

Hidradenitis suppurativa

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Background: Hidradenitis suppurativa is a chronic, recurrent, suppurative cutaneous disease. Despite its incidence, optimal medical or surgical treatment remains unclear. This review describes the disease, ranging from pathogenesis to treatment and prognosis.

Methods: Articles were sourced from PubMed and Medline, using the MeSH terms 'hidradenitis suppurativa' and 'acne inversa'. Selection of articles was based on peer review, journal, relevance and English language.

Results and conclusion: On the basis of histological findings, the disease is now considered inflammatory and originating from the hair follicle; therefore, the term 'acne inversa' is favoured by some experts. The exact aetiology remains obscure but smoking seems to be a major triggering factor. Treatment should be individualized according to the site and extent of the disease. Absolute cessation of smoking is essential in the treatment of hidradenitis. Management with antibiotics or other medications may relieve early symptoms, but radical surgery may be necessary for control and to prevent recurrence.

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Introduction

Hidradenitis suppurativa is a chronic, recurrent, suppurative, cutaneous disease, manifested by abscesses, fistulating sinus tracts and scarring¹. Synonyms are apocrinitis, acne inversa and pyoderma fistulans significa.

The disease is expressed by a variety of clinical features, and many sites may be involved. It affects apocrine gland-bearing skin; the axillae are most often involved, followed by the anogenital region. Uncommon sites include the areola of the breast, the submammary fold, the periumbilical skin, the scalp, the zygomatic and malar areas of the face, the buttocks, the thighs and the popliteal fossa². Patients affected may present to family physicians, general surgeons, gynaecologists, urologists, infectious disease consultants, dermatologists or plastic surgeons.

The exact incidence of hidradenitis suppurativa is uncertain, but the literature suggests from one in 300 to one in 600. The peak onset is between ages 11 and 30 years, whereas postmenopausal onset is rare³. Early onset in neonates or infants suggests a relatively hyperandrogenic state in the first year of life or a presenting feature of premature adrenarche^{4,5}. This condition occurs in both

sexes, but females are affected three times as often as males^{6,7}. It occurs in all races, but its incidence is higher in blacks than in Caucasians^{8,9}.

Pathogenesis

The human body has two different types of sweat gland, eccrine and apocrine glands. Eccrine sweat glands, which are smaller than apocrine glands, are distributed all over the body. Their main function is control of body temperature. The eccrine gland is a simple tubular epithelium consisting of a duct and a secretory portion. Apocrine sweat glands are mainly distributed in the axillary and anogenital skin. They represent remnants of an odour-producing organ found in lower animals¹⁰. The apocrine gland extends through the dermis into the subcutaneous tissue. Each gland consists of a deep coiled secretory component that drains via a long straight excretory duct, usually into a hair follicle¹¹.

The exact pathogenesis of hidradenitis suppurativa remains unclear. The anatomical distribution of the disease suggests that it is primarily a disorder of apocrine glands. The most accepted hypothesis is that the initiating event is occlusion of the apocrine or follicular ducts

by keratinous plugging, leading to ductal dilatation and stasis in the glandular component. Bacteria reach the apocrine gland through the hair follicle, are entrapped under the keratinous plug, and multiply rapidly in the nutrient environment of the gland. Eventually the gland will rupture, leading to inflammation extending into the surrounding tissue and adjacent glands. Bacterial infection with staphylococci and streptococci results in further local inflammation, tissue destruction and skin damage^{4,12–16}.

The theory of follicular occlusion was investigated in 1955 by Shelley and Cahn¹⁷. In an experimental model, poral occlusion was induced by manual skin depilation and application of atropine-impregnated tape. The observed changes consisted of keratinous plugging, with secondary dilatation and inflammation, followed by bacterial invasion of the apocrine duct. However, occlusive lesions were achieved in only 25 per cent of subjects, and the lesions did not progress to the traditionally chronic condition of hidradenitis suppurativa^{1,18}.

In another study, the axillary skin of patients with the disease was compared with that of a control group. After histological examination the authors concluded that the condition was a disease of follicular epithelium rather than a disease of apocrine glands¹⁸. The histopathological findings suggest that squamous epithelium-lined structures, taking the form of cysts or sinuses, are a more consistent feature of hidradenitis than inflammation of apocrine glands (present in one-third of specimens). The presence of hair shafts within these structures supports the view that they are abnormal dilated hair follicles and that these are a more constant diagnostic feature of hidradenitis than inflammation of apocrine glands, which appears to be a secondary phenomenon¹⁸.

Such arguments are supported by several histological studies. Many patients show follicular lesions compatible with occlusion, whereas primary apocrinitis, previously suggested as the typical histological feature, is present in only a few patients. Apocrine involvement from nearby inflammation was noted in one in five patients^{19,20}. On the basis of these histological findings, hidradenitis is currently considered an inflammatory disease originating from the hair follicle^{21,22}. Follicular rupture spills contents, including keratin and bacteria, into surrounding tissue causing a vigorous chemotactic response and abscess formation. Epithelial strands are generated, possibly from ruptured follicular epithelium, to form sinus tracts^{8,23,24}.

There is some discussion in the literature about the name of the disease. Some authors use the argument that at centre stage there is an inflammation of the apocrine gland after occlusion of the hair follicle and so the term 'acne inversa' should be considered more appropriate.

Otherwise, the term 'hidradenitis suppurativa' refers to a false pathogenetic concept in which the primary event is centred upon the apocrine sweat glands. Despite these arguments, the term hidradenitis suppurativa remains in widespread use in recent literature²¹.

The significance of bacterial infection in hidradenitis is controversial. Bacteria probably contribute to the pathogenesis secondarily, as they do in acne. Although bacterial superinfection with streptococci and staphylococci is considered part of the pathogenesis, cultures from lesions are frequently sterile and antibiotics are not curative. When there is extensive disease progression various pathogens may be found, including *Staphylococcus aureus*, *Streptococcus milleri* and *Chlamydia trachomatis*^{8,25–27}. In perianal disease there is an increased incidence of *Escherichia coli*, *Klebsiella* sp. and *Proteus* sp. as well as anaerobes⁹.

Aetiology

Although occlusion of hair follicles may be the primary event, there are both acquired and genetic predisposing factors. The condition seldom occurs before puberty and less than 2 per cent of patients have onset of the disease before the age of 11 years. In women the condition may persist up to the menopause, but onset thereafter is rare²⁸. These observations suggest a relationship between onset of hidradenitis and androgen levels. Several authors support the concept of the condition being androgen dependent in adults^{29–32}. Premenstrual exacerbation has been observed in women with dysfunction of the hypothalamopituitary system, but a direct correlation has not been demonstrated³⁰. In some women hidradenitis evolves after starting the combined oestrogen–progestogen pill. Changing the pill into an oral contraceptive with more oestrogenic properties causes the symptoms to disappear²⁹.

Overall, there is no evidence to support hyperandrogenism in women with the disease when compared with age-, weight- and hirsuties-matched controls. Furthermore, there have been some reports of the continuation and primary development of hidradenitis in postmenopausal women^{28,33}. Also arguing against a role for androgens is the observation that pregnancy and menstruation do not have a consistent effect on the disease and, although acne is more prevalent in males, hidradenitis is predominantly a female disease^{28,30,33}. In addition, it has been treated effectively with testosterone in selected patients, whereas many women with hidradenitis have normal androgen levels³⁰. The causal relationship between androgen levels and the condition remains under discussion.

Some publications have postulated the existence of a familial form of hidradenitis suppurativa with autosomal dominant inheritance^{34–37}. Based on the analysis of a family history of 26 consecutive probands with the disease, 11 families were found with evidence of a genetic aetiology and probable single gene transmission. The disease frequency among first-degree relatives of those with familial hidradenitis was 34 per cent. For autosomal dominant inheritance one would expect a 50 per cent chance of developing the disease.

Possible reasons for this shortfall include the fact that many of the first-degree relatives were under the age of 20 years at the time of the study. Other factors may be the variable penetrance of the hidradenitis gene and incomplete ascertainment. Several years later, the same families were reanalysed, particularly the group aged less than 20 years³⁷. In the group with a positive family history ten affected and nine possibly affected patients were found among 37 surviving first-degree relatives of disease sufferers. These findings support the concept of a familial form of hidradenitis with autosomal dominant inheritance.

The use of chemical irritants, such as deodorants, mechanical irritation and shaving, have been suggested as predisposing factors^{9,38–42}. However, comparing patients with the disease with age-matched controls has not shown any significant difference in shaving habits, the use of deodorants or the application of chemical depilatory agents⁴².

One observation that is universal throughout the literature is a high prevalence of heavy cigarette smokers among patients with hidradenitis suppurativa; some report a prevalence of as much as 90 per cent^{7,43–45}. This observation is supported by patients who claim that the severity of their disease increases with smoking more and decreases, or even disappears, when smoking is stopped. The significantly higher proportion of active smokers among patients with the condition can be expressed by an odds ratio of 9.4⁴⁴.

This all suggests that cigarette smoking is a major triggering factor. It remains unclear, however, what kind of pathogenetic mechanism is responsible for the effect of smoking on the manifestation of disease; it is probably multifactorial. It has been postulated that the disorder may be caused by the effect of nicotine on the exocrine glands. Nicotine initially stimulates glandular secretion but eventually inhibits normal function. This might provide a mechanism that predisposes to plugging of the ducts of the glands, which leads to an inflammatory reaction^{7,46}. Another hypothesis is that altered chemotaxis of polymorphic neutrophils plays a key role in the pathogenesis, as already postulated for palmoplantar

pustulosis^{47,48}. A recent immunohistochemical study has identified a causative role of the non-neuronal cholinergic system in the pathogenesis of hidradenitis by promoting infundibular epithelial hyperplasia and, consequently, follicular plugging⁴⁹.

Obesity is another commonly cited aetiological factor^{38,41}. However, when the variance from ideal body-weight of patients was compared with that of controls, no significant differences were found⁵⁰. Hidradenitis has been associated with other endocrine disorders, such as diabetes, acromegaly and Cushing's disease. However, causality has not been proven^{51,52}.

Histopathology

Early lesions of hidradenitis show hyperkeratosis of the terminal hair follicle, with subsequent occlusion of the follicle and dilatation with stasis in apocrine and exocrine glands. As the disease progresses, an extensive perifolliculitis and spongiform infundibulofolliculitis is seen, combined with cystic epithelium-lined structures containing hair shafts. The final stage is characterized by a dermis with an inflammatory cell infiltrate, granulation tissue, giant cells, subcutaneous abscesses and sinus tracts^{18,20,24,53}.

Immunohistological findings of newly formed lesions show that the initial lesion is an occluding spongiform infundibulofolliculitis²⁰.

The inflammatory changes involve a local cell-mediated immune reaction, composed of neutrophils, lymphocytes (with T cell lymphocyte predominance) and histiocytes. Histologically, the homogeneity between patients is high. Most specimens show contained poral occlusion with sinus tracts or cysts. Primary apocrine involvement is seen occasionally and fibrosis is common. Intraindividual variation is limited and so it has been postulated that hidradenitis should be reclassified as follicular disease, as poral occlusion is demonstrated more frequently than primary apocrinitis²⁴. In vulvular hidradenitis, eccrine glands are mostly present in active areas and apocrine glands are far away from active inflammation. Inflammation and eventual destruction of glands appears to be a secondary part of the disease process⁵³. Several authors have investigated the immunological status of patients with the condition and have found no primary cellular or humoral abnormalities^{2,54}.

Clinical manifestations

Lesions begin as 'blind' boils, most commonly at or soon after puberty. The onset is insidious with early symptoms consisting of pruritus, erythema and hyperhidrosis^{1,41}.

Occlusion of a hair follicle results in a nodule or cyst, which is deep and rounded with no pointing or central necrosis (unlike staphylococcal furunculosis, but similar to the comedones of acne).

The cyst may rupture spontaneously and discharge purulent material. As the area heals, it becomes fibrotic and new nodules develop adjacent to original lesions. Some lesions rupture spontaneously leading to sinus tract formation deep within the cutaneous tissue. Others remain as indurated inflammatory masses and may become infected resulting in deep abscesses, which are painful and compromise movement of the adjacent extremity (*Figs 1 and 2*). This state may culminate in chronic sepsis with sinus and fistula formation, and extensive dermal scarring¹¹. Without treatment there is progressive destruction of the normal skin with development of periductal and periglandular inflammation, and dermal and subcutaneous fibrosis. Initially, the axillary and/or anogenital regions are involved, usually with remarkable symmetry (*Fig. 3*).



Fig. 1 Inguinal hidradenitis showing nodules, fistula formation and dermal scarring

Anogenital involvement most commonly affects the groins, with extension to inguinal regions, mons pubis, inner thighs and the sides of the scrotum. Buttocks, perineum and perianal folds are often included (*Figs 4 and 5*). The sinuses may dissect deep into the tissues, involving muscle, fascia and even bowel. A labyrinth of tracts may occur in advanced disease⁸. Perianal hidradenitis may present with pain, swelling, purulent discharge, pruritus or bleeding. It can mimic several common problems, such as furunculosis, anal fistula, pilonidal disease, perianal abscesses or Crohn's disease^{7,55}. Fistulas to the anal canal in patients with hidradenitis should extend only as far as lower portion of the anal canal, in the skin of which apocrine glands can be found^{3,56}.

The disease may be mild and limited, or severe. In the latter case new lesions continually develop in zones with apocrine glands and cause chronic, widespread, deep infection. Recurrent, foul-smelling discharge from multiple sinuses typically causes soiling of clothes, and leads to a limitation of social contacts, discomfort during sexual intercourse and forfeit of employment.

Hidradenitis is very debilitating, both physically and psychologically. Measurement of impairment of quality of life with the Dermatology Life Quality Index has shown



Fig. 2 Axillary hidradenitis showing nodules, fistula formation and dermal scarring. A small resection has been performed, with secondary healing

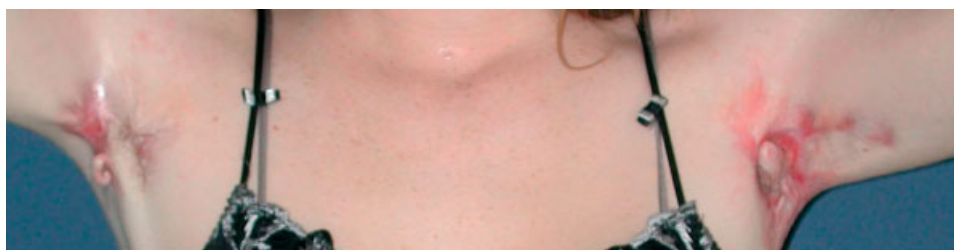


Fig. 3 Bilateral axillary hidradenitis



Fig. 4 Extensive anogenital hidradenitis with sinus tract and fistula formation

that hidradenitis causes a high degree of morbidity, with the highest scores obtained for the level of pain caused by the disease⁵⁷. Some authors have even concluded that hidradenitis causes much more impairment in quality of life scores than other dermatological conditions, including chronic urticaria, psoriasis and neurofibromatosis⁵⁸.

Case reports show that in extreme situations hidradenitis may present as a urethral cutaneous fistula, as a mamillary fistula or as an extensive lumbosacral and epidural abscess^{59–61}. Chronic irritation and infection may lead to proliferative changes, including non-melanoma skin cancer. Some reports have described a 4–6-fold increased risk of skin cancer^{62–66}.

Differential diagnosis

The diagnosis is based mainly on clinical history and physical examination. Early recognition is essential as most patients can be treated effectively when diagnosed



Fig. 5 Widespread anogenital hidradenitis with extension to inguinal regions

at an early stage. Although specific diagnostic criteria do not exist, some clinical criteria are typical of early hidradenitis: recurrent deep boils for more than 6 months in flexural, apocrine gland-bearing sites, with poor response to conventional antibiotics and a strong tendency towards relapse and recurrence. In addition, there may be onset after puberty with comedones in apocrine gland-bearing skin, combined with a personal or familial history of acne, hidradenitis or pilonidal sinus. Fertile women who smoke are mainly affected^{4,67}. Diagnosis rarely requires biopsy, especially when there are well developed lesions, as no other process produces recurrent abscesses and sinus tract formation with a characteristic distribution in apocrine gland-bearing skin.

In axillary hidradenitis, distinction should be made from septic furunculosis, inflamed epidermoid cyst, carbuncle, folliculitis, granulomatous disease, tuberculosis cutis, actinomycosis and carcinoma. Differentiation in perianal or inguinal hidradenitis is more difficult and distinction should be made from inguinal granuloma

(which, unlike hidradenitis, may involve the vagina and cervix), cryptoglandular anal fistula, Crohn's disease, pilonidal sinus, perirectal abscess, tuberculosis, actinomycosis and lymphogranuloma venereum (a sexually transmitted disease)^{2,67}.

Crohn's disease may be a source of confusion in anogenital hidradenitis as presentation may be similar and the two conditions may be associated. Approximately 20 per cent of patients with Crohn's disease have skin lesions. These cutaneous manifestations mostly occur in severe disease and complicate rather than precede the intestinal disease⁶⁸. Perianal lesions are the initial presentation in 5 per cent of all patients with Crohn's disease and, as a consequence, distinction from perianal hidradenitis can be troublesome^{56,69–71}. The clinical and histological features of established anogenital hidradenitis and cutaneous Crohn's disease may be very similar⁷².

Several authors have suggested, based on case reports, that there is an association between Crohn's disease and hidradenitis^{55,69,71–73}. There have been reports of coexistence in up to 39 per cent of patients with hidradenitis⁵⁵. It is postulated that the local swelling and inflammation associated with Crohn's disease may precipitate the development of perianal hidradenitis in patients already prone to this pathology. On the other hand, it would not explain the presence of axillary, groin and buttock disease. Another possible pathogenetic mechanism, supported by several authors, is autointoxication^{72–74}. Autointoxication is the concept that disease may result from absorption of toxins from the bowel, and is a possible explanation of the disease association with Crohn's. Minor defects of intestinal mucosal barrier function may result in failure of intestinal IgA to inactivate bacterial and dietary antigens. The IgA immune complexes formed are deposited in the skin. Evidence for this theory includes the presence of IgA immune complexes in dermatitis herpetiformis⁷⁵.

Differentiation of hidradenitis suppurativa from Crohn's disease depends on a clinical history of gastrointestinal involvement, inspection of all apocrine gland-bearing areas and endoscopy. Hidradenitis rarely involves the anal canal and, if it does, it tracks subcutaneously rather than across the sphincter; it never tracks above the dentate line^{56,76}. Combined perianal disease is a serious problem and patients may require faecal diversion or even proctectomy⁵⁵. There have been several recent reports describing the successful treatment of combined disease with the antitumour necrosis factor antibody infliximab^{77–79}.

Treatment

Treatment of hidradenitis suppurativa has proved difficult and unsatisfactory. Management must be individualized according to the site and extent of the disease, and both medical and surgical options should be discussed before initiating treatment. Initial conservative measures with medication and limited incision and drainage can alleviate the acute symptoms. Eventually, however, more radical surgery may be necessary to control the disease and prevent recurrence.

Medical treatment

Hidradenitis in its early stages is often treated with antibiotics, antiseptic compresses, and simple measures to reduce pressure and shearing forces at the affected sites. To this must be added absolute cessation of smoking^{51,80}. Some patients obtain symptomatic relief with a long-term course of antibiotics, especially antiacne antibiotics. Topical or oral clindamycin has an effect and is favoured in the literature. In one study patients were treated with either topical clindamycin or a placebo⁸¹. The number of abscesses, inflammatory nodules and pustules was significantly less in the clindamycin group than in the placebo group at each monthly evaluation, without any side-effects. In another study, systemic therapy with tetracycline (500 mg twice daily for 3 months) was as effective as topical clindamycin²⁷. Some authors postulate that the combination of clindamycin and rifampicin might be effective⁸². If specific pathogens can be isolated, then an appropriate antibiotic can be selected.

Although antibiotics can induce a remission, the condition is very likely to relapse with time. There is no evidence that long-term antibiotic therapy alters the natural history of hidradenitis^{83,84}. Still, even though antibiotics are not curative, they may reduce odour, discharge and pain.

Hormonal treatment with antiandrogens has produced mixed results in women with the disease^{85–87}. The use of ethinylestradiol combined with cyproterone acetate has proved useful in a randomized controlled trial, but continued use raises questions of safety⁸⁶.

Another approach, based on the relationship between hidradenitis and acne, is the use of retinoids^{88–90}. Despite their successful use in acne, the results in hidradenitis have been less encouraging. There has been no randomized controlled trial and studies suggest that oral isotretinoin probably has little effect⁹⁰. In the milder form of disease, the use of isotretinoin has proved more successful, possibly by correcting the follicular abnormality that initiates the processes associated with chronic disease⁹¹.

However, several authors consider this agent a useful adjunctive treatment to reduce inflammation, suppuration and oedema before and after surgery².

The use of immunosuppressive agents, such as ciclosporin, has produced moderate improvement in a few case reports^{92–94}. Although an initial benefit might be observed owing to non-specific anti-inflammatory effects, the use of such drugs is of limited value in the treatment of hidradenitis.

The association of hidradenitis with inflammatory bowel disease has permitted the use of antitumour necrosis factor α . Initial reports have described patients with combined disease who were treated successfully with infliximab⁷⁷. Since these first observations, further studies have described the positive effects of infliximab and etanercept in patients with hidradenitis without Crohn's disease^{95,96}. A recent trial has shown promising results with subcutaneously administered etanercept. The treatment was well tolerated, safe and effective. All patients reported a decrease in the extent of their disease and quality of life scores were improved⁹⁷.

Surgery

The principal treatment for chronic, relapsing and severe hidradenitis suppurativa is surgical excision, although cure can be achieved only at the excision site⁹⁸. Controversy still exists about the best surgical approach. Various factors may influence decision making, such as the site affected, the extent of the disease, and the acute or chronic nature at the time of presentation.

Simple incision and drainage of nodules and abscesses is helpful in bringing rapid relief, but this should be followed by extensive excision to prevent recurrent episodes of inflammation. Drainage alone is associated with a recurrence rate of up to 100 per cent, with a median interval to recurrence of only 3 months^{8,11}. Deroofing of sinus and fistulous tracts with marsupialization may be carried out before more definitive surgery, but again there is a high incidence of recurrent disease^{9,98,99}. If the extent of skin involvement is limited, local excision with primary closure seems ideal. On the other hand, studies indicate that such treatment carries a high risk of recurrence, with only a small proportion of patients cured⁹⁸. In summary, the recurrence rate is generally over 50 per cent and is proportional to the extent of resection¹².

Wide excision of the involved areas down to soft, normal tissue, with margins well beyond the clinical borders of activity, is considered to be most effective by many authors^{7,12,99–101}. The block of tissue excised

should include the diseased area along with the adjacent apocrine glandular zone; a margin of 2 cm is needed plus sufficient depth, meaning down to the deep fascia or a minimum of 5 mm of subcutaneous fat. This method reduces the likelihood of recurrence. The apocrine gland-bearing area can be determined with the iodine–starch method¹¹. Although controversy exists about primary closure of the skin¹⁰², it is worthy of note that several authors have reported good results in women having axillary excision because of extra skin available in the lateral mammary area¹⁰³. When primary closure is performed, a gentamicin collagen sponge may be enclosed to reduce infective complications¹⁰⁴.

Other methods of closing the skin include allowing healing by secondary intention, and variants of skin flaps and grafts. Healing by secondary intention is associated with good results, possibly even better than those of skin grafting. In one study, different methods were compared in patients with bilateral axillary involvement¹⁰⁵. Wide surgical excision was performed on both sides, with one side closed by granulation with Silastic® (Dow Corning, Reading, UK) foam dressings and the other by grafting. Most patients, and the authors, preferred the former treatment. Although wound healing occurred faster with skin grafting, it incurred a painful donor site. Patient satisfaction with wide surgical excision and healing by secondary intention was due to the minimal interruption of daily activities associated with the technique. Furthermore, closure of the wound tended to be uncomplicated, with cosmetically acceptable scars and with little limitation of movement. On the other hand, the excised area is large and wound closure may take months^{102,105–108}. Several techniques may help patients to manage their wounds at home and encourage wound closure, such as use of negative-pressure dressings^{109,110}.

Skin grafting and transposed or pedicle flaps have their advocates, and several studies have reported good results with the use of split-skin grafting^{101,107,110–116}. Although effective overall, skin grafting has some limitations. It is generally considered unsuitable for inguinoperineal disease and, when attempted, has a high risk of failure. Anal stenosis has been described as a complication of skin grafting⁹⁹. Furthermore, grafting is associated with a prolonged hospital stay, pain, the inconvenience of a donor site and immobilization of the affected limb.

Several authors have reported good results with free fasciocutaneous flaps in the axilla or groin, despite the complexity of skin type, shape and contour^{111,112,116,117}. There are no controlled trials, however, just small case series.

In the final analysis, sufficient resection of the lesion is the most important issue in treating hidradenitis suppurativa. The surgical procedure to cover the denuded area should be selected to suit the size and the site of the defect.

Carbon dioxide laser excision is a relatively new form of treatment^{40,118,119}. Under local anaesthesia, the area selected is ablated by the laser beam which is passed over the tissues. This method is described as a rapid, efficient and economical for moderate to severe disease. Furthermore, patient acceptance is high as there is less scarring and pain than with conventional surgery.

Overview

The crucial and probably the most effective step in the treatment of hidradenitis suppurativa is the absolute cessation of smoking. Other general measures that might be beneficial include weight loss, avoidance of irritation and use of antibacterial soaps. If there is no improvement, medical treatment can be used in the early stages of the disease. Topical clindamycin may be helpful, if necessary combined with systemic antibiotics (clindamycin or rifampicin). In recalcitrant disease, retinoids, antiandrogens or antitumour necrosis factor α might reasonably be tried.

When this problem becomes chronic or more severe, radical excision is the only definitive treatment. In advanced disease, sinuses and fistulas are formed and there is no medical therapy with any hope of cure. Factors that influence the type of surgical approach are the site(s) affected, the amount of skin and soft tissue involvement, and the chronicity of the disease. Antibiotics or retinoids might be used to control the disease before surgery. Incision and drainage provide only temporary relief. Marsupialization may be an aid to local control before a definitive procedure. When the disease is localized to the axilla or inguinal regions, excision with primary closure can be performed. Limited involvement of the perineum or perianal area can be treated with excision alone, with healing by secondary intention. In extensive disease, emphasis should be placed on adequate excision margins rather than the technique used to cover the defect. Extensive disease of the axilla or inguinal regions can be treated by radical excision and split-skin grafting. In extensive perineal or perianal disease, wide excision with delayed grafting can be advocated.

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