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Case Report Open Access

Hidradenitis Suppurativa

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Introduction

Hidradenitis Suppuritiva (HS) is a chronic follicular occlusive disease involving the apocrine glands of intertriginous areas of the body, including axilla, groin, genitalia, buttocks and perianal regions. There is a spectrum of disease ranging from abscess formation to chronic, painful, draining lesions with sinus tract and fistula formation. It affects up to 4% of the population [1] and causes significant pain, functional limitation and psychosocial distress for affected patients [2]. In cases of genital/perineal involvement, HS can have a profound impact on quality of life, sexual function and overall well-being [3]. Severe, recurrent cases of the disease are not amenable to medical therapy[4].Wide surgical resection is the only curative therapy for HS, yet there is no consensus on surgical techniques that produce the most favorable outcomes in recalcitrant cases of this difficult to treat disease. This article provides a review of the evidence for different techniques including both the medical management, but more extensively the surgical options for treatment. We will also provide case examples of hidradenitis where patients underwent widespread excision and split-skin grafting.

Pathophysiology

Hidradenitis is a disease resulting from follicular occlusion. The process is theorized to begin due to obstruction of the hair follicles by demonstrating ductal hyperkeratosis and follicular hyperplasia [5]. Microscopic examination of the tissues and structure of hair follicles from individuals with hidradenitis shows that there are alterations in the basement membrane zone of the follicle and sebofollicular junction [6]. One group demonstrated that abnormal basement membrane zones might be associated with pilosebaceousrupture and escape of keratinocytes into the subcutaneous tissues laterally, resulting in inflammation and super infection [5].

Infection begins via bacteria traveling through the hair follicle, into the apocrine glands where the subsequently become trapped beneath the keratinous plug and then thrive in this nutrient rich environment [7]. Bacteria commonly observed in specimens include coagulase negative Staphylococcus, Corynebacterium, Streptococcus, and in our practice, Proteus. The natural history of the disease is the result of cyclical inflammation following the inciting event. The cavity becomes lined with squamous epithelium that is colonized with skin flora.

Hurley described the progression of hidradenitis with a staging system to measure the severity of the disease as follows[8]:

- Stage I: formation of abscesses without sinuses.
- Stage II: recurrent abscess with tract formation and scarring, without sinuses.
- Stage III: abscess with scarring, coalescence of sinus tracts and draining sinuses.

Psychology

Hidradenitis causes profound quality of life issues. This is due to chronic pain, odor, drainage, and illness. The result is days of work lost, low self-confidence, impaired sexual health, and even depression in some patients. Most of the disability of HS stems from the soreness and pain of the lesions [9]. A study by Matusiak et al. revealed that of the 30 employed patients, 58.1% had reported absence from work caused by HS, and during the 2 year follow up period, 10% were let go due to the frequency of their absences [10].

Of particular importance, it was demonstrated that patients with HS suffer more sexual dysfunction compared to age, gender, and BMI matched controls [1]. Interestingly, it was found that women report greater sexual distress than men with HS of similar severity [1]. This may indicate that female patients with hidradenitis should be screened more closely for sexual dysfunction [1].

Additionally, a survey-based study by Von Der Werth and Jemec found that hidradenitis led to lower quality of life scores than other chronic skin conditions such as psoriasis and acne [9]. Depression is more prevalent in sufferers of HS as well, Vazquez et al. found that over 40% of patients with HS had a diagnosis of depression [1,11]. Thus, it would be prudent for physicians to have a lower threshold when considering referral for psychological evaluation. Overall, improvements in management of this disease would likely relieve many of these psychological sequelae.

Associated Factors

Although the precise etiology of HS remains unclear, there are a number of associated factors that have been consistently described. Many of these pre-disposing factors have insufficient or inconclusive evidence to support them. Of the factors that are well-established associations, strong evidence to explain causation remains lacking.

There is a well-recognized association between cigarette smoking and HS [1]. Cross-sectional analyses have demonstrated a strong epidemiologic correlation, with study populations reporting from 70% to as high as 90% of disease sufferers being documented smokers. Some studies have demonstrated remission of disease with smoking cessation, while others show decreased rates of recurrence of disease after surgical resection with concomitant smoking cessation post-operatively.

The role of sex hormones has been extensively reported but a clear consensus has not been reached on this topic. Evidence of hormonal

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contribution is derived from the observation that HS rarely affects pre-pubertal of post-menopausal populations. Androgens have been implicated due to their role in hyperkeratinization, a pathophysiologic feature shared with acne vulgaris. There is evidence that anti-androgenic medications like finasteride reduce HS severity. The use of OCP's has been known to induce or exacerbate HS, while using anti-androgen progesterone containing OCP's has been shown to reduce disease severity. Thus, hormones play a role in the disease process, requires further study.

Obesity is another commonly reported associated factor in HS. Cross-sectional studies vary in the reported rates of obesity among HS suffers but it is clearly the majority with ranges from 54-82%. Most authors offer a large body habitus explanation for this linkage, arguing that shearing forces stimulate hyperkeratinization and follicular rupture while areas of large skin folds encourage warm, humid microenvironments where bacteria flourish. Additionally, obesity is a sub-acute, chronic inflammatory state with alterations in blood sugar metabolism. And there is evidence demonstrating higher rates of HS in Type II diabetes mellitus, metabolic syndrome and acromegaly. However, the exact causal link is yet to be determined.

There is a genetic component to HS as evidenced by the fact that up to 40% of disease suffers report a known relative with the disease. The role is likely polygenetic, as no clear pattern of inheritance or single gene association has been demonstrated. There have been no monozygotic twin studies examining the role of genes in development of HS. Noting the fact that HS is a chronic-inflammatory disease, researchers have looked for a correlation with HLA phenotype but none has been observed.

Medical Management

The most common medical treatments hidradenitis include antibiotics, retinoids, and immune modulators. The most commonly used and most recently studied are antibiotics, and immune modulators.

Antibiotics

Antibiotic therapy has been used with variable success for the treatment. The most common therapies involve clindamycin as it has a broad coverage for skin flora. Combinations of clindamycin and rifampin have been shown to be helpful though evidence is weak [12]. In a study by van der Zee et al., a combination of clindamycin and rifampin over a 10 week course was 82% effective for improvement, and benefited 47% of patients with remission [13]. However, after completion of antibiotic therapy 61% of patients had recurrences after 5 months [13]. Other rifampin combinations have not had the same successes; recent study evaluation rifampin-moxifloxacin-flagyl required 12 months of therapy to reach effect in 57% of patients [14]. Additionally groups have tested dapsone showing 62% non-efficacy, with an 8% cessation in treatment for side effects [15]. A notable finding of the review is the duration of therapy required before effect is attained. The collaborative community treating hidradenitis must note the cost burden and the medical, societal, and bacterial consequences of using antibiotics in this manner.

Immunomodulators

A recent emergence in medical therapy of hidradenitissuppurtiva is the use of immunomodulating drugs. Currently there is emerging data on the use of the medications however studies are of limited power and there is not enough evidence to develop a standardized set of guidelines; both efficacy and long term data are poor [16]. Anti-proliferative drugs such as colchicine have been met with mixed success [17,18]. Immunomodulation through monoclonal antibody drugs have also shown variable efficacy. Infliximab has been shown to lack of response in 20% and relapse in 50% after 37 week treatment courses [19]. Two recent studies on adalimumab have shown variable clinical response of 17% to 100% demonstrating that higher powered studies are needed to elucidate the topic [20,21]. The common drawback to adalimumab therapy is the high rate of recurrence, 62% as shown by Arebergerova et al. [20].

Etanercept is another medication which has been recently studied in the immunomodulator class that has been met with variable success. Adams et al. showed no difference in outcome from the placebo group [22]. Another study on etanercept showed 30% no change, 30% long term disease improvement and effectiveness, and a 30% requiring additional dosing after 1 year for recurrence of disease; a similar efficacy rate was also observed by Lee et al. at 20% [23,24]. The required total dose and time to effect was shown to be higher in patients with more severe disease show by Pelekanou et al., requiring up to 24 weeks in severe hidradenitis to become efficacious; the time to recurrence in this study of Etanercept was 4-8 weeks [24].

The concern for the various medical treatments is the duration of required treatment, and the long term exposure to potential side effects. Antibiotics have been shown to cause GI distress, hematologic alterations, mood changes, malaise [25]. Anti-TNF agents have been shown to cause infectious complications myalgias, hepatitis, lipid alterations, neuralgias, headache, and GI distress [25]. Additionally, medical therapy has poor overall efficacy with a high rate of recidivism. Given the high cost of medicines, and the high rate of failure, more study is required to determine the true cost effectiveness and utility of some of these immune modulating drugs.

Surgical management

Surgical options have been well accepted to offer the best chance at a cure for advanced disease [26,27]. Though medical management may lead to symptomatic relief, studies have suggested that it is unlikely that these treatments change the natural course of HS [28-30]. There are several options for surgical management including incision and drainage, deroofing, local excision with primary closure, and wide excision techniques. Selection of the treatment should take into consideration the severity, the areas affected, the duration of the disease, and the patient's comorbidities [27,31].

Incision and drainage has been shown to have nearly 100% recurrence and its role in management is typically a temporary measure utilized in the acute phase [31-33]. Deroofing involves removing the "roof" of a sinus, cyst, or abscess with electrosurgery, while leaving the floor epithelium unaffected and thus re-epithelialzation occurs more rapidly [34]. Yet another option is local excision with primary closure. As with deroofing, this technique allows for a greater conservation of tissue, however studies have shown a significantly higher rate of recurrence when compared to more radical exsions [7,3,34]. Ritz et al. demonstrated 100% recurrence with incision and drainage, 42.8% after limited, and 27% after more extensive excision ($p<0.5$) [35].

Thus, wide excision has been extensively demonstrated to offer patients the lowest chance of recurrence and therefore, the highest possibility of cure for HS, particularly when the disease is more severe

[7,27,30,31]. Wide excision involves removal of all of the affected areas until healthy, normal tissue is reached with a margin clearly beyond the diseased site [7]. Margins of 1-2cm have been advocated in the literature [7,29,34]. While wide excision has been confidently shown to offer the best chance at a cure, the most superior method for closure is still under debate. Options include primary closure, healing by secondary intention, skin grafting, and flap coverage. The rate of recurrence may be higher with primary closure and flap techniques, perhaps due to inadequate excision to allow for primary closure, as well as the possible transfer of the same diseased tissue into the newly excised area [36].

Another option for reconstruction is skin grafting. Several studies have reported good results with the use of split-skin grafting yet there is no consensus on surgical techniques that produce the most favorable outcomes in recalcitrant cases of this difficult to treat disease. We have provided some cases where we have had success with wide surgical excision followed by skin grafts or closure.

Case Examples

Case 1 (COX): This is a 48 year old black man who presented with extensivehidradenitissuppurativa in the inguinal and anogenital region medically managed with Remicadec (Figures 1-3). He underwent widespread excision and split-thickness skin graft using cadaver graft and autologous skin graft to the groin and perineum (Figures 4-6). At F/U two weeks later, patient had small areas of hypertrophic granulation tissue which were treated with silver nitrate, but there was no recurrence of HS (Figure 7). At 6 weeks, patient is doing well with no recurrence (Figure 8).

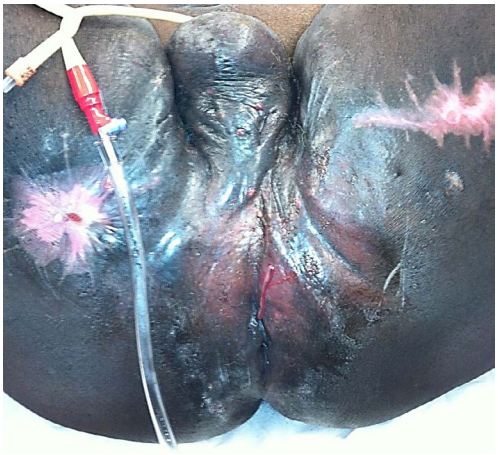

Figure 1: Extensive anogenital hidradenitis with sinus tract and fistula formation.

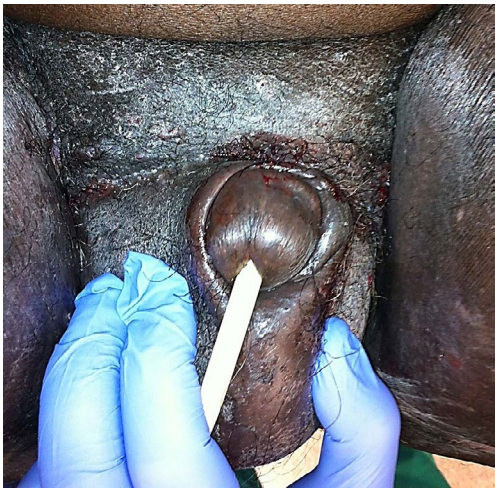

Figure 2: Extensive hydradenitis in genital and inguinal regions with penile skin fibrosis and restriction.

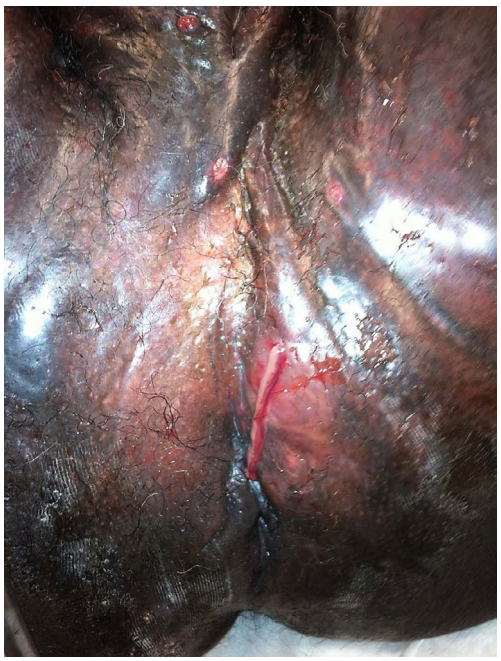

Figure 3: Case 1 – Perineal hidradenitis with sinus tract and fistula formation and active drainage.

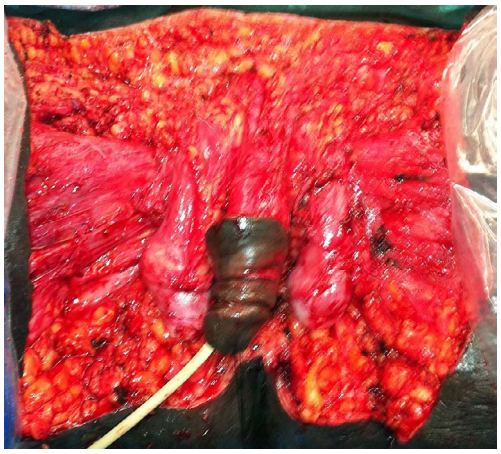

Figure 4: Anogenital Hidradenitis Suppurativa after wide surgical excision.

Case 2: This is a 45 year old black man who presented with widespread anogenitalhidradenitis with extension to the inguinal and buttock regions (Figures 9 and 10). He underwent widespread excision and split thickness skin graft using autologous skin graft both to the groin and perineum (Figures 11-15). At 6 week F/U patient has had complete healing (Figures 16 and 17).

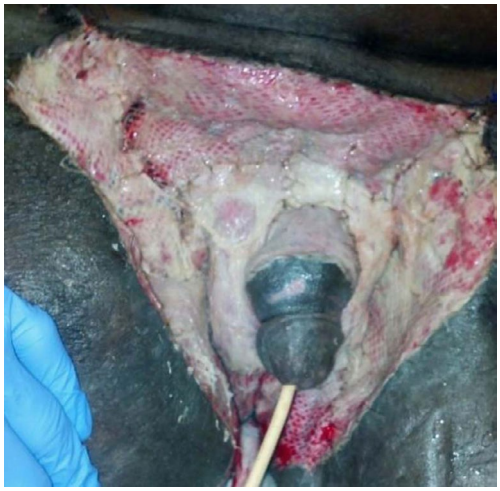

Figure 5: Anogenital Hidradenitis Suppurativa after placement of cadaver graft.

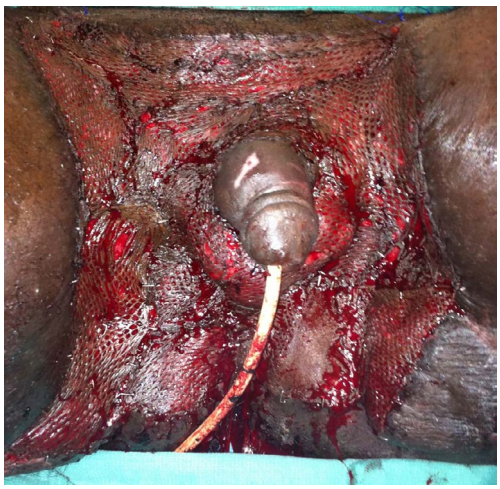

Figure 6: Anogenital Hidradenitis Suppurativa post-operative photo after placement of autologous split-skin graft.

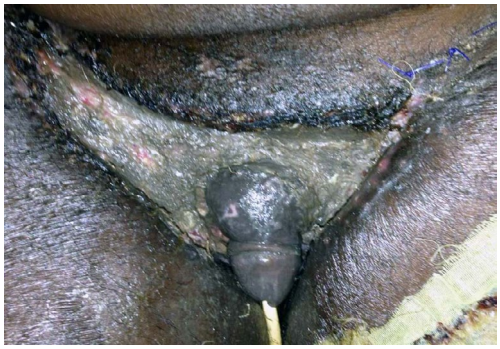

Figure 7: Anogenital Hidradenitis Supprativa –photo of cadaver graft and autologous graft at 2 weeks follow-up visit.

Case 3: This is a 28 year old black woman with no history of smoking hidradenitis in bilateral axillas, under her breasts, and in her groin, vulva, and perineum (Figure 18). Prior to her surgery, she was initially treated with antibiotics (minocycline, clindamycin) triamcinolone

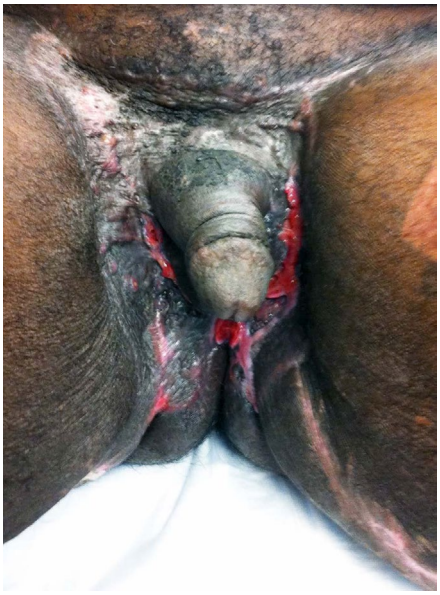

Figure 8: Anogenital Hidradenitis Supprativa –photo of cadaver graft and autologous graft at 6 weeks follow-up visit demonstrates almost complete healing with some hypertrophic granulation tissue.

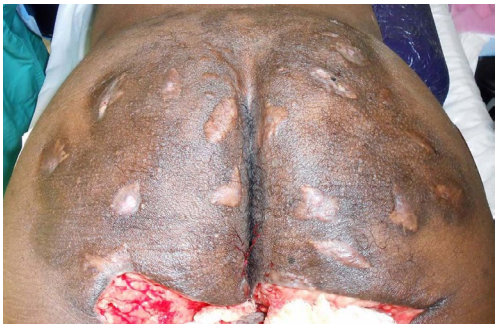

Figure 9: Extensive buttocks hidradenitis Suppurativa before wide excision.

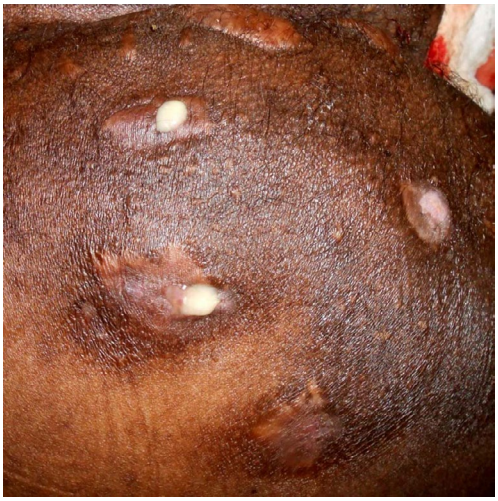

Figure 10: Extensive buttocks hidradenitis Suppurativa intra-op image showing multiple sinuses draining pus.

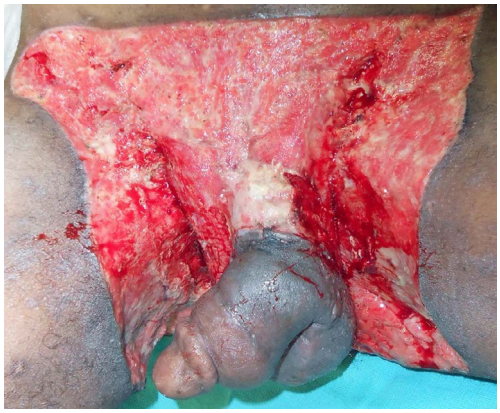

Figure 11: Extensive bilateral inguinal Hidradenitis Suppurativa after wide excision.

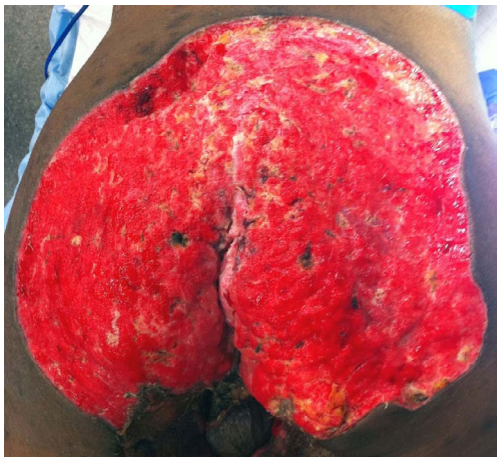

Figure 12: Extensive buttocks hidradenitis Suppurativa after wide excision.

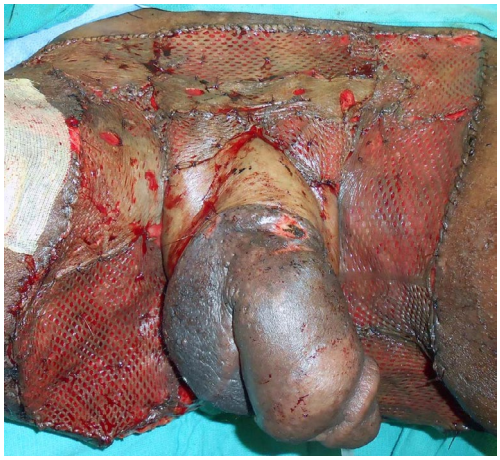

Figure 13: Extensive bilateral inguinal Hidradenitis Suppurativa post-operative photo after autologous split skin graft placement.

topical to each lesion, Adalimubab (Humira) 40mg Qweekly, and Prednisone 40 mg daily. A wide excision without skin grafting was performed (Figures 19 and 20). At 3 week follow up, patient was doing well, and her wounds were becoming smaller, and there was no drainage. By 6 weeks, the wound underneath her left breast completely

Conclusion

There are various modalities in treating hidradenitissuppurativa. Surgery is a therapeutic an effective option for severe cases that have had minimal response to conservative modalities. While incision and drainage may provide relief, it is temporary, and patients who have chronic or more severe cases that have sinus and fistula formation

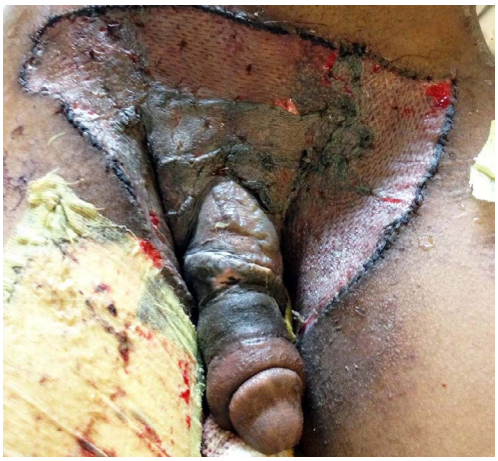

Figure 14: Extensive bilateral inguinal Hidradenitis Suppurativa after wide excision and split thickness skin graft at 2 week follow up visit.

Figure 15: Buttocks hidradenitis after wide excision and split thickness skin graft at 2 week follow up visit.

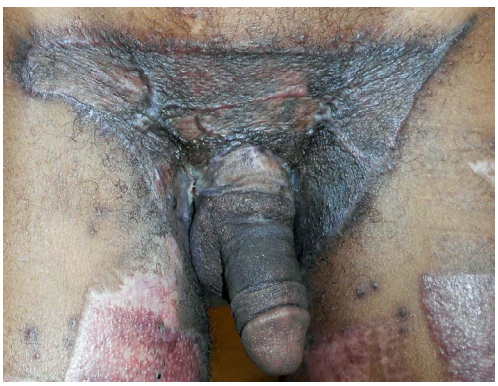

Figure 16: Extensive bilateral inguinal Hidradenitis Suppurativa after wide excision and split thickness skin graft at 6 week follow up visit.

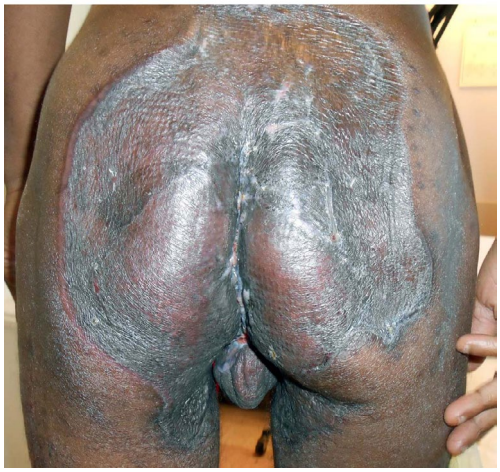

Figure 17: Extensive buttock Hidradenitis Suppurativa after wide excision and split thickness skin graft at 6 week follow up visit.

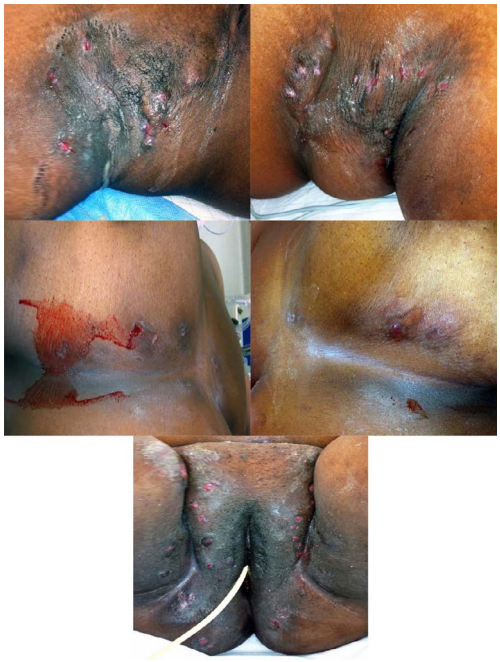

Figure 18: Case 3. Hidradenitis under right axilla (top left), left axilla (top right), right breast (middle left), left breast (middle right), groin/vulva/perineum (bottom).

may need another modality besides medical treatment. Wide surgical excision along with split skin grafting can be done to achieve the best outcome for such patients. Factors that influence the type of surgical approach are the sites affected, the amount of skin and soft tissue involved, and the chronicity of the disease.

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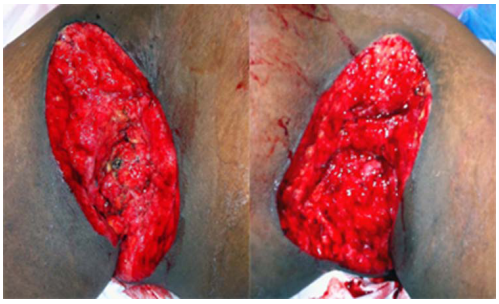

Figure 19: Bilateral axillary hidradenitis supporativa after wide surgical excision - right axilla(left); left axilla (right).

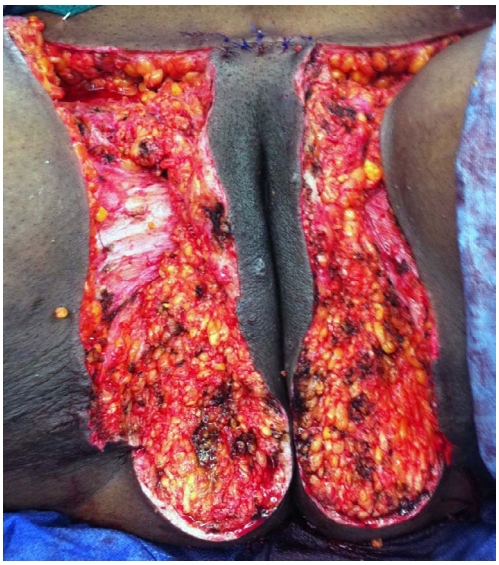

Figure 20: Hidradenitis supporativa in the vulva regions after wide surgical excision.

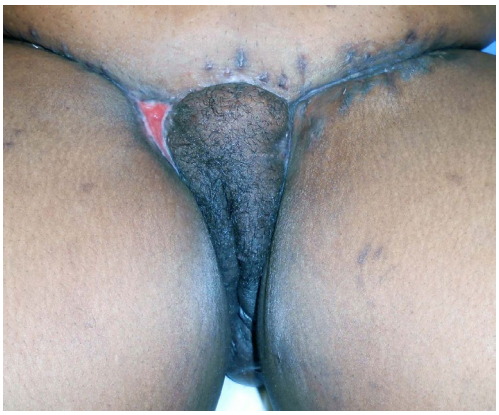

Figure 21: Vulva Hidradenitis suppurativa after excision and closure photo at 3 week follow up visit.

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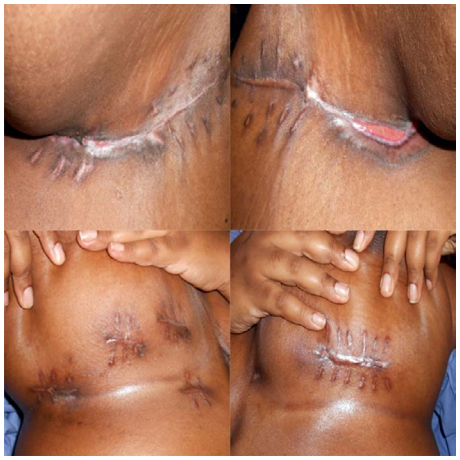

Figure 22: Hidradenitis after excision and closure in right axilla (top left), left axilla (top right), right breast (bottom left), left breast (bottom right) at 3 week follow up visit.

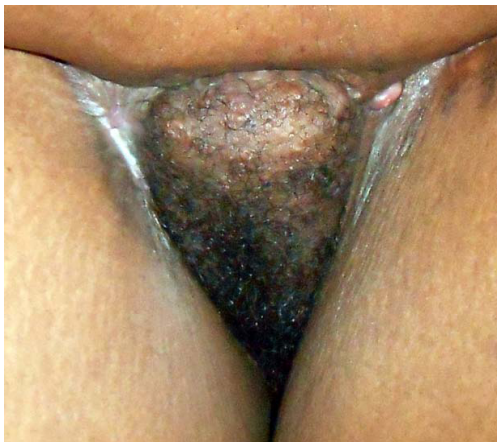

Figure 23: Vulva Hidradenitis suppurativa after excision and closure photo at 3 month week follow up visit.

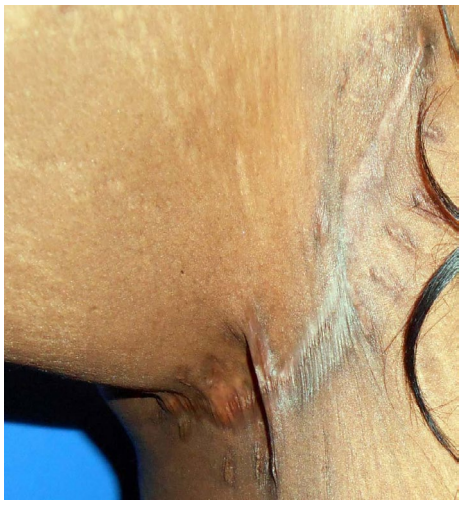

Figure 24: Right axillary Hidradenitis suppurativa after excision and closure photo at 3 month follow up visit.

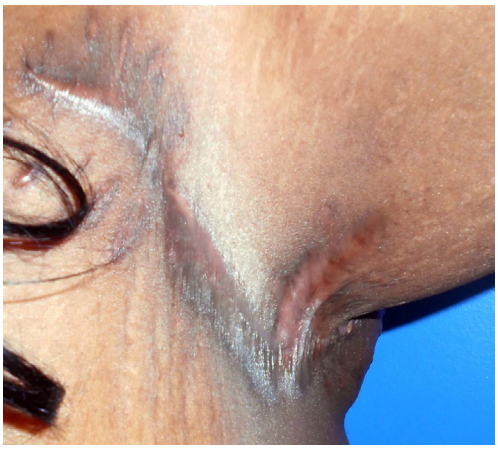

Figure 25: Left axillary hidradenitis suppurativa after excision and closure photo at 3 month follow up visit.

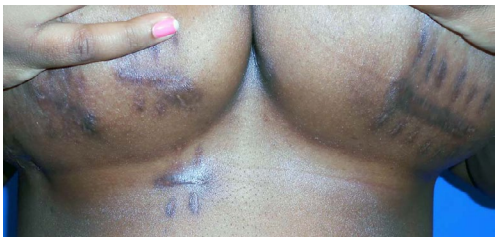

Figure 26: Bilateral breasts Hidradenitis suppurativa after excision and closure photo at 3 month follow up visit.

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