination of the wound. [68 and 70] Negative-pressure dressings are particularly useful in the treatment of contoured wounds needing closure with skin grafts. In particular, it is ideal for complex anatomical regions such as axillae, where conventional skin graft stabilisation techniques are cumbersome and ineffective. A recent study has used vacuum assisted closure (VAC) coupled with SSG for large, complex open wounds with 95% graft take in all patients.[71]

6.6. Radical excision and cover with local or distant flaps

This is not an exhaustive account of reconstruction techniques, suffice to say that local flaps, fasciocutaneous flaps, pedicled flaps and free flaps have been described in the literature for defect closure in HS surgery.[18, 63, 70, 72, 73, 74, 75, 76, 77 and 85]

6.7. Laser treatment

An exciting therapeutic advance is carbon dioxide laser treatment, with healing by secondary intention, which provides a rapid, efficient and economical treatment of HS. Laser treatment is suitable for those with mild to moderate/severe disease without the need for hospitalisation. Early return to work was reported,[47 and 86] and the range of complete healing time was between 3 and 5 weeks. This however, was not the experience of the senior author whose two patients required 4 months off work each (pain being a major problem). Other advantages reported include improved haemostasis, better visualisation and therefore a more accurate assessment of diseased tissue to be excised. [78] Patients reported less scarring and pain than that of previous surgeries or the disease itself. [79]

7. Complications

Fibrosis and scarring can lead to contractures and decreased mobility of limbs, particularly, in the axilla. Fistula formation; anal, rectal or urethral, can occur with anogenital disease.[23] Anaemia can be associated with HS. [80] Squamous cell carcinoma is a rare consequence of
chronic HS but is only described in the anogenital region. [81] Hypoproteinaemia, and amyloidosis, rarely can progress to renal failure and death. [82] Arthropathy associated with HS can present with variable clinical features, ranging from asymmetrical pauciarticular arthritis to a symmetrical polyarthritis/arthralgia syndrome. [83] There seems to be an association between HS disease activity and arthropathy activity. [84]

8. Conclusions and suggestions for the future

HS is a complex and debilitating disease, which is still poorly understood. The control of sinuses is probably the key to the condition. No medical treatment resolves sinuses once formed and only radical surgery may succeed in its ability to eradicate sinuses. Early recognition, collaboration between dermatologist and surgeon and good uncompromising surgical principles can lead to effective control. Common sense tells us that surgery is likely to be more effective if undertaken when inflammation/infection is less severe, however it is often undertaken when disease activity is greatest. Our main recommendations for effective management are early diagnosis, control of the disease prior to surgery and careful surgical planning.

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