17
Pilonidal Disease and Hidradenitis Suppurativa

Eric K. Johnson

Key Concepts

- Pilonidal disease presents with a wide range of symptoms and multiple treatment options exist. Treatment should be tailored to the severity of disease, anatomy of disease, and patient expectations.
- Because of the wide array of available surgical options, the surgeon treating pilonidal disease should master 3–4 approaches that are applicable to a wide range of disease presentations.
- Treatments applied to both pilonidal disease and hidradenitis suppurativa should not be more disabling for the patient than the disease itself.
- There are numerous medical options available to treat hidradenitis suppurativa. They should be investigated and attempted prior to aggressive radical surgical management.
- Radical excision of hidradenitis suppurativa with surgical reconstruction offers the best hope to avoid disease recurrence.

Background

The term "pilonidal" is derived from the root words "pilus" (a hair) and "nidus" (nest). Since 1880 when Dr. R.M. Hodges coined the term pilonidal sinus [1], the diagnoses of pilonidal cyst, sinus, and abscess have been used interchangeably and somewhat indiscriminately to mean the same thing, though they most certainly do not—in the case of abscess. It is largely for this reason that the more modern nomenclature of "pilonidal disease" (PD) is used to describe the spectrum of disorders that may be encountered. The first published description of this disease occurred in 1847 when Dr. A.W. Anderson described a case of "hair extracted from an ulcer" [2]. The first pilonidal abscess was described in 1854 [3], though there is no question that this condition was encountered earlier. It wasn't until World War II that surgeons became much more familiar with this disease entity, likely because of the large number of cases seen in members of the military. In fact, the disorder was known as "jeep disease" and was thought to be related to modern mechanized warfare, which required soldiers to ride in vehicles for extended periods of time [4].

It is clear from early publications that little has changed in terms of the issues that confront both the patient and surgeon. A 1955 publication from the Veteran's Administration health system reveals that the debate over open and closed wound management is not new [5]. In this study, patients managed with primary wound closure developed recurrence 40% of the time and required hospital stays of approximately 17 days, while those managed with open technique stayed for 30 days and had a recurrence rate of 35%! While we have seen significant reductions in both length of hospital stay and recurrence, it is clear that we still do not have the ideal answer for this condition.

Etiology

There has been considerable debate over whether PD is congenital or acquired, but most would currently agree that it is an acquired disease. It is generally believed that the initiating event is traumatization of the skin and surrounding hair follicles in the natal cleft. This occurs secondary to trapping of hairs, not necessarily those arising locally in the natal cleft. The local anatomy creates an unfavorable environment where friction, warmth, moisture, and perhaps local hypoxia lead to local trauma secondary to the barbed texture of the hair. A granulomatous foreign body-type reaction results. There is even some histological and immunohistochemical evidence that PD may represent a unilocalized type of hidradenitis suppurativa [6]. Disease typically begins as

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a small sinus that may drain fluid but then can progress to numerous sinuses with associated cystic dilation and potential abscess formation. In some cases, unless the process is interrupted, it can become more widespread leading to worsening symptoms. Disease can range from the asymptomatic single sinus found incidentally up to a severe locally destructive process associated with significant disability.

PD is not limited to the natal cleft area, and there are several reports of disease occurring in the interdigital areas in hair dressers [7], as well as in other areas such as the umbilicus [8]. The presence of disease in these atypical areas further supports the above theory. PD has been reported to affect males more commonly than females; however recent data from the armed forces suggests that the incidence rates are similar at 1.9 and 1.7 per 1000 person-years, respectively [9]. There are several risk factors that have been implicated in the development of PD including positive family history of disease, elevated body mass index (BMI > 25), poor hygiene, hirsutism, deep natal cleft anatomy, occupation that requires prolonged sitting, and excessive sweating [10–12]. It is not uncommon to see disease affect an individual who lacks many or most of these factors however. A prospective study comparing 587 patients with PD to 2780 healthy controls showed that hirsute individuals that sit down for more than 6 h per day and who bathe two or fewer times per week have a 219-fold increased risk for sacrococcygeal PD [12]. A positive family history may not only predispose to disease occurrence, but may also be associated with increased recurrence rates after surgery as well as earlier onset of disease [11].

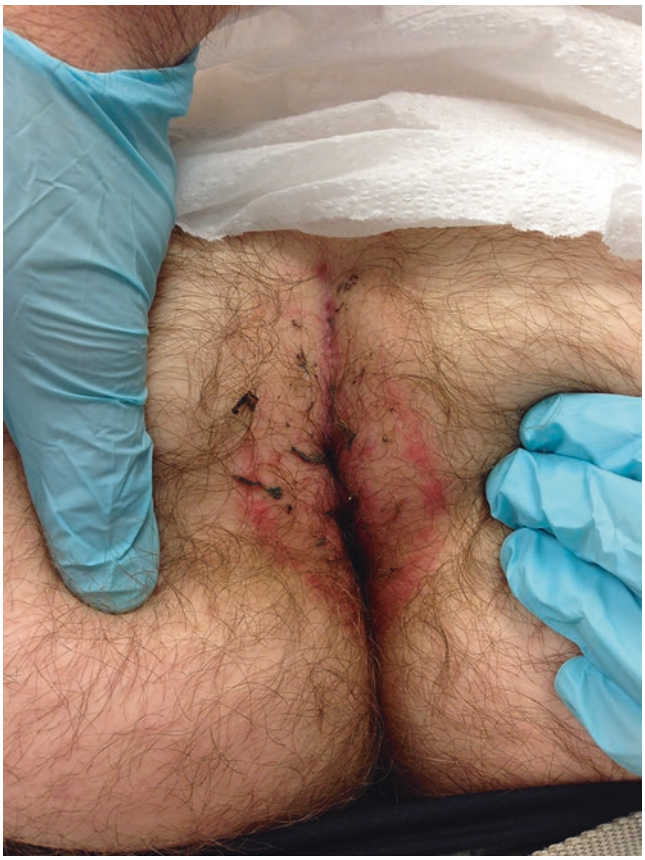


FIGURE 17-1. This image shows a hirsute individual with midline "pits" that could go unnoticed. Note the poor hygiene.

Clinical Presentation/Diagnosis

Patient presentation can range from a referral for completely asymptomatic and incidentally discovered disease to a person who is significantly disabled by locally destructive disease. Commonly encountered scenarios are the patient who has an acute pilonidal abscess that requires drainage, and the surgical office visit to discuss definitive surgical therapy after either acute abscess drainage or persistent disease of moderate severity impacting the patient's quality of life. Often, in the military setting, disease that would otherwise be ignored requires operative management secondary to its impact upon an individual's ability to perform at a high physical level or live in an austere environment.

Establishing a diagnosis is rather simple and does not require extensive testing or imaging. Simple history taking and a physical exam will in most cases solidify the diagnosis. Patients will often complain of pain over the sacrococcygeal area with drainage of clear fluid or bleeding. In the case of acute abscess, fever may also occur. Physical exam will reveal "pits" in the midline. There may be several pits, or only one small pit that could be easily overlooked if the examiner does not consider this diagnosis (Figure 17-1). Examination may also often reveal induration just lateral to midline that can be unilateral or bilateral. This may also be associated with additional draining sinuses. In more significant cases, there may be open wounds that can have a large range in size (Figure 17-2a–c). Acute abscess is typically associated with overlying erythema, fluctuance, and severe local tenderness (Figure 17-3). In a less common scenario, the examiner may mistake PD as an anorectal fistula if a sinus is present close to the anus. It is important to examine the midline overlying the sacrum for pits. If they are present, then pilonidal sinus should be included in the differential diagnosis in these individuals (Figure 17-4a, b).

Recurrent disease in the patient who has already undergone surgical excision is another commonly encountered scenario (Figure 17-5). Recurrence may occur either early (within 1 year) or late. Early recurrence is often actually persistence of an open wound that never healed after surgery. This may be thought of as PD, but in many cases is actually nothing more than a non-healing midline sacrococcygeal wound. Wounds placed in the midline often demonstrate delayed healing or non-healing. The pathophysiology related to a non-healing wound may actually be different than that related to PD; however the methods we use to treat these maladies are similar. Recurrence presents similarly to primary PD, and may be related to poor surgical technique,

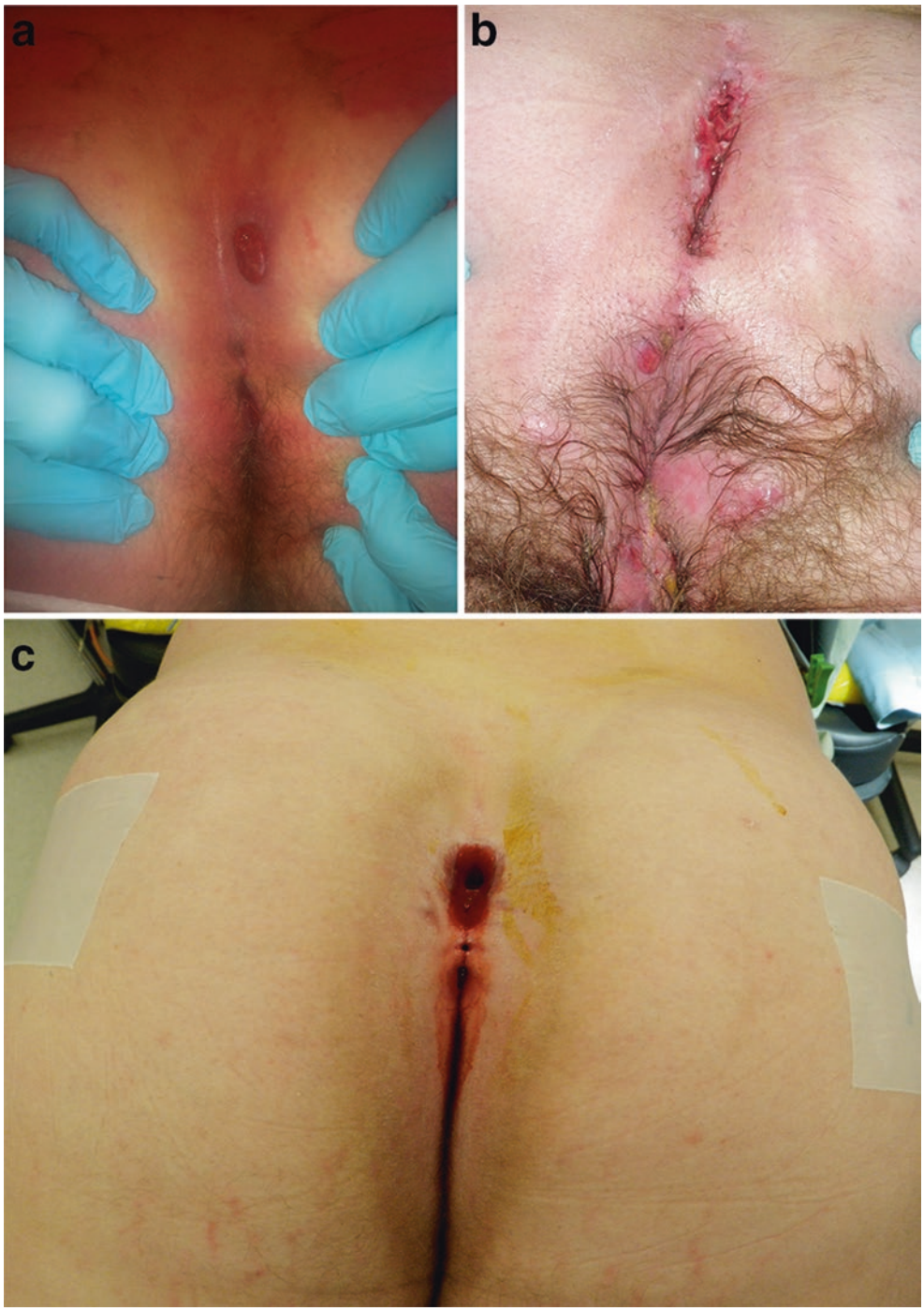


FIGURE 17-2. (**a**–**c**) These images show a range of open wounds that may be seen with pilonidal disease.

patient noncompliance, or failure to modify the pre-existing risk factors that led to disease in the first place. Recurrence may also simply be the natural history of disease.

Treatment

There are numerous treatment options available to address PD. An important overriding concept that should be completely clear is that the treatment should be tailored to the patient's expectations, disease anatomy, and disease severity. Options range from nonoperative therapies up to wide local excision with local flap reconstruction. The debate of open wound management versus closed management remains, and even when primary closure is performed, wound care and physical limitations may be required for an extended period of time. Given the large number of operative choices available, it is likely not practical to be well versed in all. A good recommendation would be to be familiar with three or four operative options that range from simple to complex and provide a solution for several different anatomic configurations of disease.

Nonoperative Management

It is first important to recognize when PD requires no invasive management at all. As stated earlier, some patients are referred based simply upon the incidental finding of midline pits in the natal cleft. If the patient is asymptomatic, and physical examination reveals no concerning findings, they require no operative management. You never want the treatment to be worse than the disease, and that is exactly what one will discover in this setting. The patient may still benefit from counseling regarding ways to reduce their risk of developing symptomatic disease. Risk factor modification such as weight loss, avoidance of prolonged sitting at work, improved hygiene, and weekly clipping of hair in and adjacent to the natal cleft may reduce the chance that a patient will develop symptoms related to PD. These are also appropriate in the setting of active and symptomatic disease. These measures may lead to either improvement in symptoms or quiescence in mild cases. A study published in 1994 showed that these measures combined with limited lateral incision and drainage in the setting of acute abscess led to fewer occupied hospital bed days when compared to excisional procedures [13]. Over a 17-year follow-up, only 23 of 101 cases went on to require excisional therapy.

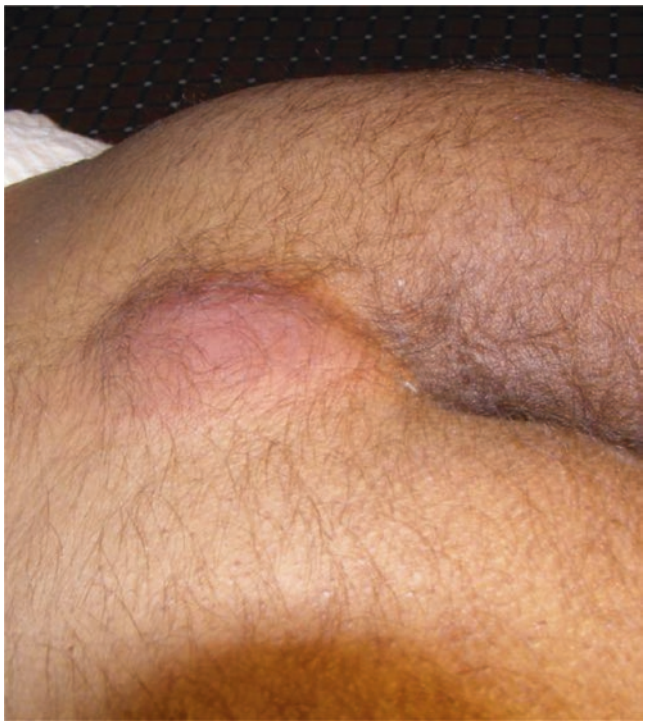


FIGURE 17-3. This image depicts an acute pilonidal abscess.

Given that weekly shaving has been associated with success, many have advocated laser hair removal as a long-lasting alternative for the conservative management of PD. Despite the interest in this mode of therapy, we lack any robust data to support its use. Small studies of 6–14 patients have shown some benefit to laser epilation in the setting of recurrent PD [14, 15]. This procedure is uncomfortable for the patient and often requires local anesthetic. Treatments are performed over 3–11 sessions at 6–8-week intervals and can be quite costly. A study of laser epilation in teenagers with PD, 25/28 of which were managed initially with surgery, showed only one recurrence over a mean follow-up of 2 years [16]. The authors concluded that use of the laser was a safe method for addressing intergluteal hair that may reduce recurrence rates.

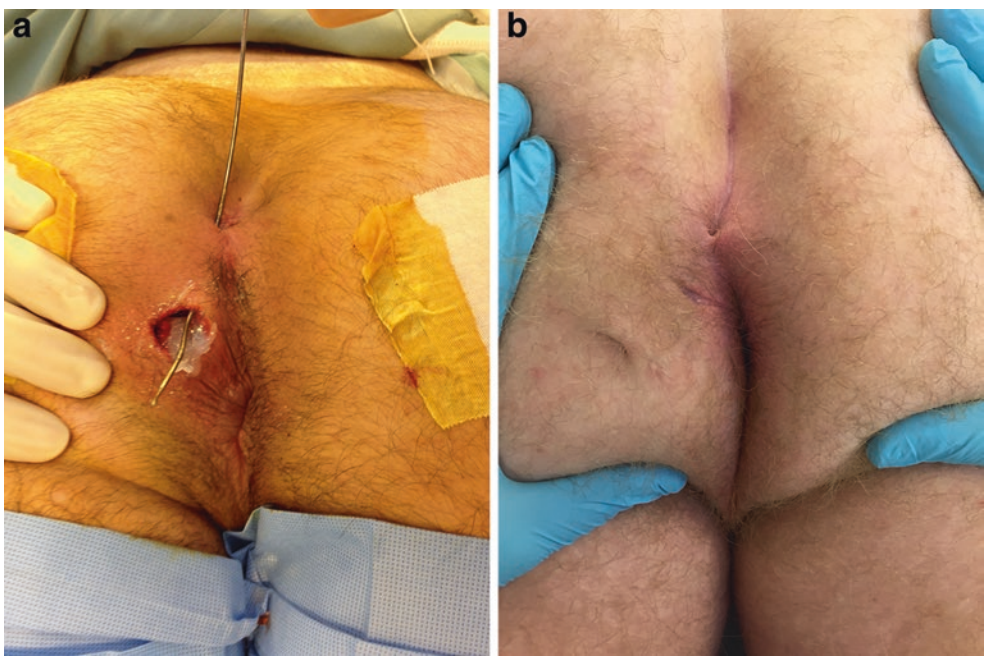


FIGURE 17-4. (**a**, **b**) These images show two different patients that presented with draining perianal sinuses. Note the midline pits over the sacrum that make one more suspicious of pilonidal disease as the cause.

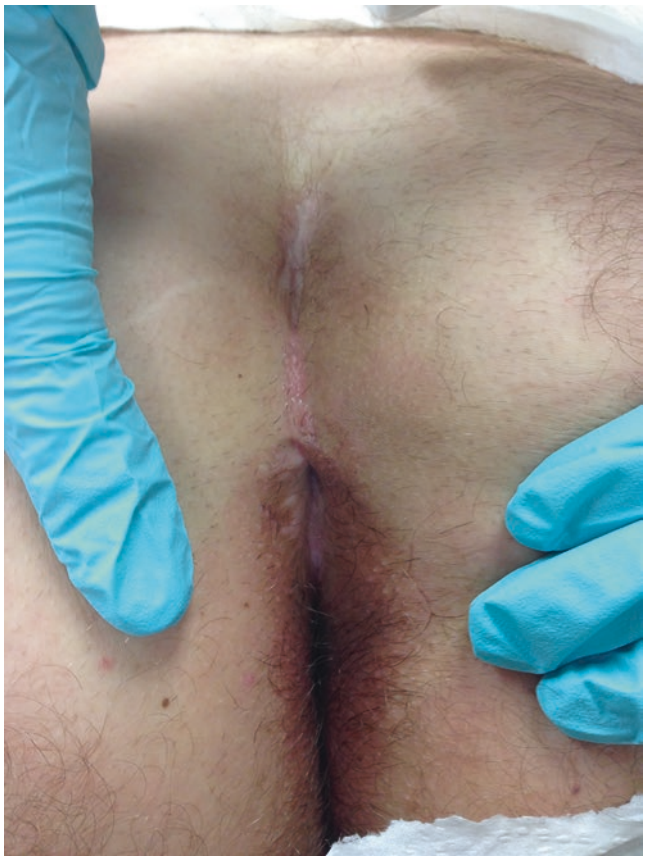


FIGURE 17-5. This image shows a patient who developed recurrence after an attempt at a cleft lift procedure. Incorrect performance of the distal portion of the procedure may have led to this recurrence.

A randomized trial comparing laser hair removal to traditional methods as an adjunctive therapy after surgery for PD demonstrated a lower recurrence rate in the laser-treated group [17]. This appeared to be related to noncompliance with traditional hair removal methods after 1 year. There is however some debate over the benefit of hair removal in the setting of PD that has been managed operatively. A retrospective analysis of patients that had undergone surgery to treat PD was performed with focus on those who performed razor hair removal vs. those that did not [18]. Recurrence was observed in 30% of those who shaved vs. 19% of those who did not shave ($p=0.01$). This would suggest a potential negative effect of postoperative razor epilation. Future studies should likely focus on a comparison between laser hair removal and no hair removal in the adjunctive setting.

While some form of hair removal may lead to reduced recurrence rates as well as reduced requirement for excisional therapy, this method alone is unlikely to lead to disease cure—especially in the setting of more significant or severe disease. Often the hair that is found inside of sinus tracts is clearly noted to be long hair from other parts of the body. It is theorized that longer hairs can fall into the natal cleft, become trapped, and result in disease. Clearly, local epilation alone will not eliminate this threat.

Although not necessarily considered nonoperative (maybe non-excisional) therapy, methods employing the use of phenol or fibrin glue injection to ablate sinus tracts have been investigated in small series by many [19–26]. These techniques often employ tract curettage, debridement, and hair removal, which contribute significantly to success. Use of phenol as an ablative agent has been associated with success rates of 60–95% [19–21]. Fibrin glue injection combined with a variety of techniques has shown success in the range of 90–100% [22–25]. A recent evaluation of individuals treated with fibrin glue revealed that 79% of patients were satisfied, 71% were back to normal activities within 2 weeks, and 74% required no further treatment [26]. A video-assisted ablative technique has also been described using a 4 mm rigid hysteroscope with a five french working channel [27]. Continuous irrigation is used, hair is removed, and the cavity and tracts are ablated using a bipolar electrode. Only one recurrence was detected over 12 months in 27 patients. This may represent a potential option for minimally invasive/non-excisional therapy. The potential advantages of these therapies over excisional methods are more rapid recovery and less post-procedural pain.

Operative/Excisional Management

There are numerous methods available for the operative management of PD. The literature is filled with a large number of publications reporting results from various procedures. The typical manuscript is a retrospective review examining the results from a small series of patients that have undergone one specific type of operative procedure. There are several randomized trials comparing one surgical method vs. another with variable results. Essentially, it is possible to find evidence to support whatever procedure one prefers to perform. Results are likely related to variations in how patients are cared for postoperatively as well as differences in surgical technique. It is best to review some of the more common methods of operative management beginning with those that are considered simple and progressing to the complex. A well-prepared surgeon will be familiar with most of these methods, and will tailor their management to disease severity, disease anatomy, and patient expectations.

Basic Procedures

Outside of incision and drainage of a pilonidal abscess, the simplest procedure to perform is laying open of the cyst and all sinus tracts. This may also be termed "unroofing" of disease. This and wide local excision of all disease down to the post-sacral fascia were the procedures performed most commonly in the early days of PD management. Often, unroofing was combined with marsupialization of the wound. Recurrence rates of 15–35% [5] led many to seek out more effective methods of surgical management. It is important to ensure that as much of the surgical wound as possible be kept

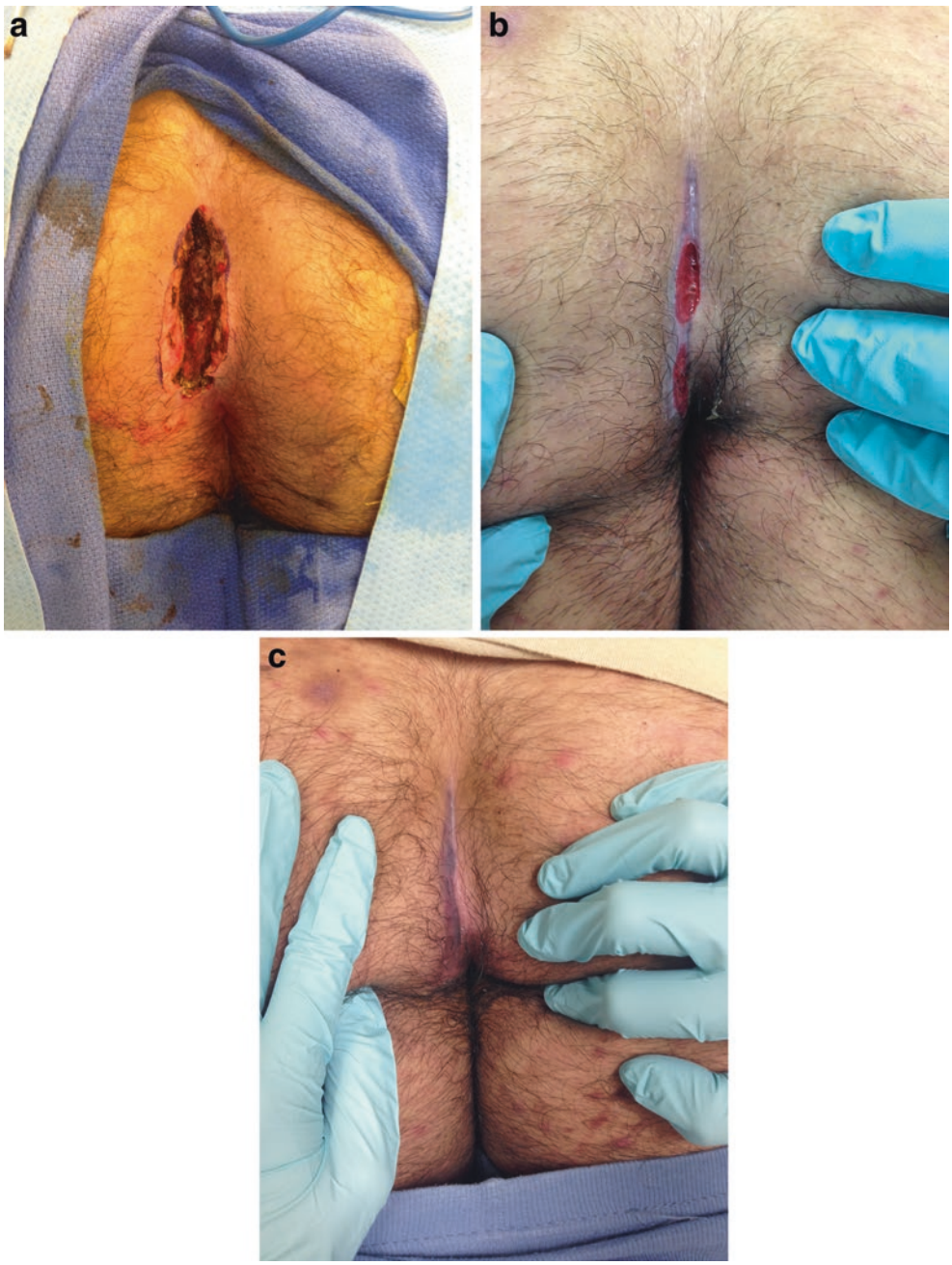


FIGURE 17-6. (**a**–**c**) These images show yet another patient who presented with what was thought to be an anal fistula. Midline pits were noted and the disease was treated with a lay-open technique, which resulted in rapid healing. Of note, this could potentially represent hidradenitis suppurativa.

off the midline, as midline wounds tend to have some difficulty with healing. Simple tract unroofing and curettage are particularly helpful in the setting of minor disease affecting the perianal area (often mistaken as an anal fistula). The bulk of this wound will lie off the midline and will heal quickly (Figure 17-6a–c). There continues to be debate over which approach is superior, though recent data would suggest that a higher volume of excised specimen is associated with a higher surgical site infection rate and likely a higher risk of recurrent disease [28]. The next logical step was to perform excision combined with primary wound closure which can often require the mobilization of minor skin flaps.

Primary closure has been combined with drainage in some settings with a wide variation in results. The use of a drain in this setting has been studied, but has not been shown to result in improved results as far as patient satisfaction, healing, or infection is concerned [29]. A meta-analysis of this subject showed that there were no statistically significant differences in outcomes with or without the use of a drain in the setting of primary wound closure [30]. A recent randomized controlled trial comparing the laying open method to wide excision with primary closure showed that healing occurred faster in the primary closure group with no differences in the groups noted at 1 year of follow-up [31]. Interestingly, this

group of investigators made no effort to keep the majority of the wound off the midline. Rao and colleagues in 2010 published a prospective randomized study comparing the lay-open technique to primary closure augmented by the placement of gentamicin-impregnated collagen [32]. The antibiotic-impregnated material was placed in the base of the wound with overlying tissue closure. The results showed improved healing at 4 weeks, improved postoperative pain, and lower cost in the primary closure group. Recurrence rates were no different at 5 years.

Another group performed a 4-arm randomized trial comparing primary closure, primary closure with hydrogen peroxide irrigation, wide local excision, and wide local excision with hydrogen peroxide irrigation [33]. The wide local excision combined with peroxide irrigation group showed the lowest recurrence rate and the fastest time to healing. The investigators attributed this to the ability to clearly delineate all tracts and disease with peroxide irrigation, allowing them to perform a more precise and low-volume excision. Similarly, another group performed a retrospective analysis of PD patients that had undergone surgery and concluded that use of methylene blue injection to delineate disease was associated with a lower recurrence rate [34].

There have been several descriptions of "pit picking" procedures over the years. These procedures are relatively minor in terms of the amount of tissue excised; they result in small wounds, and may be ideal for those suffering with mild to moderate levels of disease. These procedures are not suitable for the patient with a large open wound or in those with severe recurrent disease. The basic premise of this method is that the central pits are excised with minimal surrounding tissue, hair and debris are removed, the old adjacent abscess cavity or "cyst" is excised through a lateral incision using an undermining technique, the pit excision sites are closed primarily, and the lateral incision is closed partially to allow for drainage. This results in a good cosmetic result with minimal pain, early return to work, and rapid healing (Figure 17-7) [35]. A punch biopsy knife of appropriate size may be used to perform the pit excision and is ideal for this application. This procedure has been modified slightly by many, but the basic tenets remain in the various methods. The use of phenol as a sclerosing agent has been combined with pit excision and has resulted in good outcomes [36].

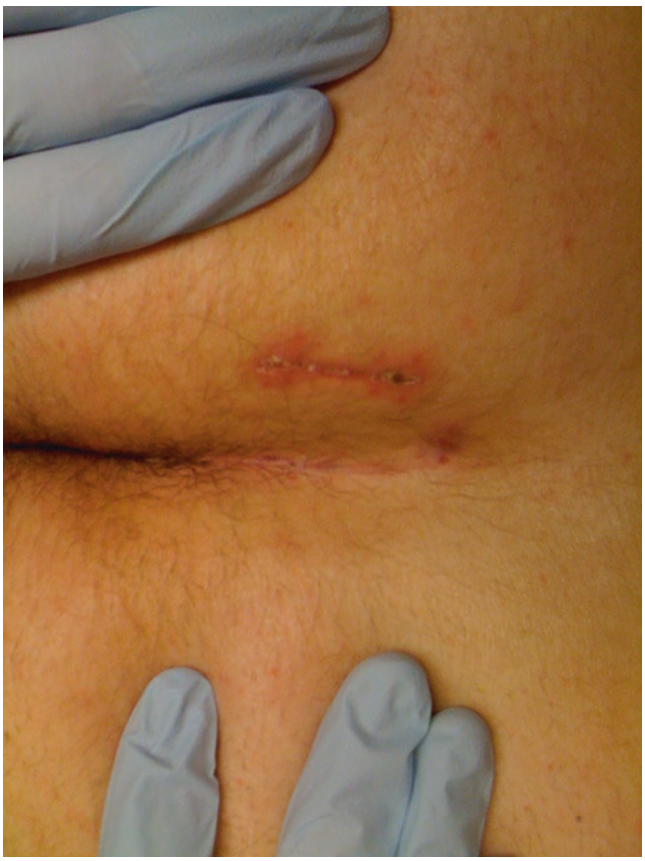


FIGURE 17-7. This image shows a patient 2 weeks after a simple Bascom, or "pit picking" operation.

Complex Procedures

The common thread among all "complex" procedures is the mobilization of adjacent tissue to achieve primary wound closure—in effect, the creation of a local flap. Some of these procedures combine wide local excision of diseased tissue with flap reconstruction, while others preserve as much local tissue as possible. These procedures also range from simple to complex. While there are numerous options, attention will be devoted to the discussion of the Karydakis flap, the Bascom cleft-lift procedure, and the rhomboid or Limberg flap procedure and its modifications. There are additional flap procedures such as the z-plasty, V-Y advancement flap, and other rotational flap techniques that will not be discussed. Many support the use of flap procedures in primary PD, while others believe that they should only be used in the setting of disease recurrence after primary surgery. It is possible that these procedures are more effective in curing disease, because they result in a modification of the natal cleft anatomy. The majority of these techniques result in a flattening of the natal cleft, which may prevent disease recurrence.

Karydakis Flap

This procedure is performed first by excising the affected tissue in the midline, typically leaving an elliptical defect. A beveled skin flap is then created and mobilized across the midline to facilitate a primary closure that is lateral of midline (Figure 17-8). A closed suction drain may be used or omitted. The purported advantages of this procedure are the tension-free closure that is out of the midline coupled with some flattening of the natal cleft. This is probably the easiest flap procedure to perform. This procedure has been shown to be superior to simple primary midline closure in terms of patient satisfaction, recurrence rate, and rate of postoperative complications [37]. It has also been reported to be comparable to other more complex flap procedures such as the modified Limberg flap [38, 39].

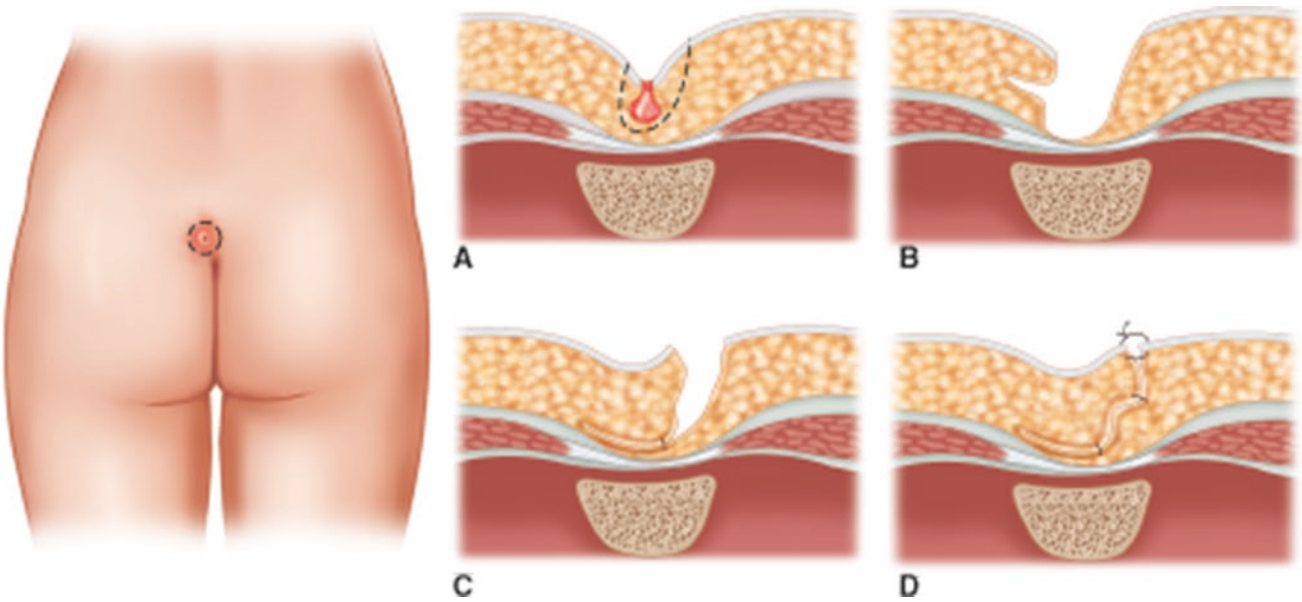


FIGURE 17-8. This drawing depicts one method of performing a Karydakis flap.

Cleft Lift Procedure (See Video 17-1)

The cleft lift procedure was originally described and popularized by Dr. John Bascom, and is often referred to as the Bascom cleft lift. This is a simple but intricate procedure that is designed to "lift" the natal cleft and result in an incision that is closed off the midline. Interestingly, wide excision is not required—in fact, the only tissue that is excised is the overlying skin on one side of the natal cleft. This procedure requires that the patient be marked prior to incision to establish a "safe zone," beyond which no dissection is performed. The patient is placed in the prone position and the buttocks are squeezed together (Figure 17-9). The area where the skin on both sides of the natal cleft touches is marked with a magic marker. This establishes the safe zone. The buttocks are then taped apart exposing the disease (Figure 17-10). After skin preparation, the area to be excised is marked with another marking pen (Figure 17-11). This proposed incision will be partially elliptical and should extend from the midline pits out to one side of the safe zone. The distal portion of this incision is scimitar shaped in order to facilitate closure near the anus without causing local deformity.

Local anesthetic is injected and the incision is made down to the level of the subcutaneous fat. The overlying skin is excised taking care to leave the subcutaneous fat in place. A flap is then raised across the midline out to the opposite safe zone border (Figure 17-12). The thickness of this flap should approximate that of a breast flap that would be created during a mastectomy. When creating the flap down toward the distal portion of the incision (near the anus), the flap should be thicker to prevent dimple formation near the anus. Any disease-related debris or granulation tissue should be gently debrided with a surgical sponge and irrigation with saline

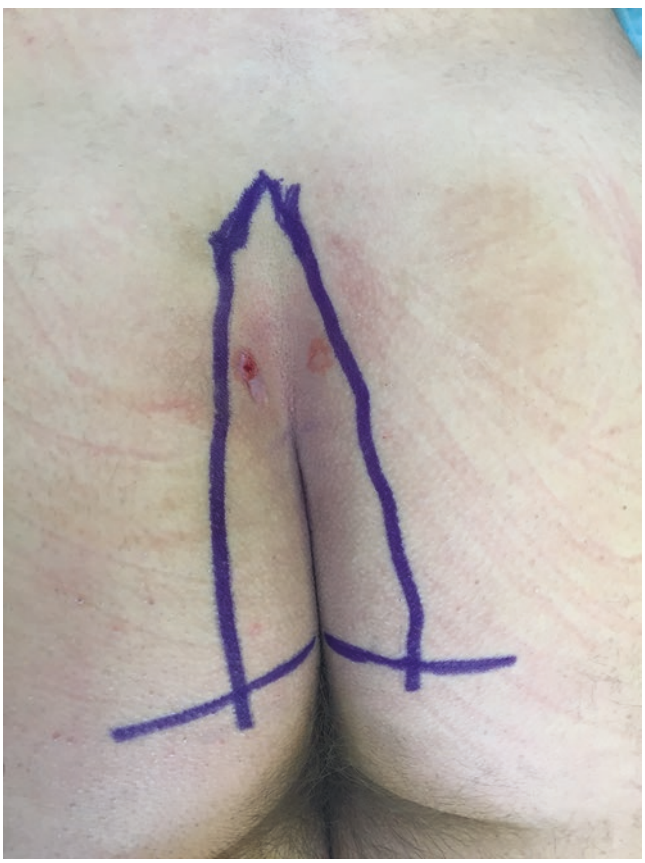


FIGURE 17-9. After squeezing the buttocks together and marking the safe zone.

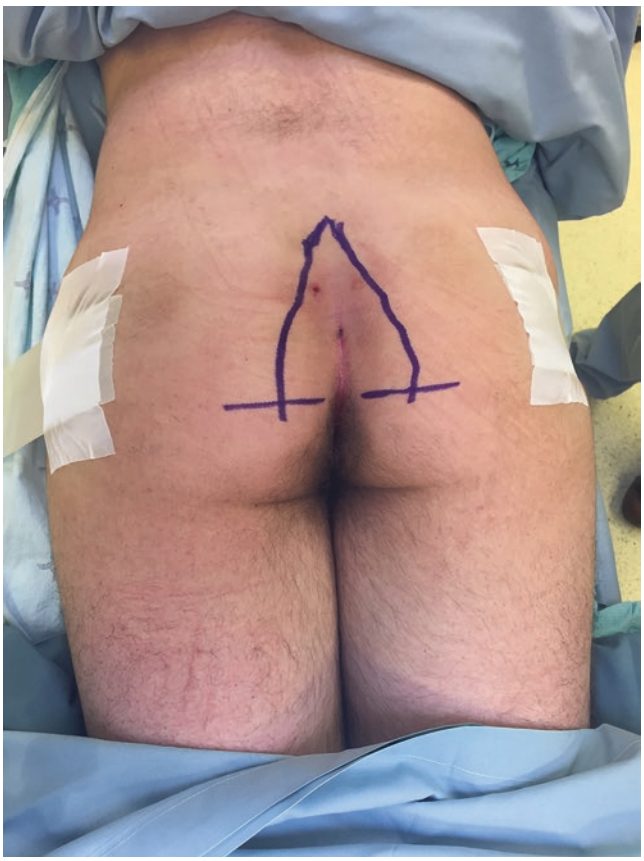
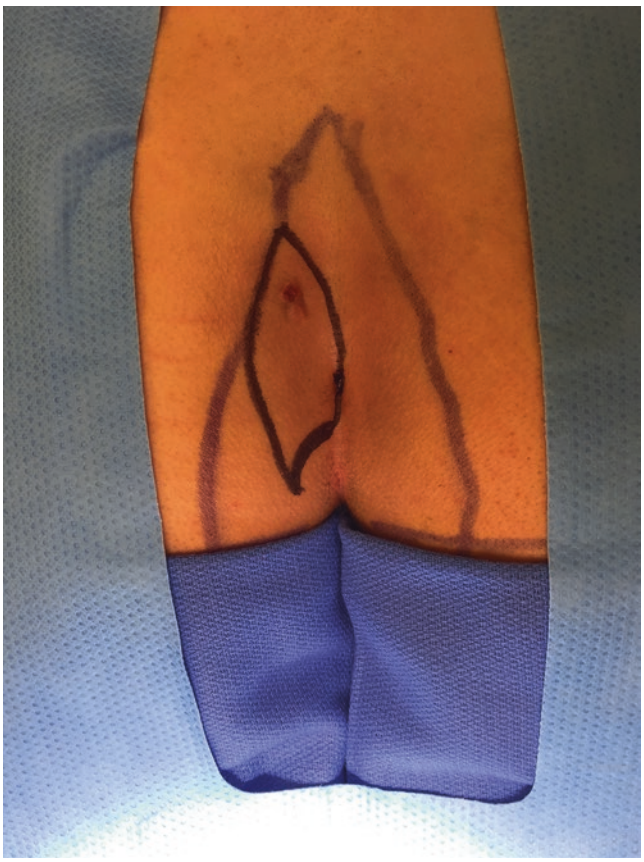


FIGURE 17-10. Image showing the buttocks taped apart under tension providing excellent operative exposure.



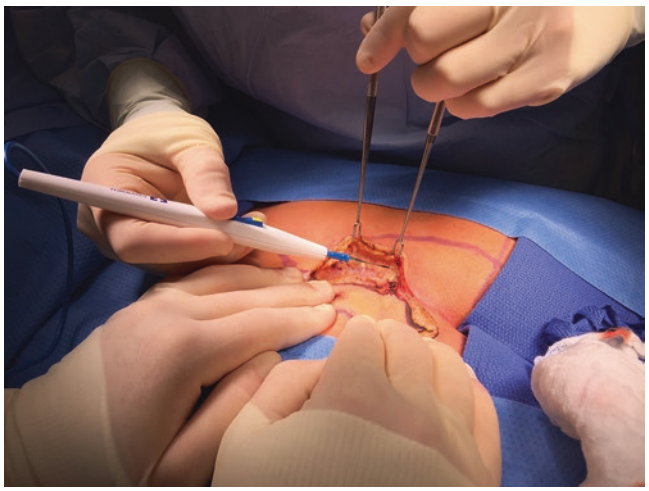


FIGURE 17-12. This image shows the operative creating the flap to be used for the cleft lift.

should be undertaken. Any remaining "cyst wall" or tissue contracture can be divided into squares with a scalpel or electrocautery device. The subcutaneous tissue is then closed in layers with an absorbable suture. The superficial layers are reapproximated in layers, lastly with a subcuticular suture (Figure 17-13a,b). Use of a drain is optional, but certainly not necessary.

A case-control study published in 2011 compared the results of the cleft lift procedure to wide excision and packing in 70 patients [40]. A total of 97% of patients undergoing cleft lift healed completely while only 73% of wide excision patients healed. Three of nine patients with chronic wounds underwent subsequent cleft lift with a 100% success rate. Recurrence was noted in 2.5% of cleft lifts and in 20% of wide excisions. Others have shown similar success in rates of healing with the cleft lift procedure as compared to wide excision and packing and excision and primary midline closure [41]. This technique has also been compared to the Limberg flap in a randomized prospective fashion [42]. Short-term outcomes of 122 patients were analyzed and revealed that those undergoing the cleft lift had shorter operative durations, less excised tissue weight, improved pain scores, and fewer physical limitations on postoperative day 10. There were no differences in healing, complications, or early recurrences.

There is little question that this technique is easier to perform, takes less time, and removes less tissue than the more complex flap procedures such as the rhomboid flap. It results in flattening of the natal cleft, which is likely desirable. Unfortunately, not every patient with PD is a candidate for this procedure. Those with complex recurrent disease and large open wounds may not be ideal candidates, and may require more extensive flap procedures. Disease that is very close to the anus may cause difficulty with this technique, though if open wounds are able to be moved off the midline, they may still heal.

FIGURE 17-11. The area to be excised is marked. Typically this excision is performed on the side where induration or a "cyst" is located.

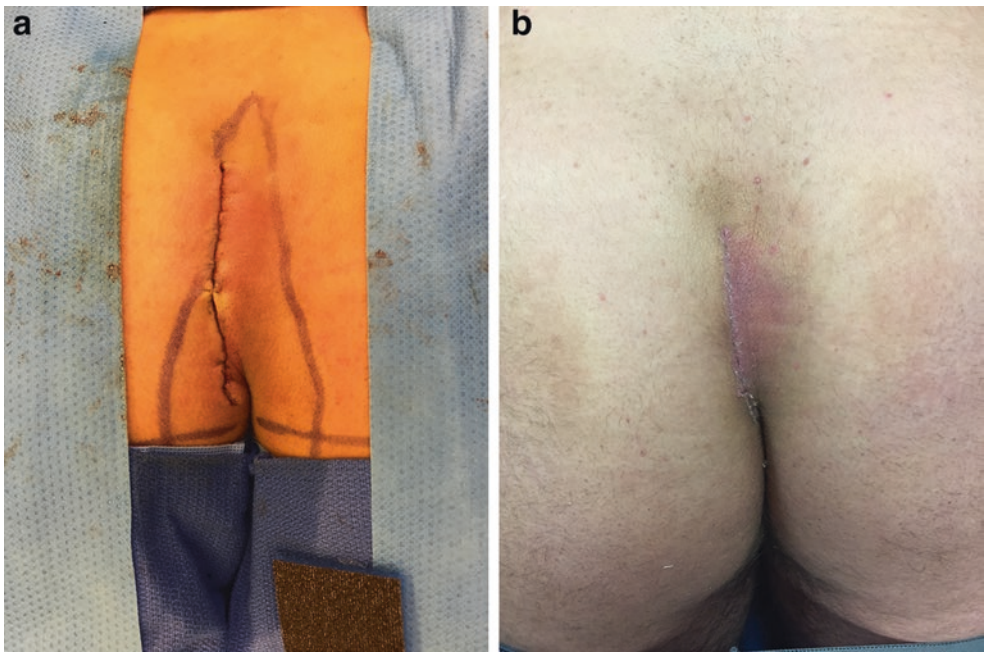


FIGURE 17-13. (**a**, **b**) These images show the procedure at the completion of the case and at 3-week follow-up with complete healing.

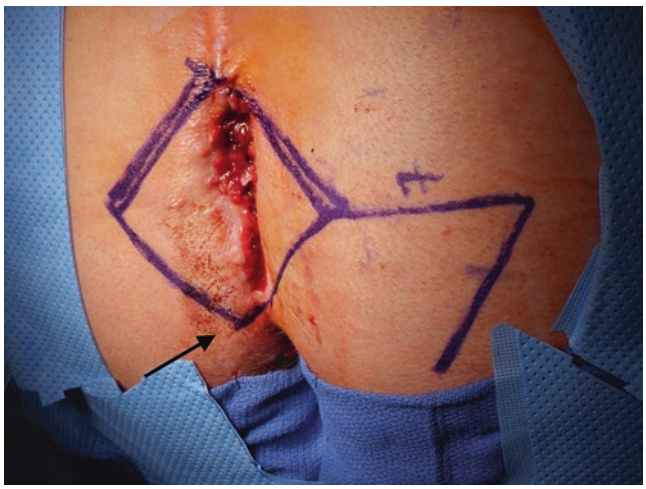


FIGURE 17-14. This image shows the planned lines of incision for the rhomboid flap. Note that the caudal tip is NOT located directly over the anus. This modification results in a wound that does not come to a point at the location of highest risk.

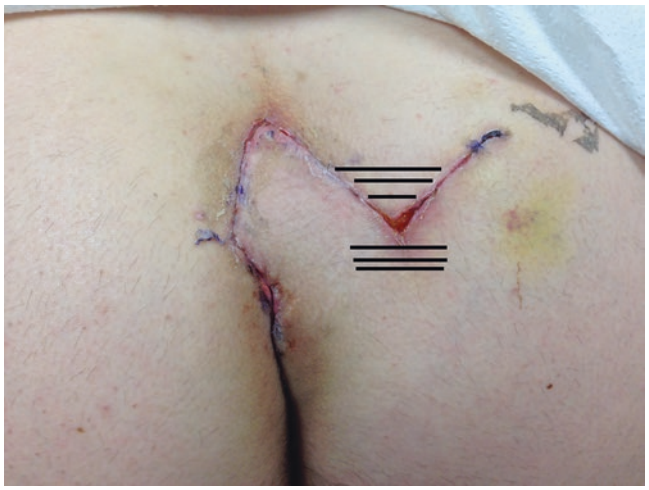


FIGURE 17-15. This image shows areas at the point of maximal tension that must be undermined to facilitate closure.

Rhomboid/Limberg Flap (See Video 17-2)

The rhomboid flap is a useful but more complex procedure that can be used in any setting of PD, but is typically reserved for more severe cases. The procedure involves a "diamond-" or rhombus-shaped area of wide excision encompassing all disease in the midline (Figure 17-14). While most will excise tissue down to the level of the post-sacral fascia, this is not entirely necessary. One must ensure however that the thickness of the mobilized lipocutaneous flap approximates the thickness of the tissue that is excised. This technique works particularly well in the setting of complex recurrent disease. The planned incision is marked, and the flap is raised with electrocautery. It is recommended to handle the flap gently during mobilization. It is important to take care to undermine the areas adjacent to the flap so that the most tension-free closure can be obtained (Figure 17-15). Once the flap is mobilized completely (Figure 17-16), it is anchored to the post-sacral tissues with an absorbable suture. A closed-suction drain is placed and a layered closure takes place using absorbable suture. The skin can be closed using a variety of techniques, none of which has proven to be superior. Some will cover the final closure with glue to create a watertight seal (Figure 17-17). A modification of this procedure was created in order to keep the caudal point of the incision away from the anus (Figure 17-14).

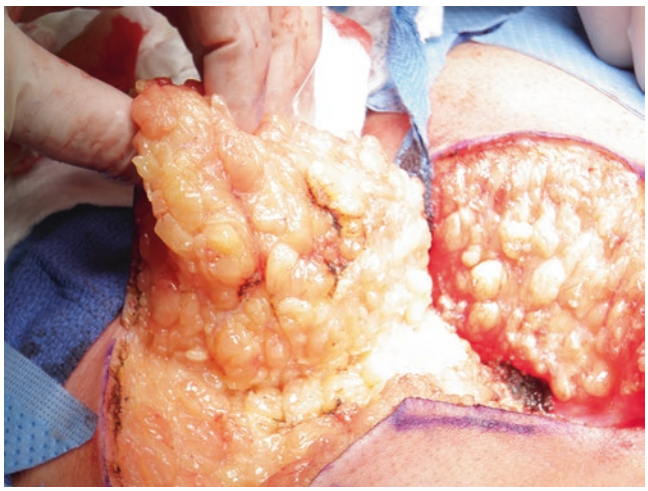


FIGURE 17-16. The Limberg flap after full mobilization, just prior to closure.

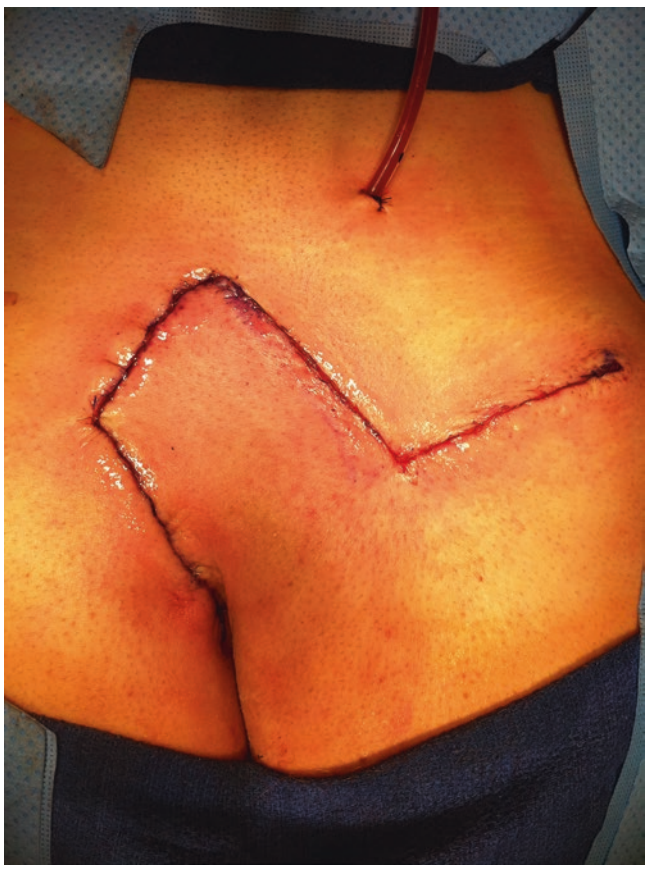


FIGURE 17-17. The appearance at the completion of the Limberg flap procedure. The wound has been covered with glue.

The drain can be left in place for 48 h or until it has produced 30 ml or less daily for 2 consecutive days. The patient should avoid any strenuous activity for 2–4 weeks. It is not uncommon for these wounds to separate slightly in one or two areas over the ensuing 2 weeks (Figure 17-18). This will require some minor wound care and is typically well

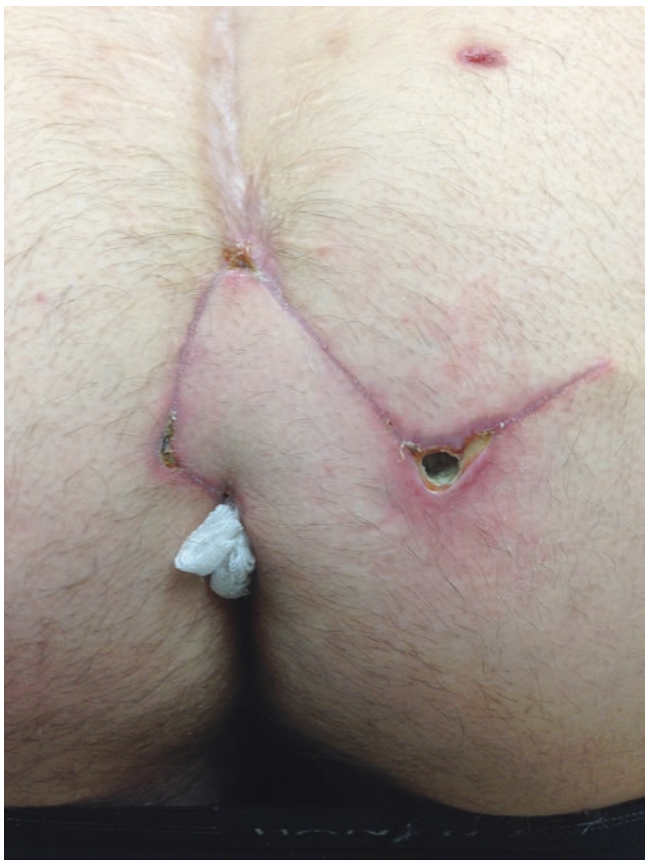


FIGURE 17-18. Follow-up will often reveal areas of minor wound separation that will require some ongoing basic wound care.

tolerated. Occasionally it will take 4–8 weeks for the wound to completely heal. In some cases, the disease spans a very large area over the sacrum extending from the perianal area for a long-distance cephalad. Many are uncomfortable creating such a large area of excision and flap in this setting. When this is the case, the technique can still be used but may be modified. The most difficult area in which to achieve healing is the caudal midline. An excision can be performed, and flap created such that the caudal midline is covered leaving an open wound cephalad (Figure 17-19). The remaining wound can be managed in a variety of ways, but the use of a negative pressure wound therapy device makes this management easy (Figure 17-20a, b). This device can be used in the standard fashion until the remaining wound is small enough to manage using standard dressings. The area will typically heal quickly, and does not impair the flap in any way.

Potential surgical site-related postoperative complications include wound dehiscence, flap necrosis, hematoma, wound infection, and seroma. These occur at rates of 4%, 0–2%, 1%, 3–5%, and 3%, respectively [43, 44]. Recurrence can be seen in approximately 4% [44]. Several series have compared outcomes associated with the Limberg flap (LF), modified Limberg flap (MLF), and excision with primary midline closure [45–48]. The evidence indicates that the LF or MLF is associated with faster return to work, lower rates of surgical

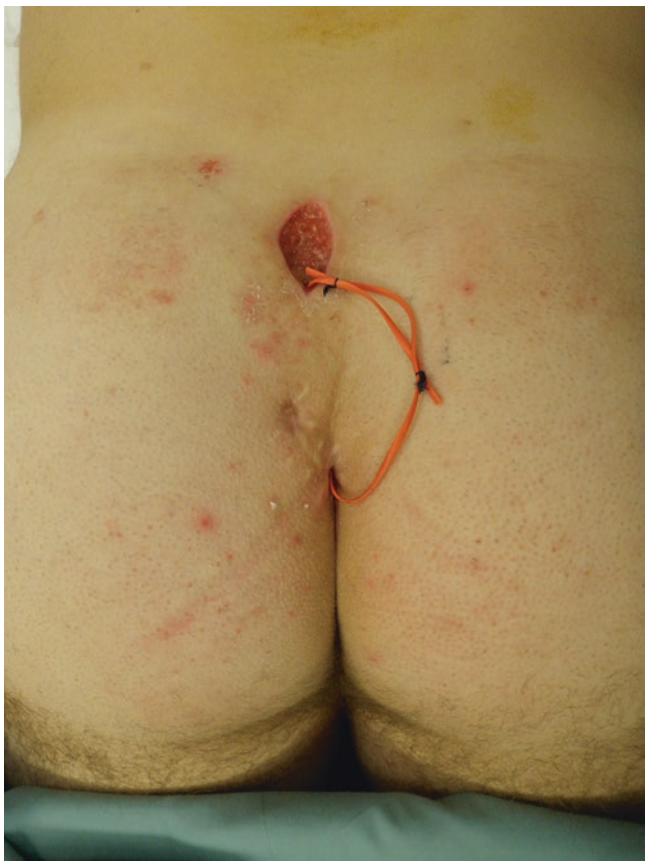


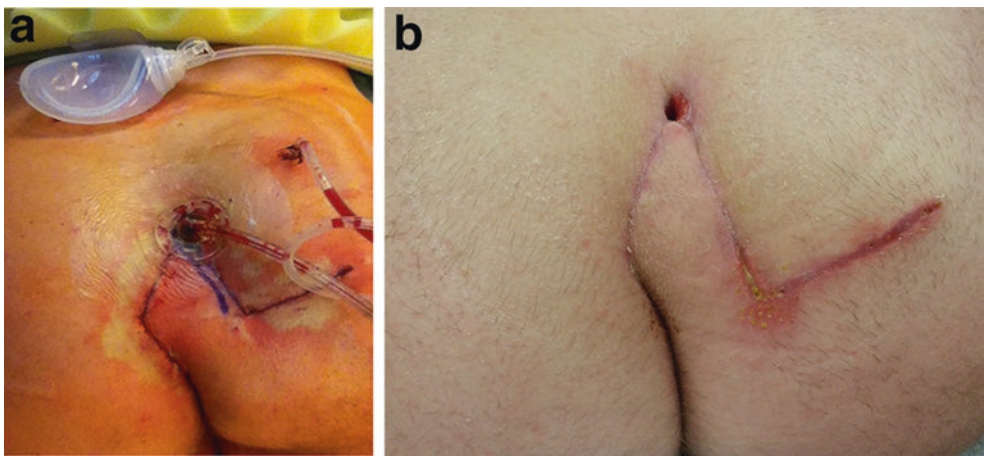
FIGURE 17-19. This image shows a patient with recurrent disease that resulted in a large abscess that was drained superiorly and required some tissue debridement. This resulted in a large area of disease to be addressed.

site infection, lower recurrence, and lower rates of wound dehiscence. Comparisons of the MLF, LF, and Karydakis flap show similar superiority for the LF and MLF [49, 50], while others have shown equivalence [51].

Disease Recurrence

Given that there are several known risk factors that predispose to the occurrence of PD, many have attempted to investigate factors that may predict disease recurrence. Familial history of disease, increased sinus number, larger cavity diameter, and primary wound closure have been shown to be associated with higher rates of recurrence [52]. Interestingly, tobacco smoking and body mass index > 25 have NOT been shown to increase recurrence [53]. Recurrence has been shown to be lower in those that undergo surgical incision and drainage prior to definitive surgery as compared to those who have spontaneous abscess rupture [54]. Along these lines, surgery performed in the "after-hours" and potentially emergent setting has been associated with higher recurrence rates [55]. Many publications that report on recurrence are criticized secondary to a lack of long-term follow-up. Doll and colleagues analyzed data from German military members and performed a telephone survey specifically investigating for recurrence [56]. They were able to demonstrate recurrence rates that were 22% higher than previously reported through collection of data over a longer period of follow-up. Recurrences up to 20 years after surgery were seen, and they recommended that studies investigating long-term outcomes should have at least 5 years of follow-up.

FIGURE 17-20. (**a**, **b**) This image shows a patient similar to that in Figure 17-19. The flap was created and closed leaving an open wound superiorly that was treated with negative pressure wound therapy and healed easily.



Hidradenitis Suppurativa

Background

The term hidradenitis suppurativa (HS), also known as acne inversa, was coined in 1864 by Verneuil [57] and literally refers to "sweat gland inflammation producing pus." The disease is a chronic inflammatory disorder involving the skin of apocrine gland-bearing areas, typically the perineum, inguinal, inframammary, and axillary regions. Colorectal surgeons are often consulted for assistance in managing those with perianal and perineal disease. Individuals afflicted with HS suffer a tremendous impact upon their quality of life with effects on both their physical and mental health [58, 59]. Practitioners in Europe have suggested that HS has the highest impact upon quality of life among all assessed dermatologic diseases [60].

The prevalence of HS is estimated to be 127.8 per 100,000 or 0.13%, with a higher prevalence among women, based on data from the Rochester epidemiology project [61]. This translates to fewer than 200,000 affected patients in the USA, 93% of which are between the ages of 18 and 64 years [62]. The reported mean age of onset is between 20 and 24 years of age, with less than 8% of affected individuals developing disease earlier than 13 years of age [63]. Early-onset disease seems to be correlated with family history of disease. When compared to psoriasis, another chronic skin disease, HS patients consume more health care and generate higher health care costs [64].

Etiology/Presentation/Diagnosis

Much like with pilonidal disease, the etiology of HS has been debated for quite some time. It was once thought purely to be secondary to infection of the apocrine sweat glands, but there is now general agreement that this is not true. The disease is characterized by chronic follicular occlusion resulting in secondary inflammation of the apocrine glands [65]. The initial inciting event is believed to be hyperkeratosis that leads to follicular occlusion [66, 67]. Others have proposed that the follicular occlusion occurs as a result of a defect in the follicular support system [68]. In any case, there is ultimate dysfunction in the entire folliculopilosebaceous unit (FPSU) that leads to follicular rupture and secondary bacterial infection involving the apocrine glands. Disease manifests initially as open comedones, typically with a few "heads," and tender subcutaneous papules [69]. In many this leads to a chronic and progressive worsening of symptoms in which additional nodules form, rupture, and drain a thick mucopurulent foul-smelling liquid. Over time this leads to sinus tract formation, fibrotic subcutaneous scarring, and potentially disabling contractures of the affected limb [69].

There are a number of variables that have been identified as risk factors for disease. Tobacco smoking and obesity have been associated with both the presence of disease and lower remission rates [70]. Weight loss has been shown to be temporally associated with remission [71, 72], with one report demonstrating disease quiescence with rapid weight loss after gastric bypass surgery. Sweating, shaving, deodorant use, and friction have also been implicated as potential exacerbating factors [73]. It is also believed that there may be dietary triggers that worsen disease (high carbohydrate diet, milk consumption) [74].

Diagnosis is typically made based on common physical exam findings including skin thickening, induration, abscess formation, the presence of draining sinuses, and contractures in the regions of the body considered at risk. There are several other diagnoses in the differential that should be considered (Table 17-1). The diagnosis can be confirmed histologically with a biopsy specimen. Given that disease can present with a wide range of severity, there have been two classification or staging systems proposed to grade disease, the Hurley system and the Sartorius system (Tables 17-2 and 17-3). The Hurley system is used more commonly as it seems to be better suited to clinical as opposed to research use. Because of some criticism related to the simplicity of the Hurley staging system, a French group have introduced a latent classification system, which better groups HS patients into three distinct phenotypes (Table 17-4) [75]. Despite its weaknesses however, the Hurley system seems to be most useful to physicians making treatment recommendations for affected individuals.

Several comorbid conditions have well-known association with HS. There is a well-established link between acne and HS, as well as with pilonidal disease [66]. Some other commonly

TABLE 17-1. A list of diagnoses that should be considered in the differential diagnosis of hidradenitis suppurative [67]

Diseases to be considered in the differential diagnosis
Acne
Actinomycosis
Anal fistula
Carbuncles
Cat scratch disease
Cellulitis
Crohn's disease
Cutaneous blastomycosis
Dermoid cyst
Granuloma inguinale
Erysipelas
Furuncles
Inflamed epidermoid cyst
Lymphadenopathy
Lymphogranuloma venereum
Nocardia infection
Noduloulcerative syphilis
Perirectal abscess
Pilonidal disease
Tuberculous abscess
Tularemia

Table 17-2. Description of the Hurley classification of hidradenitis suppurativa, likely the most useful in the clinical setting

Hurley staging system of hidradenitis suppurativa	
Stage I	Abscess formation, single or multiple, without scarring or sinus tracts
Stage II	Recurrent abscesses with tract formation and scarring, single or multiple, with widely separated lesions
Stage III	Multiple interconnected tracts and abscesses throughout an entire region

Table 17-3. The Sartorius scoring or staging system

Sartorius staging system/Sartorius score	
Involvement in specific body areas	3 points for each area involved
Nodules	2 points for each
Fistulas	4 points
Scars	1 point
Other findings	1 point
Longest distance between two lesions	2–4 points
If lesions are separated by normal skin	Yes—0 points, No—6 points

Some have modified the system by adding value to the presence of pain, drainage, or odor. This may be a more useful system in the research setting to quantify severity of disease

Table 17-4. Latent or phenotypic classification proposed by Canoui-Poitrine et al. [75]

Latent classification	Phenotype	Affected region
LC1	Axillary-mammary	Axilla, breast, perineum, inguinal
LC2	Follicular	Ears, chest, back, legs, axillary, breast
LC3	Gluteal	Gluteal fold

associated diseases include inflammatory bowel disease (particularly Crohn's disease), spondyloarthropathy, genetic keratin disorders, and squamous cell carcinoma [76]. In some cases it can be difficult to differentiate between the diseases, particularly in pilonidal disease and Crohn's disease with anal involvement. It is not entirely surprising that there can be considerable overlap in how all of these associated diseases are treated.

Treatment

As with treatment of any disease, it is important to identify the goals of therapy and patient expectations of the outcome, as well as what they will have to go through to achieve the desired end point. Medical therapy with the ultimate goal of suppression, coupled with the occasional procedure to drain an abscess, may suit a patient with Hurley stage I or II disease quite well. Conversely, the patient with Hurley stage III (Figure 17-21) disease may be so affected by their disease that they may be willing to undertake a radical surgical procedure to achieve "cure." The best way to achieve the lowest recurrence rate is to aggressively remove all apocrine gland-bearing tissue in the affected area, which will often require a complex reconstructive approach [67].

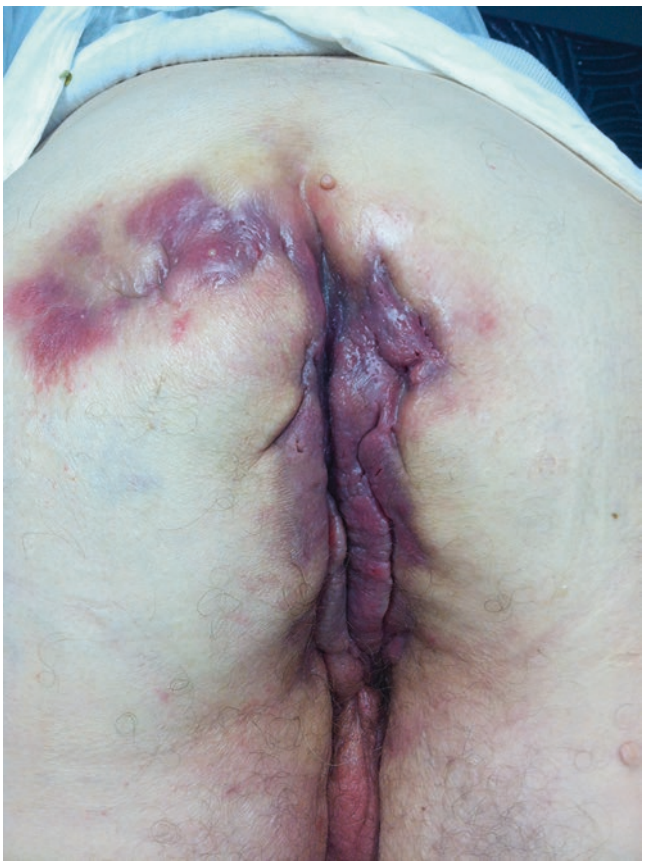


Figure 17-21. This image shows a patient with Hurley stage III hidradenitis suppurativa.

Medical Therapy

There are several different forms of medical therapy that can be considered, many of which work via different mechanisms. It appears that treatment is most successful when used in combined fashion as opposed to monotherapy [66]. Forms of medical therapy include antibacterial washes, topical antibiotics, systemic antibiotics, topical and systemic retinoids, antiandrogens, intralesional and systemic corticosteroids, and immunosuppressives [77]. Oral metformin has also been shown to be useful in treating individuals that have been unresponsive to traditional treatments [78]. Systemic antibiotics cannot be used for extended periods of time secondary to the selection of resistant strains of bacteria. While bacterial infection may be a secondary event in HS, it is clear from published research that persistence of bacterial colonization, likely in the form of biofilms, plays some role in the progression of disease [79]. Retinoids are likely beneficial secondary to their effect on normalization of epithelial cell proliferation and differentiation, which in turn may reduce the occurrence of follicular occlusion [80]. While these drugs are very effective in women of child-bearing age, their use must be cautioned due to their risk of teratogenicity. There are several reports of treatment success associated with their use [81, 82]. While antiandrogen therapy is often used (estrogen/progestin

combinations, finasteride, spironolactone), the evidence to support its use is fairly weak [83].

Given the association of HS with inflammatory bowel disease, some have suggested that HS is a systemic process and could be treated similarly [84–86]. There are several reports of the use of tumor necrosis alpha (TNF alpha) inhibitors in the treatment of HS, with infliximab supported by the majority of available data [87–94]. There is also support for the use of other TNF alpha inhibitors [95]. It may be useful to employ these newer drugs if the effect of infliximab seems to fade or if the patient develops a sensitivity to the medication. Newer reports show some success with the use of photodynamic therapy [96, 97], as well as the use of intense pulsed light therapy [98]. Lasers have been used to treat HS both superficially [99] and when used as an instrument for excision in lieu of a scalpel or other energy devices [100].

Surgical/Excisional Therapy

For patients intolerant of or unwilling to undergo medical therapy, or for those with disease of significant severity, surgical excisional therapy may present the only viable option. Excisional therapy is based on the premise that wide excision of all apocrine gland-bearing tissue in the affected region is the best method to sustain low recurrence rates. This is typically achieved through a radical approach whereby all affected skin and subcutaneous fat is excised down to the fascial level. This will often result in a very large defect that cannot be addressed through simple primary closure. Local flap closure or split-thickness skin grafting (Figure 17-22a–d) is commonly necessary to achieve adequate tissue coverage of the wound. This may require the involvement of a plastic surgeon. Attempts at simple unroofing of sinus tracts seem to be associated with higher rates of recurrence. A technique referred to as STEEP (skin tissue-sparing excision with electrosurgical peeling) has been proposed as an alternative to the above techniques [101, 102]. In this technique the sinus roof is incised with a wire-loop electrosurgical instrument, which is similar to the "unroofing" technique. Affected tissue is then tangentially excised which results in sparing of the sinus floors and surrounding subcutaneous tissue. Wounds are left to heal by secondary intention. The premise behind this technique is that it is "tissue sparing" and leads to faster healing with improved outcomes.

There are several case series reporting the outcomes associated with the use of a wide variety of radical excisional procedures employing the use of different reconstructive techniques [103–105]. Whatever technique is chosen should be based on the anatomy of disease, patient expectations, risk of recurrence, and possibility of functional limitations. Vacuum-assisted closure devices can also be helpful in wounds that are too large to close primarily, but may not require more complex reconstructive options [106]. In cases where skin grafting may be used, a two-stage approach has been described [107]. The radical excision is performed initially which is immediately followed by coverage with artificial dermis. This allows for formation of granulation tissue as well as some surrounding wound contraction which may lead to a requirement for less grafting as well as improved graft take. In almost every case, the skin graft must be placed in an area subject to high levels of motion and

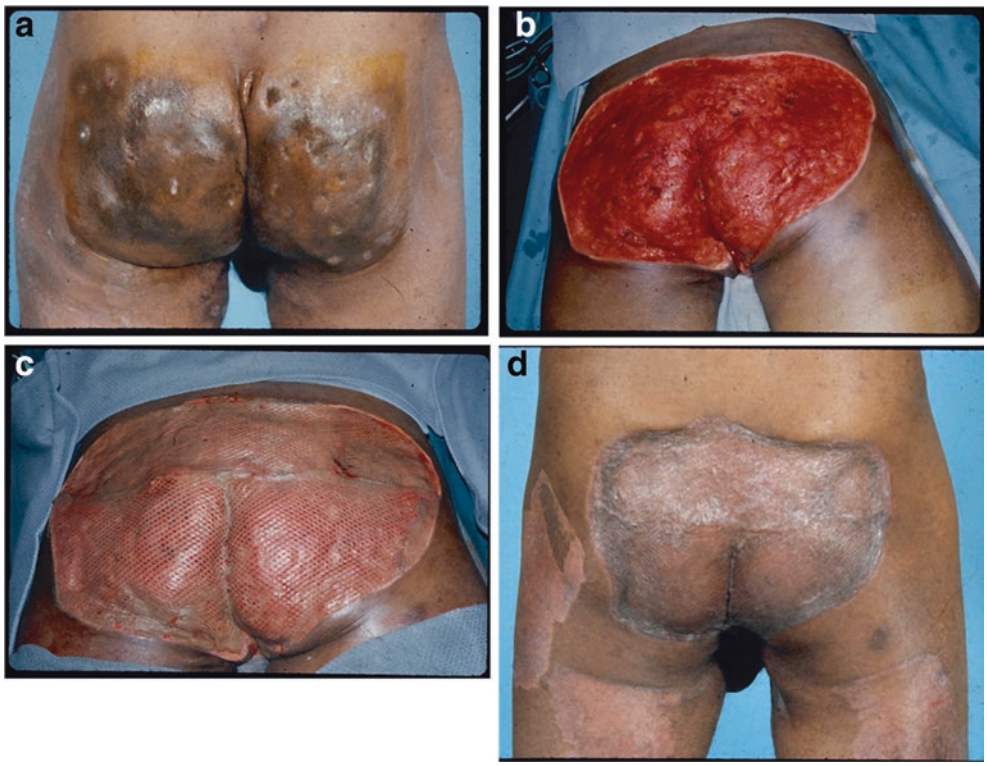


FIGURE 17-22. (**a–d**) This series of images shows a patient with Hurley stage III disease who underwent radical excision and closure with split-thickness skin grafting.

potential friction—none of which are good for a fresh skin graft. Perhaps staging the grafting approach decreases motion under the graft, which can potentially lead to improved outcomes.

Conclusion

Both pilonidal disease and hidradenitis suppurativa represent chronic inflammatory processes that can present with a wide spectrum of severity, but invariably disable those affected and result in a substantial decrease in their quality of life. While treatment of these disease processes may not seem to be surrounded in glamour, it most certainly results in a grateful patient. Pilonidal disease is quite common, while HS is much less so, but any colorectal surgeon can be expected to care for a number of individuals afflicted with these diseases. In order to ensure optimal treatment and outcomes, it is critical to tailor recommendations to the severity of disease, anatomy of disease, and our patient's expectations of risks and expected outcomes.

References

1. Hodges RM. Pilonidal sinus. Boston Med Surg J. 1880; 103:485–6.
2. Anderson AW. Hair extracted from an ulcer. Boston Med Surg J. 1847;36:74.
3. Warren JM. Abscess, containing hair, on the nates. Am J Med Sci. 1854;28:113.
4. Buie LA. Jeep disease (pilonidal disease of mechanized warfare). South Med J. 1944;37:103–9.
5. Close AS. Pilonidal cysts: an analysis of surgical failures. Ann Surg. 1955;141:523–6.
6. Von Laffert M, Stadie V, Ulrich J, Marsch WC, Wohlrab J. Morphology of pilonidal sinus disease: some evidence of its being a unilocalized type of hidradenitis suppurativa. Dermatology. 2011;223:349–55.
7. Uysal AC, Alagoz MS, Unlu RE, Sensoz O. Hair dresser's syndrome: a case report of an interdigital pilonidal sinus and review of the literature. Dermatol Surg. 2003;29:288–90.
8. Coskun A, Bulus H, Akinci OF, Ozgonul A. Etiological factors in umbilical pilonidal sinus. Indian J Surg. 2011;73:54–7.
9. Armed Forces Health Surveillance Center (AFHSC). Pilonidal cysts, active component, U.S. Armed Forces, 2000–2012. MSMR. 2013;20:8–11.
10. Bolandparvaz S, Moghadam DP, Salahi R, Paydar S, Bananzadeh M, Abbasi HR, Eshraghian A. Evaluation of the risk factors of pilonidal sinus: a single center experience. Turk J Gastroenterol. 2012;23:535–7.
11. Doll D, Matevossian E, Wietelmann K, Evers T, Kriner M, Petersen S. Family history of pilonidal sinus predisposes to earlier onset of disease and a 50% long-term recurrence rate. Dis Colon Rectum. 2009;52:1610–5.
12. Harlak A, Mentes O, Kilic S, Coskun K, Duman K, Yilmaz F. Sacrococcygeal pilonidal disease: analysis of previously proposed risk factors. Clinics. 2010;65:125–31.
13. Armstrong JH, Barcia PJ. Pilonidal sinus disease. The conservative approach. Arch Surg. 1994;129:914–7.
14. Odili J, Gault D. Laser depilation of the natal cleft—an aid to healing the pilonidal sinus. Ann R Coll Surg Engl. 2002;84:29–32.
15. Landa N, Aller O, Landa-Gundin N, Torrontegui J, Azpiazu JL. Successful treatment of recurrent pilonidal sinus with laser epilation. Dermatol Surg. 2005;31:726–8.
16. Lukish JR, Kindelan T, Marmon LM, Pennington M, Norwood C. Laser epilation is safe and effective therapy for teenagers with pilonidal disease. J Pediatr Surg. 2009;44:282–5.
17. Ghnnam WM, Hafez DM. Laser hair removal as adjunct to surgery for pilonidal sinus: our initial experience. J Cutan Aesthet Surg. 2011;4:192–5.
18. Petersen S, Wietelmann K, Evers T, Huser N, Matevossian E, Doll D. Long-term effects of postoperative razor epilation in pilonidal sinus disease. Dis Colon Rectum. 2009;52:131–4.
19. Schneider IH, Thaler K, Kockerling F. Treatment of pilonidal sinus by phenol injections. Int J Colorectal Dis. 1994;9:200–2.
20. Dogru O, Camci C, Aygen E, Girgin M, Topuz O. Pilonidal sinus treated with crystallized phenol: an eight-year experience. Dis Colon Rectum. 2004;47:1934–8.
21. Hegge HG, Vos GA, Patka P, Hoitsma HF. Treatment of complicated or infected pilonidal sinus disease by local application of phenol. Surgery. 1987;102:52–4.
22. Stansby G, Greatorex R. Phenol treatment of pilonidal sinuses of the natal cleft. Br J Surg. 1989;729–30.
23. Lund JN, Leveson SH. Fibrin glue treatment of pilonidal sinus: results of a pilot study. Dis Colon Rectum. 2005;48:1094–6.
24. Seleem MI, Al-Hashemy AM. Management of pilonidal sinus using fibrin glue: a new concept and preliminary experience. Colorectal Dis. 2005;7:319–22.
25. Greenberg R, Kashtan H, Skornik Y, Werbin N. Treatment of pilonidal sinus disease using fibrin glue as a sealant. Tech Coloproctol. 2004;8:95–8.
26. Elsey E, Lund JN. Fibrin glue in the treatment for pilonidal sinus: high patient satisfaction and rapid return to normal activities. Tech Coloproctol. 2013;17:101–4.
27. Milone M, Musella M, Di Sardo Spiezio A, Bifulco G, Salvatore G, Sosa Fernandez LM, Bianco P, Zizolfi B, Nappi C, Milone F. Video-assisted ablation of pilonidal sinus: a new minimally invasive treatment—a pilot study. Surgery. 2014; 155:562–6.
28. Alptekin H, Yilmaz H, Kayis SA, Sahin M. Volume of excised specimen and prediction of surgical site infection in pilonidal sinus procedures (surgical site infection after pilonidal sinus surgery). Surg Today. 2013;43:1365–70.
29. Milone M, Musella M, Salvatore G, Leongito M, Milone F. Effectiveness of a drain in surgical treatment of sacrococcygeal pilonidal disease. Results of a randomized and controlled clinical trial on 803 consecutive patients. Int J Colorectal Dis. 2011;26:1601–7.
30. Milone M, Di Minno MN, Musella M, Maietta P, Ambrosino P, Pisapia A, Salvatore G, Milone F. The role of drainage after excision and primary closure of pilonidal sinus: a meta-analysis. Tech Coloproctol. 2013;17:625–30.
31. Lorant T, Ribbe I, Mahteme H, Gustafsson UM, Graf W. Sinus excision and primary closure versus laying open in pilonidal disease: a prospective randomized trial. Dis Colon Rectum. 2011;54:300–5.

32. Rao MM, Zawislak W, Kennedy R, Gilliland R. A prospective randomized study comparing two treatment modalities for chronic pilonidal sinus with a 5-year follow-up. Int J Colorectal Dis. 2010;25:395–400.
33. Aldagal SM, Kensarah AA, Alhabboubi M, Ashy AA. A new technique in management of pilonidal sinus, a university teaching hospital experience. Int Surg. 2013;98:304–6.
34. Doll D, Novotny A, Rothe R, Kristiansen JE, Wietelmann K, Boulesteix AL, Dusel W, Petersen S. Methylene Blue halves the long-term recurrence rate in acute pilonidal sinus disease. Int J Colorectal Dis. 2008;23:181–7.
35. Colv EP, Bertelsen CA. Short convalescence and minimal pain after out-patient Bascom's pit pick operation. Dan Med Bull. 2011;58:A4348.
36. Olmez A, Kayaalp C, Aydin C. Treatment of pilonidal disease by combination of pit excision and phenol application. Tech Coloproctol. 2013;17:201–6.
37. Can MF, Sevinc MM, Yilmaz M. Comparison of Karydakis flap reconstruction versus primary midline closure in sacrococcygeal pilonidal disease: results of 200 military service members. Surg Today. 2009;39:580–6.
38. Can MF, Sevinc MM, Hancerliogullari O, Yilmaz M, Yagci G. Multicenter prospective randomized trial comparing modified Limberg flap transposition and Karydakis flap reconstruction in patients with sacrococcygeal pilonidal disease. Am J Surg. 2010;200:318–27.
39. Bessa SS. Comparison of short-term results between the modified Karydakis flap and the modified Limberg flap in the management of pilonidal sinus disease: a randomized controlled study. Dis Colon Rectum. 2013;56:491–8.
40. Gendy AS, Glick RD, Hong AR, Dolgin SE, Soffer SZ, Landers H, Herrforth M, Rosen NG. A comparison of the cleft lift procedure vs wide excision and packing for the treatment of pilonidal disease in adolescents. J Pediatr Surg. 2011;46:1256–9.
41. Dudink R, Veldkamp J, Nienhuijs S, Heemskerk J. Secondary healing versus midline closure and modified Bascom natal cleft lift for pilonidal sinus disease. Scand J Surg. 2011;100:110–3.
42. Guner A, Boz A, Ozkan OF, Ileli O, Kece C, Reis E. Limberg flap versus Bascom cleft lift techniques for sacrococcygeal pilonidal sinus: prospective, randomized trial. World J Surg. 2013;37:2074–80.
43. Altintoprak F, Gundogdu K, Ergonenc T, Dikicier E, Cakmak G, Celebi F. Retrospective review of pilonidal sinus patients with early discharge after limberg flap procedure. Int Surg. 2014;99:28–34.
44. Kaya B, Eris C, Atalay S, Bat O, Bulut NE, Mantoglu B, Karabulut K. Modified Limberg transposition flap in the treatment of pilonidal sinus disease. Tech Coloproctol. 2012;16:55–9.
45. Osmanoglu G, Yetisir F. Limberg flap is better for the surgical treatment of pilonidal sinus. Results of a 767 patients series with an at least five years follow-up period. Chirurgia (Bucur). 2011;106:491–4.
46. Horwood J, Hanratty D, Chandran P, Billings P. Primary closure or rhomboid excision and Limberg flap for the management of primary sacrococcygeal pilonidal disease? A meta-analysis of randomized controlled trials. Colorectal Dis. 2012;14:143–51.
47. Khan PS, Hayat H, Hayat G. Limberg flap versus primary closure in the treatment of primary sacrococcygeal pilonidal disease; a randomized clinical trial. Indian J Surg. 2013;75:192–4.
48. Dass TA, Zaz M, Rather A, Bari S. Elliptical excision with midline primary closure versus rhomboid excision with limberg flap reconstruction in sacrococcygeal pilonidal disease: a prospective, randomized study. Indian J Surg. 2012;74:305–8.
49. Sit M, Aktas G, Yilmaz EE. Comparison of the three surgical flap techniques in pilonidal sinus surgery. Am Surg. 2013;79:1263–8.
50. Arslan K, Said Kokcam S, Koksal H, Turan E, Atay A, Dogru O. Which flap should be preferred for the treatment of pilonidal sinus? A prospective randomized study. Tech Coloproctol. 2014;18:29–37.
51. Saylam B, Balli DN, Duzgun AP, Ozer MV, Coskun F. Which surgical procedure offers the best treatment for pilonidal disease? Langenbecks Arch Surg. 2011;396:651–8.
52. Onder A, Girgin S, Kapan M, Toker M, Arikanoglu Z, Palanci Y, Bac B. Pilonidal sinus disease: risk factors for postoperative complications and recurrence. Int Surg. 2012;97:224–9.
53. Sievert H, Evers T, Matevossian E, Hoenemann C, Hoffman S, Doll D. The influence of lifestyle (smoking and body mass index) on wound healing and long-term recurrence rate in 534 primary pilonidal sinus patients. Int J Colorectal Dis. 2013;28:1555–62.
54. Doll D, Matevossian E, Hoenemann C, Hoffman S. Incision and drainage preceding definitive surgery achieves lower 20-year long-term recurrence rate in 583 primary pilonidal sinus surgery patients. J Dtsch Dermatol Ges. 2013;11:60–4.
55. Doll D, Evers T, Krapohl B, Matevossian E. Is there a difference in outcome (long-term recurrence rate) between emergency and elective pilonidal sinus surgery? Minerva Chir. 2013;68:199–205.
56. Doll D, Krueger CM, Schrank S, Dettmann H, Petersen S, Duesel W. Timeline of recurrence after primary and secondary pilonidal sinus surgery. Dis Colon Rectum. 2007;50:1928–34.
57. Verneuil A. De l'hidrosadenite phlegmoneuse et des asbces sudoripares. Arch Gen Med. 1864;2:537–57.
58. Alavi A, Anooshirvani N, Kim WB, Coutts P, Sibbald RG. Quality-of-life impairment in patients with hidradenitis suppurativa. A Canadian study. Am J Clin Dermatol. 2015;16:61–5.
59. Shavit E, Dreiher J, Freud T, Halevy S, Vinker S, Cohen AD. Psychiatric comorbidities in 3207 patients with hidradenitis suppurativa. J Eur Acad Dermatol Venereol. 2015;29:371–6.
60. Zouboulis CC, Desai N, Emtestam L, Hunger RE, Ioannides D, Juhasz I, Lapins J, Matusiak L, Prens EP, Revuz J, Schneider-Burrus S, Szepietowski JC, van der Zee HH, Jemec GB. European S1 guideline for the treatment of hidradenitis suppurativa/acne inversa. J Eur Acad Dermatol Venereol. 2015. doi:10.1111/jdv.12966. [Epub ahead of print].
61. Shahi V, Alikhan A, Vazquez BG, Weaver AL, Davis MD. Prevalence of hidradenitis suppurativa: a population-based study in Olmstead County, Minnesota. Dermatology. 2014;229:154–8.
62. McMillan K. Hidradenitis suppurativa: number of diagnosed patients, demographic characteristics, and treatment patterns in the United States. Am J Epidemiol. 2014;179:1477–83.
63. Deckers IE, van der Zee HH, Boer J, Prens EP. Correlation of early-onset hidradenitis suppurativa with stronger genetic susceptibility and more widespread involvement. J Am Acad Dermatol. 2015;Pii:S0190-9622(14)02202-6. doi:10.1016/j.jaad.2014.11.017.
64. Kirby JS, Miller JJ, Adams DR, Leslie D. Health care utilization patterns and costs for patients with hidradenitis suppurativa. JAMA Dermatol. 2014;150:937–44.

65. Micheletti RG. Hidadenitis suppurativa: current views on epidemiology, pathogenesis, and pathophysiology. Semin Cutan Med Surg. 2014;33(3 suppl):S48–50.
66. Gill L, Williams M, Hamzavi I. Update on hidradenitis suppurativa: connecting the tracts. F1000Prime Rep. 2014;6:112.
67. Asgeirsson T, Nunoo R, Luchtefeld MA. Hidradenitis suppurativa and pruritus ani. Clin Colon Rectal Surg. 2011;24:71–80.
68. Danby FW, Jemec GB, Marsch WC, von Laffert M. Preliminary findings suggest hidradenitis suppurativa may be due to defective follicular support. Br J Dermatol. 2013;168:1034–9.
69. Micheletti RG. Natural history, presentation, and diagnosis of hidradenitis suppurativa. Semin Cutan Med Surg. 2014;33(3 suppl):S51–3.
70. Kromann CB, Deckers IE, Esmann S, Boer J, Prens EP, Jemec GB. Risk factors, clinical course and long-term prognosis in hidradenitis suppurativa: a cross-sectional study. Br J Dermatol. 2014;171:819–24.
71. Thomas CL, Gordon KD, Mortimer PS. Rapid resolution of hidradenitis suppurativa after bariatric surgical intervention. Clin Exp Dermatol. 2014;39:315–7.
72. Kromann CB, Ibler KS, Kristiansen VB, Jemec GB. The influence of body weight on the prevalence and severity of hidradenitis suppurativa. Acta Derm Venereol. 2014;94:553–7.
73. Von der Werth JM, Williams HC. The natural history of hidradenitis suppurativa. J Eur Acad Dermatol Venereol. 2000;14:389–92.
74. Melnik BC. Acneigenic stimuli converge in phosphoinositol-3 kinase/Akt/Foxo 1 signal transduction. J Clin Exp Dermatol Res. 2010;1:101.
75. Canoui-Poitrine F, Le Thaut A, Revuz JE, Vialette C, Gabison G, Poli F, Pouget F, Wolkenstein P, Bastuji-Garin S. Identification of three hidradenitis suppurativa phenotypes: latent class analysis of a cross-sectional study. J Invest Dermatol. 2013;133:1506–11.
76. Fimmel S, Zouboulis CC. Comorbidities of hidradenitis suppurativa (acne inversa). Dermatoendocrinology. 2010;2:9–16.
77. Rambhatla PV, Lim HW, Hamzavi I. A systematic review of treatments for hidradenitis suppurativa. Arch Dermatol. 2012;148:439–46.
78. Verdolini R, Clayton N, Smith A, Alwash N, Mannello B. Metformin for the treatment of hidradenitis suppurativa: a little help along the way. J Eur Acad Dermatol Venereol. 2013;27:1101–8.
79. Jahns AC, Killasli H, Nosek D, Lundskog B, Lenngren A, Muratova Z, Emtestam L, Alexeyev OA. Microbiology of hidradenitis suppurativa (acne inversa): a histological study of 27 patients. APMIS. 2014;122:804–9.
80. Blok JL, van Hattem S, Jonkmann MF, Horvath B. Systemic therapy with immunosuppressive agents and retinoids in hidradenitis suppurativa: a systematic review. Br J Dermatol. 2013;168:243–52.
81. Matusiak L, Bieniek A, Szepietowski JC. Acitretin treatment for hidradenitis suppurativa: a prospective series of 17 patients. Br J Dermatol. 2014;171:170–4.
82. Verdolini R, Simonacci F, Menon S, Pavlou P, Mannello B. Alitretinoin: a useful agent in the treatment of hidradenitis suppurativa, especially in women of child bearing age. G Ital Dermatol Venereol. 2014. [Epub ahead of print].
83. Alikhan A, Lynch PJ, Eisen DB. Hidradenitis suppurativa: a comprehensive review. J Am Acad Dermatol. 2009;60:539–61.
84. Marzano AV, Borghi A, Stadnicki A, Crosti C, Cugno M. Cutaneous manifestations in patients with inflammatory bowel diseases: pathophysiology, clinical features, and therapy. Inflamm Bowel Dis. 2014;20:213–27.
85. Kelly G, Sweeney CM, Tobin AM, Kirby B. Hidradenitis suppurativa: the role of immune dysregulation. Int J Dermatol. 2014;53:1186–96.
86. Dessinioti C, Katsambas A, Antoniou C. Hidradenitis suppurativa (acne inversa) as a systemic disease. Clin Dermatol. 2014;32:397–408.
87. Grant A, Gonzalez T, Montgomery MO, Cardenas V, Kerdel FA. Infliximab therapy for patients with moderate to severe hidradenitis suppurativa: a randomized, double-blind, placebo-controlled crossover trial. J Am Acad Dermatol. 2010;62:205–17.
88. Sullivan TP, Welsh E, Kerdel FA, Burdick AE, Kirsner RS. Infliximab for hidradenitis suppurativa. Br J Dermatol. 2003;149:1046–9.
89. Fernandez-Vozmediano JM, Armario-Hita JC. Infliximab for the treatment of hidradenitis suppurativa. Dermatology. 2007;215:41–4.
90. Usmani N, Clayton TH, Everett S, Goodfield MDJ. Variable response of hidradenitis suppurativa to infliximab in four patients. Clin Exp Dermatol. 2007;32:204–5.
91. Bahillo Monne C, Honorato Guerra S, Schoendorff Ortega C, Gargallo Quintero AB. Management of hidradenitis suppurativa with biological therapy: report of four cases and review of the literature. Dermatology. 2014;229:279–87.
92. Kerdel FA. Current and emerging nonsurgical treatment options for hidradenitis suppurativa. Semi Cutan Med Surg. 2014;33(3 suppl):S57–9.
93. Zhang J, Reeder VJ, Hamzavi IH. Use of biologics in the treatment of hidradenitis suppurativa: a review of the Henry Ford hospital experience. Br J Dermatol. 2014;171:1600–2.
94. Moriarty B, Jiyad Z, Creamer D. Four-weekly infliximab in the treatment of severe hidradenitis suppurativa. Br J Dermatol. 2014;170:986–7.
95. Diamantova D, Lomickova I, Cetkovska P. Adalimumab treatment for hidradenitis suppurativa associated with Crohn's disease. Acta Dermatovenerol Croat. 2014;22:291–3.
96. Fadel MA, Tawfik AA. New topical photodynamic therapy for treatment of hidradenitis suppurativa using methylene blue niosomal gel: a single-blind, randomized, comparative study. Clin Exp Dermatol. 2014. doi:10.1111/ced.12459. [Epub ahead of print].
97. Scheinfeld N. The use of photodynamic therapy to treat hidradenitis suppurativa a review and critical analysis. Dermatol Online J. 2015;21(1):Pii:13030/qt62j7j3c1.
98. Piccolo D, DiMarcantonio D, Crisman G, Cannarozzo G, Sannino M, Chiricozzi A, Chimenti S. Unconventional use of intense pulsed light. Biomed Res Int. 2014;2014:618206. doi:10.1155/2014/618206. Epub 2014 Sep 3.
99. Tierney E, Mahmoud BH, Hexsel C, Ozog D, Hamzavi I. Randomized controlled trial for the treatment of hidradenitis suppurativa with a neodymium-doped yttrium aluminum garnet laser. Dermatol Surg. 2009;35:1188–98.
100. Hazen PG, Hazen BP. Hidradenitis suppurativa: successful treatment using carbon dioxide laser excision and marsupialization. Dermatol Surg. 2010;36:208–13.
101. Blok JL, Spoo JR, Leeman FWJ, Jonkman MF, Horvath B. Skin-tissue-sparing excision with electrosurgical peeling (STEEP):

a surgical treatment option for severe hidradenitis suppurativa Hurley stage II/III. J Eur Acad Dermatol Venereol. 2014.
102. Blok JL, Boersma M, Terra JB, Spoo JR,Leeman FW, van den Heuvel ER, Huizinga J, Jonkman MF, Horvath B. Surgery under general anesthesia in severe hidradenitis suppurativa: a study of 363 primary operations in 113 patients. J Eur Acad Dermatol Venereol. 2015. doi:10.1111/jdv.12952. [Epub ahead of print].
103. Alharbi Z, Kauczok J, Pallua N. A review of wide surgical excision of hidradenitis suppurativa. BMC Dermatol. 2012;12:9.
104. Mizukami T, Fujiwara M, Ishikawa K, Aoyama S, Fukamizu H. Reconstruction for extensive groin hidradenitis suppurativa using combination of inferior abdominal flap and medial thigh-lift: a case report. Aesthetic Plast Surg. 2014;38:745–8.
105. Chen ML, Odom B, Santucci RA. Surgical management of genitoperineal hidradenitis suppurativa in men. Urology. 2014;83:1412–7.
106. Chen YE, Gerstle T, Verma K, treiser MD, Kimball AB, Orgill DP. Management of hidradenitis suppurativa wounds with an internal vacuum-assisted closure device. Plast Reconstr Surg. 2014;133:370e–7.
107. Yamashita Y, Hashimoto I, Matsuo S, Abe Y, Ishida S, Nakanishi H. Two-stage surgery for hidradenitis suppurativa: staged artificial dermis and skin grafting. Dermatol Surg. 2014;40:110–5.