Plastic Surgical Management of Hidradenitis Suppurativa

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PATIENT SAFETY

Learning Objectives: After studying this article, the participant should be able to: 1. Describe the pathogenesis of hidradenitis suppurativa. 2. Discuss perioperative multimodal therapy of hidradenitis suppurativa, including medical optimization. 3. Determine an appropriate surgical plan with excision and reconstruction based on hidradenitis suppurativa severity, size, and anatomical location.
Summary: Successful treatment of hidradenitis suppurativa requires a multidisciplinary team approach and multimodal therapy. (*Plast. Reconstr. Surg.* 147: 479e, 2021.)

Hidradenitis suppurativa, or acne inversa, is a debilitating inflammatory disease of the folliculopilosebaceous units, often resulting in malodorous drainage, physical discomfort, and psychological distress. This disease frequently manifests as nodules, abscesses, scars, and sinus tracts in the axillae, groins, perineum, buttocks, and inframammary areas. Because areas affected by hidradenitis suppurativa are often covered with clothes, the disabling social stigma may contribute to the variability in the reported incidence of the disease, which ranges from 0.05 to 4.1 percent of the population in the literature.[1] Recent work and increasing expert consensus suggest the prevalence is likely to be approximately 1 percent.[2] Hidradenitis suppurativa presents most often in the third and fourth decades of life, with epidemiologic prevalence in women (3:1 female-to-male ratio) and in those of African descent.[1,3,4]

Recent advances in treatment modalities, understanding of pathophysiology, and an increasing presence of organized patient groups with this disease have given new hope to patients suffering from hidradenitis suppurativa.[3,5] This review focuses on the current landscape of multimodal treatment of hidradenitis suppurativa, with specific emphasis on the surgical management of acute and chronic lesions, reconstructive options, and anatomical considerations to guide surgical decision-making.

PATHOGENESIS

Occlusion of the folliculopilosebaceous unit, which includes the hair follicle, sebaceous glands, and apocrine glands, leads to rupture of the sebofollicular canal, which leads to perifollicular lymphohistiocytic inflammation.[6] Anatomical regions affected by hidradenitis are regions with high concentration of folliculopilosebaceous units, including axillary, inguinal, perineal, gluteal, inframammary, and lower abdominal regions. Axillary hidradenitis is a common anatomical site, seen in approximately 61 percent of patients undergoing surgical excision.[4,7] Of patients with axillary hidradenitis, approximately one-third have bilateral axillary disease and 8 percent have extra-axillary involvement.[8] The inguinal region is the most commonly affected area,

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involving approximately 70 percent of patients undergoing surgical excision.[7] Lesions also commonly involve the perineum, labia majora, scrotum, and penis. Gluteal hidradenitis presents with tracts throughout the gluteal soft tissue, often with extensive tunneling. Breast and lower abdominal hidradenitis lesions often present in overweight patients.

No predominant mutation across the spectrum of disease has to date been identified, but genetic mutations associated with hidradenitis suppurativa include the γ-secretase complex/Notch signaling pathway, tumor necrosis factor (TNF) single nucleotide polymorphisms, interleukin (IL)-12RB1 mutation, and type 1 and type 17 T-helper cell dysregulation.[3] Biomarkers associated with hidradenitis suppurativa include increased erythrocyte sedimentation rate, C-reactive protein, TNF, IL-6, and IL-17A. IL-17A increases the production of cytokines, chemokines, metalloproteinases, and antimicrobial peptides that participate in the inflammatory response. IL-17A is dramatically increased in both gene expression and mRNA expression when comparing hidradenitis suppurativa lesions with normal skin.[9–11] A decrease in leukotriene A4 hydrolase, follicle stimulating hormone, human chorionic gonadotropin, and luteinizing hormone are also associated with hidradenitis suppurativa.[3]

Hidradenitis suppurativa has been associated with numerous comorbidities. Obesity is the most common comorbidity, affecting 75 percent of patients with hidradenitis suppurativa. The incidence of type 2 diabetes mellitus and polycystic ovarian syndrome is 1.5 to 3 times higher in affected patients.[3,12,13] Among patients who lost over 15 percent of their body weight, nearly half had complete remission and 20 percent had substantial improvement of hidradenitis suppurativa lesions.[10] Bariatric surgery has been associated with a 35 percent decrease in self-reported hidradenitis suppurativa with a reduction in the mean number of lesions from 1.9 to 1.2. However, concomitant skin reduction surgery may be important, as some patients also report worsening disease under redundant skin folds. Additional studies suggest that patients with hidradenitis suppurativa have associated conditions of acne, autoinflammatory syndromes, thyroid disease, inflammatory bowel disease, pyoderma gangrenosum, inflammatory arthropathies, lymphoma, Down syndrome, depression, and psychiatric conditions.[3]

Hidradenitis suppurativa is commonly seen in tobacco smokers, with 75 percent of hidradenitis suppurativa patients actively smoking and 15 percent of hidradenitis suppurativa patients previously smoking.[3] Dietary restrictions with some suggestion of improvement include dairy avoidance, brewer's yeast (*Saccharomyces cerevisiae*) avoidance, and zinc supplementation. No link has been found between hidradenitis suppurativa and shaving, deodorants, or antiperspirants.[14] Friction, and how to avoid it, has an unclear link to hidradenitis suppurativa, with one survey reporting worsening of symptoms with tight clothing in 16 percent of patients and improvement of symptoms with loose or cotton clothing in 11 percent of patients.[15,16]

CLASSIFICATION

The Hurley staging system provides an intuitive and simple classification framework to guide treatment, reflects overall disease severity over time, and was designed to guide surgical management (Fig. 1 and Table 1). Mild disease (Hurley stage I) is characterized by recurrent nodules and abscesses but minimal scar; moderate disease (Hurley stage II) by recurrent nodules, abscesses, and a limited number of scar or sinus tracts; and severe disease (Hurley stage III) by widespread scar and sinus tracts. Medical therapy is often selected based on the Hurley stage of disease, with Hurley stage I and II treated with systemic antibiotics, anti-inflammatory drugs, hormonal therapies (oral contraceptives and spironolactone), and/or a possible short course of oral steroids. Hurley stage III disease typically requires advancement to higher dose systemic glucocorticoids, cyclosporine, and biological agents such as TNF-α inhibitors. Wide excision is reserved for Hurley stage III, although it is used occasionally for milder disease in patients that cannot tolerate medical therapies. Alternative classification systems count the number of nodules and abscesses, which also aids in the diagnosis and direction of therapy.[3]

PREOPERATIVE TREATMENT

First-line treatments for Hurley stage I and II disease include medical management with agents such as systemic antibiotics, hormonal therapies, and oral corticosteroids. Hurley stage II and III disease may require additional biological and medical therapies for effective treatment before surgical intervention.

Antibiotics

Although hidradenitis suppurativa lesions are typically caused by follicular occlusion and

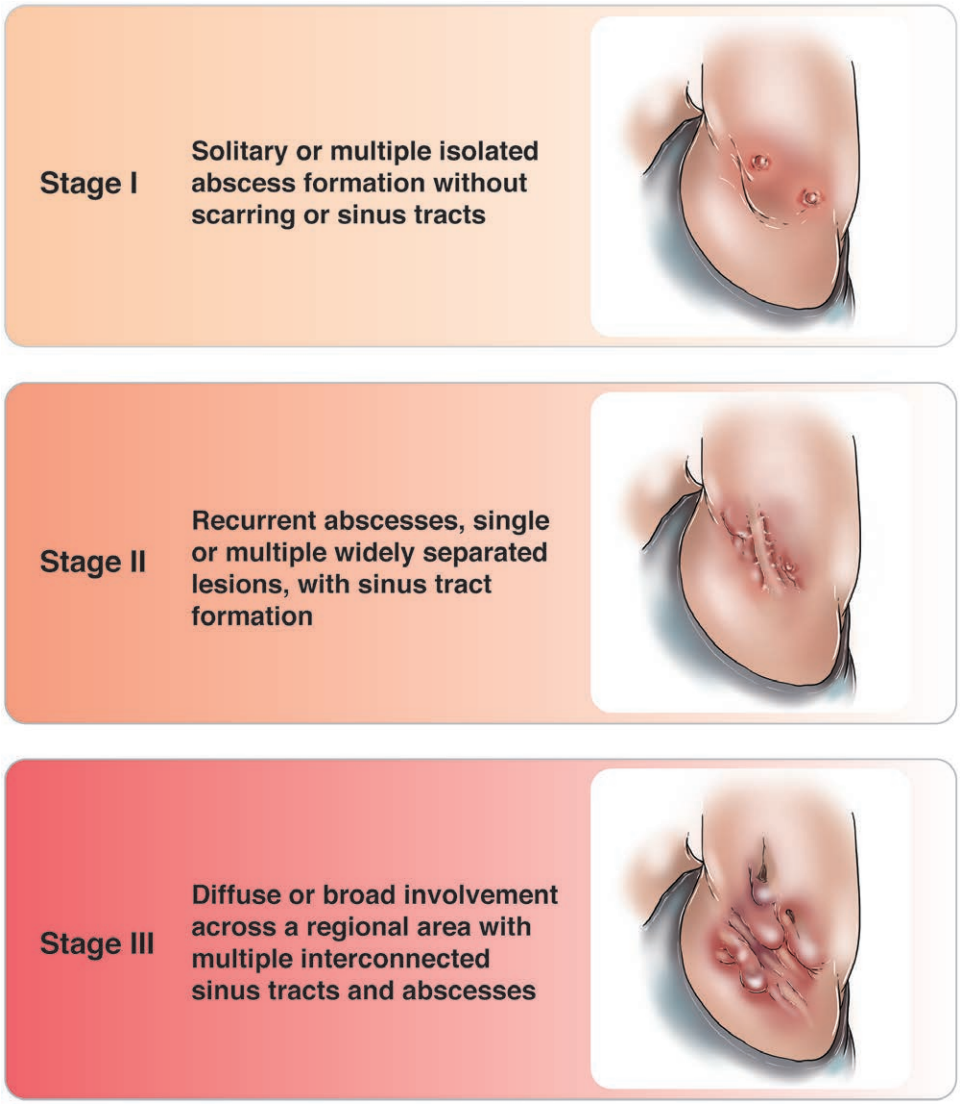

Fig. 1. Hurley classification system for hidradenitis suppurativa.

Table 1. Hurley Stage with Medical and Surgical Management

Hurley Stage	Medical Therapy	Surgical Treatment
I	Antibiotics, antiinflammatory biologics	With or without punch débridement, unroofing
II	Antibiotics, antiinflammatory biologics, steroid	With or without punch débridement, unroofing
III	Systemic glucocorticoids, cyclosporine, antiinflammatory biologics	With or without punch débridement, unroofing; subsequent wide excision and reconstruction

not infection, reducing the microbiotic load and taking advantage of the antiinflammatory properties of antibiotics can be helpful. Infections that secondarily develop in hidradenitis suppurativa lesions benefit from antibiotic therapy targeted based on culture data. Antibiotic regimens include administration of tetracycline, clindamycin, or rifampin and can be subsequently adjusted based on culture data.[17] In severe stage II or III disease, intravenous ertapenem followed by a combination of rifampicin, moxifloxacin, and metronidazole may be used.[1,5]

Hormonal Therapy

Hormonal treatments include oral contraceptives and spironolactone, which can be particularly helpful in Hurley stage I disease. Patients can be treated with multiple modalities simultaneously. Chronic administration is often required, and it may take months for peak treatment effects to be achieved.

Steroids

Local flares can be managed with intralesional steroid injections; oral steroids with doses of 40 to 60 mg/day for 2 to 3 days followed by a 7- to 10-day steroid taper. In addition, antibiotics can be helpful for more extensive exacerbations.

Biologics and Additional Therapies for Severe Disease

Many treatments have been tried in moderate to severe disease: modalities that have been reported to have some ameliorative effects include dapsone, apremilast, metformin, methotrexate, tofacitinib, acitretin, and cyclosporine. Adalimumab, a human immunoglobulin G1 monoclonal antibody that inhibits TNF-α from binding to its membrane-bound and soluble receptors, is the only treatment approved by the U.S. Food and Drug Administration for the treatment of moderate to severe hidradenitis suppurativa. Dose requirements are on the higher end of the adalimumab administration spectrum: 40 mg subcutaneously weekly.[18] Infliximab, a chimeric monoclonal antibody that inhibits TNF-α by binding both transmembrane and soluble TNF-α, allows for weight-based dosing,[19] and high levels (up to 10 mg/kg) are sometimes required. Other biologics are emerging as antiinflammatory treatment for severe or refractory hidradenitis suppurativa disease, and many phase II and III clinical trials are underway, including approaches with IL-17 and IL-23 inhibitors. Retinoids, including isotretinoin, acitretin, and alitretinoin, are considered second- or third-line treatments in severe disease or in patients with concomitant acne but should be avoided in pregnancy.[17]

Multimodal Therapy

Moderate to severe disease requires multimodal medical and surgical treatment, necessitating close collaboration with interdisciplinary team members. Multidisciplinary teams, including dermatologists with expertise in hidradenitis suppurativa treatment, in addition to specialists in plastic surgery, colorectal surgery, urology, and gynecology, allow for development of a comprehensive treatment plan. Refractory cases benefit from medical optimization of the disease before surgical management to decrease inflammation of hidradenitis suppurativa lesions. Although medical therapy alone has shown benefit, cessation of therapy often leads to recurrence.[20] Although many regimens exist, there is no single optimal medical treatment regimen in the literature, leading many physicians to choose treatment regimens based on experience. Plastic surgeons should collaborate with dermatologists specializing in hidradenitis suppurativa to guide medical management and follow patients long term.

SURGICAL MANAGEMENT

There is a wide repertoire of surgical methods used to treat hidradenitis suppurativa patients that can be tailored to parameters such as disease stage, size of the defect, degree of bacterial contamination, and anatomical area, in addition to consideration of scar location and need for revision (Figs. 2 and 3).

Surgical Unroofing

In Hurley stage I disease, acutely inflamed nodules may benefit from punch débridement with a 5- to 7-mm punch biopsy instrument followed by débridement and closure by secondary intention. Chronically inflamed disease with underlying sinus tracts can be initially managed with surgical unroofing, which involves removal of the superficial portion of the lesion. Importantly, incision and drainage, which may temporarily provide relief for fluid-filled inflamed areas, is ineffective as a definitive treatment of hidradenitis suppurativa, with high rates of recurrence of up to 100 percent.[21,22] Hurley stage III disease interconnecting sinus tracts are amenable to unroofing[23]; however, deep interconnecting sinus tracts are more challenging to unroof than shallow, more limited sinus tracts, thus requiring wide excision. Ablative lasers may also be used to unroof nodules and sinus tracts in inflamed disease, with subsequent healing by secondary intention. Carbon dioxide laser also opens hidradenitis suppurativa nodules and sinus tracts, resulting in flat scars in 4 to 8 weeks with a low recurrence rate.[24,25]

Wide Excision of Chronic Disease

Ideally, wide excision is approached after acute inflammation is mitigated with multimodal management. Large amounts of local anesthesia with epinephrine with the tumescent technique can assist with hemostasis and visualization, facilitating precise excision with the knife. The sinus tracts should be totally excised down into normal tissue, removing folliculopilosebaceous units while sparing normal subcutaneous areas. Normal fat tends to be more yellow than diseased fat and is also softer and less stiff than diseased tissue that is often associated with substantial scar. Care should be taken to avoid deep critical structures.

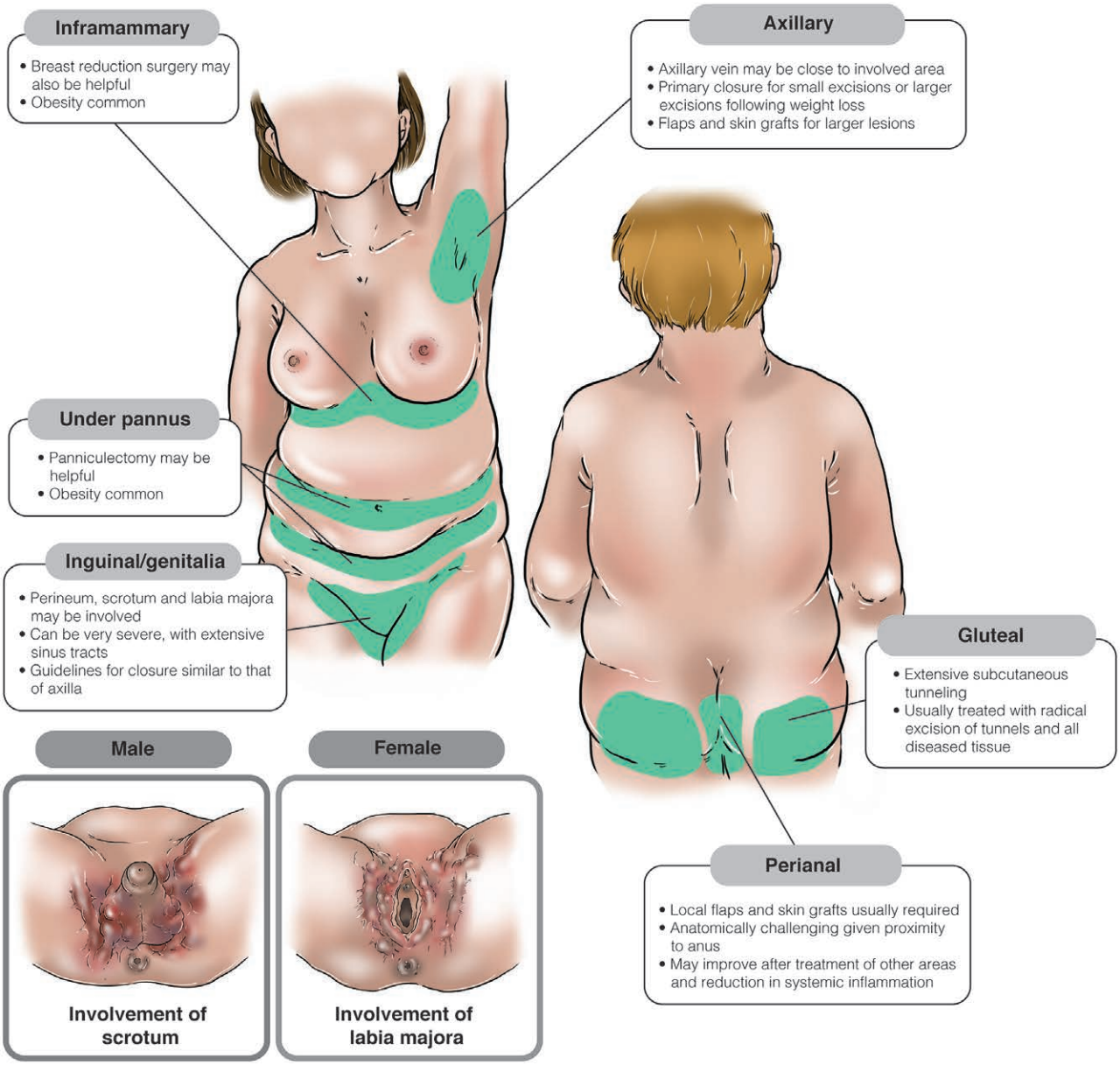

Fig. 2. Anatomical areas commonly involved in hidradenitis suppurativa, including the axillary, inframammary, inguinal/genital, perianal, and gluteal regions, and under the pannus.

For example, in the axilla, the axillary vein is often at the base of the lesions, and careful dissection is required to eradicate diseased areas and leave adequate soft-tissue coverage over the vein. Excision usually is superficial to the fascia but on occasion goes deeper and requires a more extensive dissection.

Normal skin and fat should be preserved to minimize the scarring and simplify the reconstruction. This can be facilitated by performing zigzag incisions around the lesion, which not only preserves normal skin between zigzags that an ellipsoid incision would traditionally discard (thus minimizing the size of the reconstruction) but also facilitates a W-plasty closure, which allows the eventual scar to lengthen and maximizes the surface area for the wound to heal. [**See Video 1 (online)**, which displays staged management of suprapubic hidradenitis using an internal vacuum-assisted closure device and split-thickness skin grafting in a 57-year-old man with a large area of hidradenitis suppurativa of the suprapubic region (stage I). Wide local excision was performed with W-plasty incision. Partial closure with an internal vacuum-assisted closure device was used with a plan to return to the operating room 2 days later.

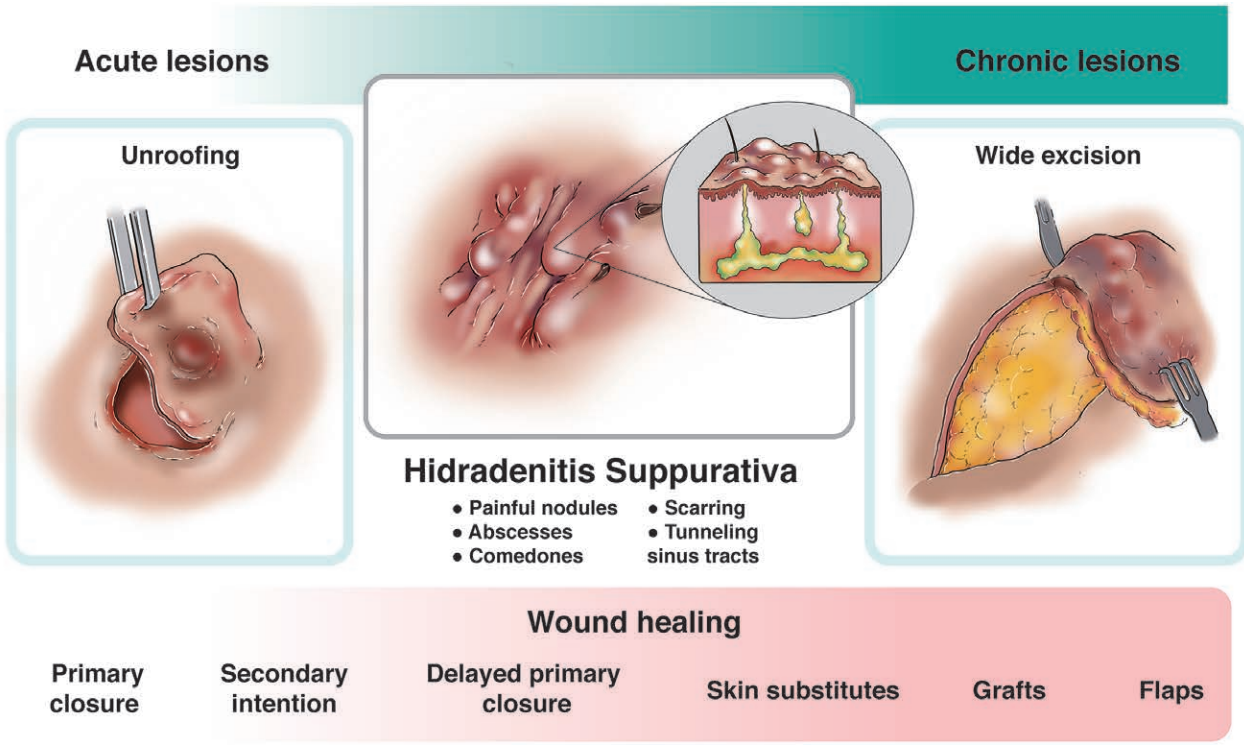

Fig. 3. Surgical management of hidradenitis suppurativa. Acute disease often requires unroofing, whereas chronic disease is amenable to wide excision of hidradenitis suppurativa–affected areas. Reconstruction after excision depends on the size of the defect, anatomical location, severity of disease, and available local options for reconstruction.

See Video 2 (online), which displays staged management of suprapubic hidradenitis using internal vacuum-assisted closure therapy and split-thickness skin grafting. In stage II, 2 days after stage I (**Video 1**), the internal vacuum-assisted closure device was removed and residual disease sharply excised. Split-thickness skin graft was applied to the wound bed and a negative-pressure wound therapy device applied over skin graft.] Tissue excised is routinely sent for pathologic evaluation, as human papillomavirus and squamous cell cancer can be present in anal hidradenitis suppurativa wounds.[26]

Given that hidradenitis suppurativa–affected areas are highly contaminated and often closed under substantial tension, surgeons have historically been reluctant to perform wide excision procedures for fear of high complication rates. Managing expectations is key in these scenarios: patients should be prepared for the possibility of a postoperative wound infection or wound dehiscence and should be counseled that these procedures can take several weeks and sometimes months to heal. Effective patient communication and education are critical to ensure that patients have an accurate understanding of what the postoperative course will be.

Reconstruction

Stage of disease, size of defect, degree of bacterial contamination, and anatomical location will affect reconstructive technique. Reconstructive options include healing by secondary intention, primary closure, local flaps, regional flaps, staged closure with negative-pressure wound therapy, conventional and recycled skin graft, and dermal substitute with subsequent skin graft (Figs. 4 through 6). Regardless of the modality, the overarching goals of the reconstruction are to (1) eliminate the vast majority of the disease in the affected area, (2) provide stable coverage, (3) minimize infection risks, (4) minimize risks of dehiscence, and (5) reduce the risk of scar contractures.

Although healing by secondary intention is appropriate in acutely inflamed or infected lesions, larger wounds can require months for healing,[27] lead to contracture, or cause deformity. Compared to conventional moist saline dressings with frequent changes, alternative dressings such as alginates, hydrogels, and negative-pressure wound therapy devices are attractive options that achieve less painful and faster healing and minimize dressing changes.[28] However, these devices come at an increased cost and complexity, so normal saline dressings may be a reasonable alternative in many patients.

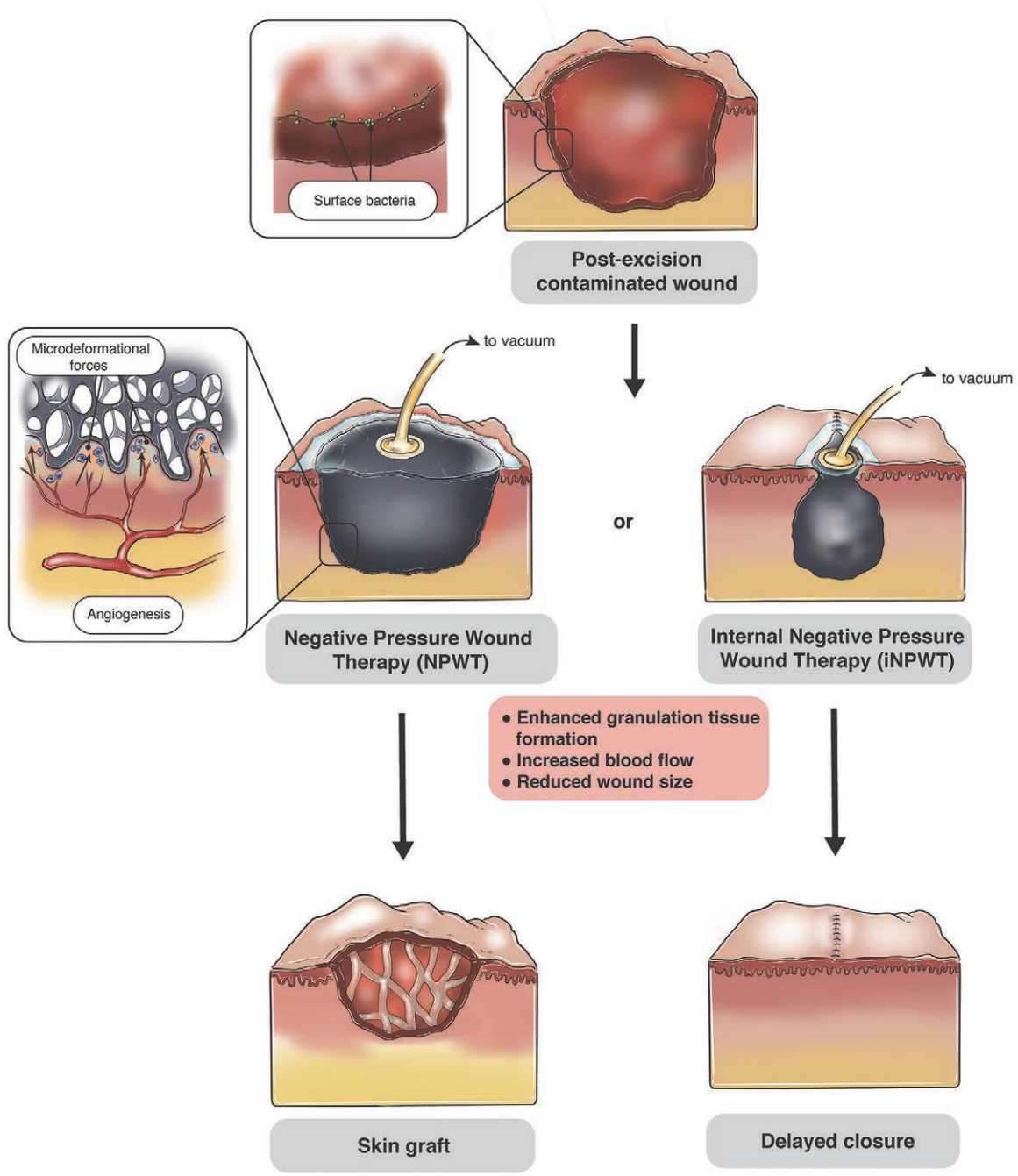

Fig. 4. Negative-pressure wound therapy can be beneficial in contaminated or large wounds following excision. Negative-pressure wound therapy enhances granulation tissue, increases blood flow, and reduces wound size. Internal vacuum-assisted closure allows for staged closure of a wound, with the remainder of the wound closed primarily after a few days of internal vacuum-assisted closure therapy.

Primary closure allows the elimination of painful dressing changes; thus, it is a preferred option in cases of small lesions or less severe disease with low bacterial load (Fig. 7). If wound dehiscence does occur, dressing changes can be implemented and the wound allowed to heal by secondary healing while allowing continued drainage. Resuturing wounds is usually not successful.

Negative-pressure wound therapy has emerged as a useful adjunct to reduce the risks of infection and dehiscence and serves as a bridge to definitive closure in staged reconstruction (Fig. 4). Following wide excision, the wound can be largely

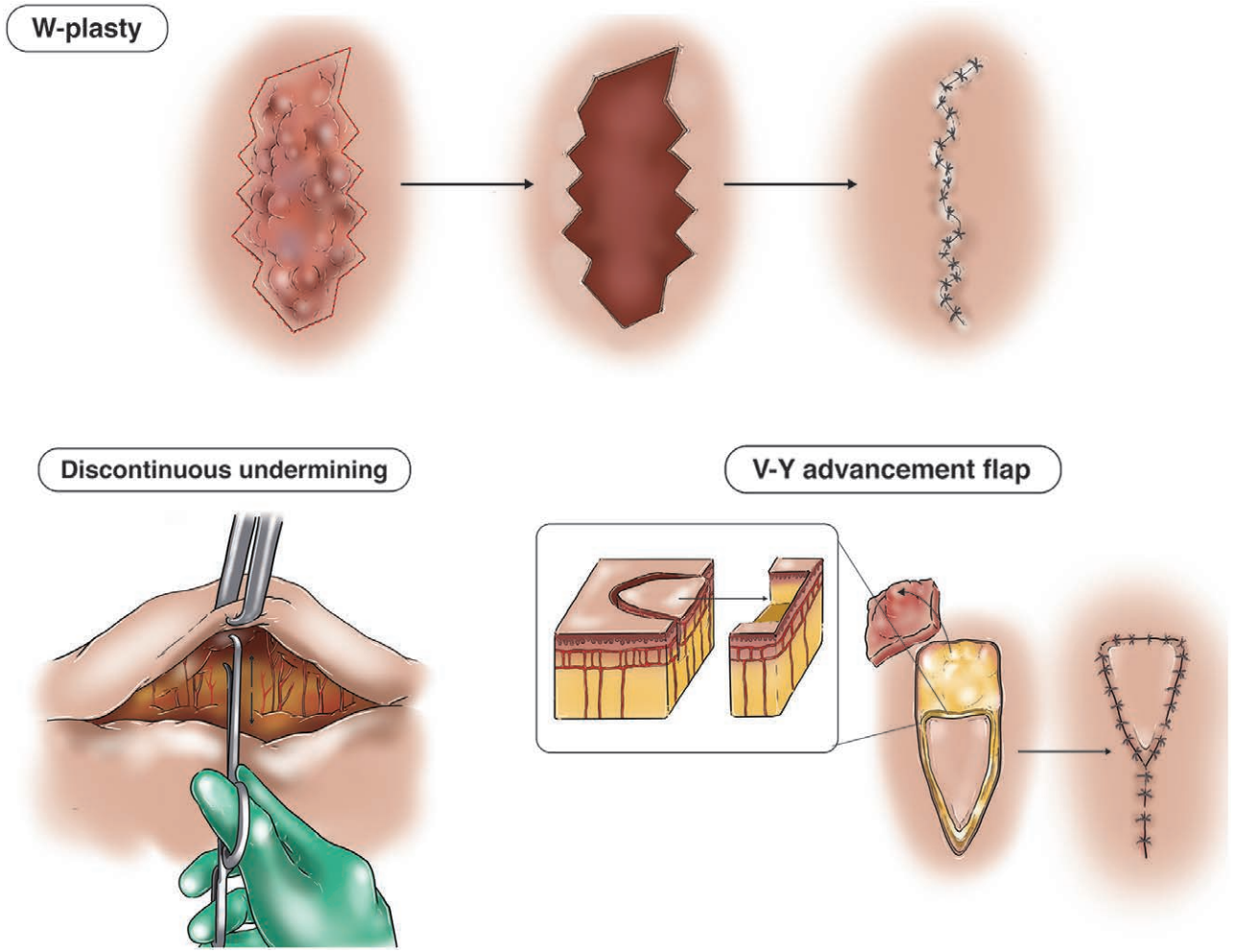

Fig. 5. Closure methods including W-plasty closure after zigzag incisions around lesions; discontinuous undermining; and V-Y advancement flap.

closed over a negative-pressure wound therapy device with the sponge surface contacting with the wound edges under the closed wound. Referred to as an internal vacuum-assisted closure device, this method effectively evacuates fluid produced by the wound through a sponge that exits through a small opening in the wound. The sponge is left in place for 2 to 4 days before removal and inspection of the wound. Wounds that appear clean may be closed at that time, whereas those suspicious for infection require additional débridement followed by negative-pressure wound therapy until final closure.[28] [**See Video 3 (online)**, which displays staged management of gluteal hidradenitis and pilonidal disease using internal vacuum-assisted closure therapy in a 19-year-old man with a history of extensive hidradenitis and pilonidal disease. The video demonstrates the second stage of surgical treatment, 2 days after wide local excision of the gluteal region and placement of an internal vacuum-assisted closure device. The internal vacuum-assisted closure device was removed, the wound was irrigated, layered closure was performed, and an incisional negative-pressure wound therapy device was applied.]

A retrospective review of 60 cases of hidradenitis suppurativa in 27 patients at a single institution, including 30 cases with primary closure and 30 cases with delayed primary closure, found that patients managed with delayed primary closure with internal vacuum-assisted closure had wounds that were on average four-fold larger.[28] Healing times between the two groups were similar at 2.2 months and 2.7 months for delayed primary with internal vacuum-assisted closure and immediate primary closure, respectively. No patients with internal vacuum-assisted closure had recurrence of disease (average follow-up, 2.3 months). The authors recommend a treatment algorithm based

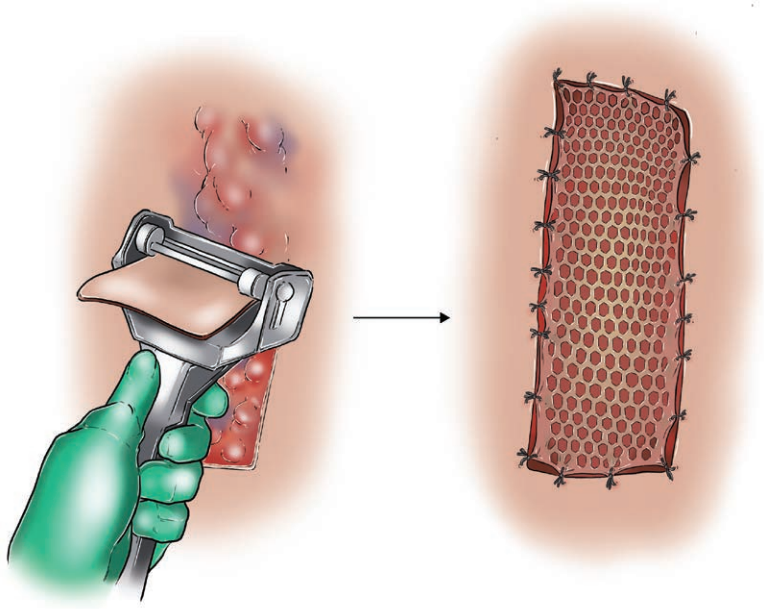

Fig. 6. Reused skin graft technique in which partial-thickness skin graft is taken over hidradenitis suppurativa–affected areas, preserved, and then reapplied to the wound bed after wide excision of hidradenitis suppurativa–affected tissue. The folliculopilosebaceous units affected in hidradenitis suppurativa are not included in the partial-thickness skin graft, thus allowing the graft to be reapplied to the wound bed.

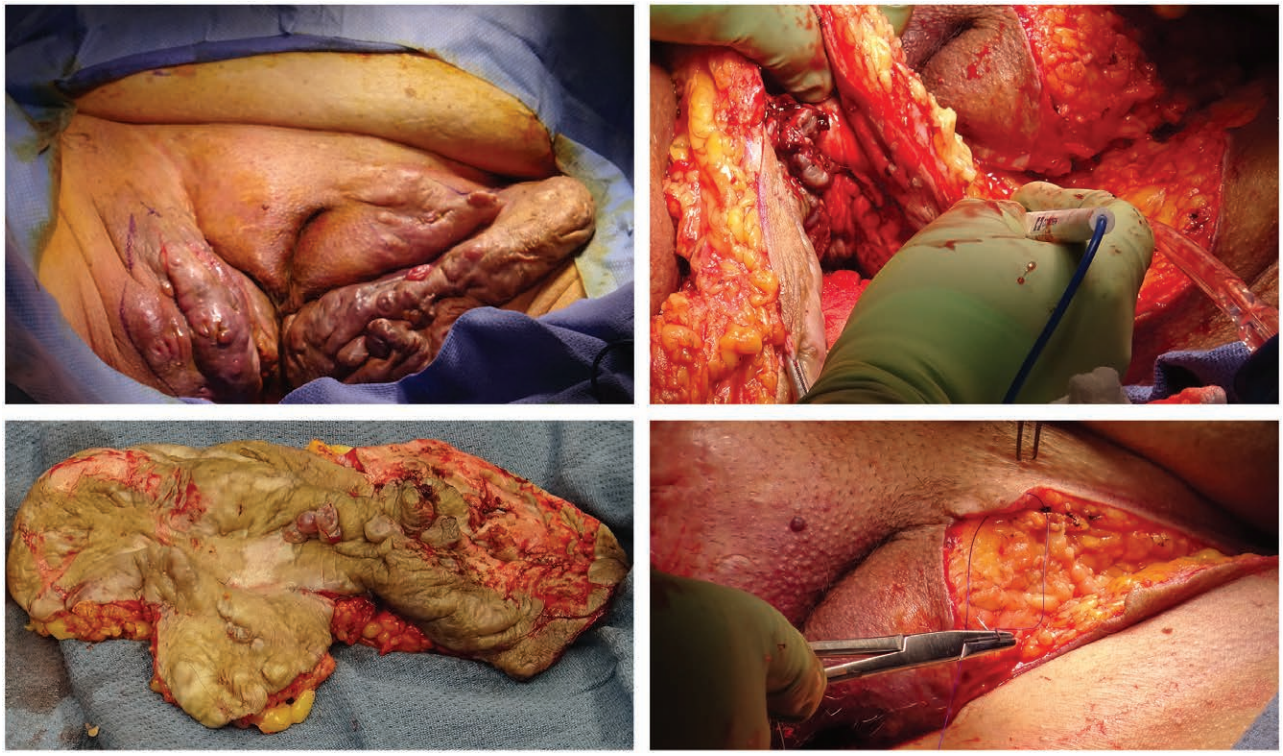

Fig. 7. Advanced case of hidradenitis suppurativa in a female patient, with severe involvement of the groin, perineal region, and labia majora (*above*, *left*). Surgical management included wide local excision (*above*, *right*, and *below*, *left*) and primary closure (*below*, *right*).

on wound assessment at the time of wide local excision: if a wound is small (i.e., <70 cm^2) with a clean base, immediate primary closure is performed. If a wound is large (i.e., >70 cm^2) or with persistent infection, internal vacuum-assisted closure is performed for 2 to 4 days with subsequent delayed primary closure.

Interestingly, the superficial surface of the skin in areas affected by hidradenitis suppurativa may be uninvolved in the disease process and can be used as a source for a skin graft, which is known as the reused skin graft technique.[29,30] Skin grafts can be taken 0.12 to 0.020 inch with a dermatome or knife and meshed for increased surface area coverage. The skin graft is superficial to the follicular pilosebaceous units, which are excised after the skin graft is taken (Fig. 6). A retrospective review of 18 male patients who underwent the reused skin-graft technique for gluteal hidradenitis suppurativa with mean follow-up of 61 months had no patients with recurrence at the surgical site.[31] Split-thickness skin graft from an alternate donor site is often performed; however, the reused skin graft technique should be considered, as it eliminates donor-site morbidity. Although the use of dermal scaffolds with subsequent skin grafting presents a potential method for improved cosmesis compared to skin graft alone, the benefits of such combinatorial methods must be evaluated against the disadvantages of increased cost and prolonged hospitalization.[32]

Local flaps allow for movement of well-vascularized tissue into the wound bed, good cosmesis, and shorter time to wound closure. Discontinuous undermining, or subcutaneous blunt dissection in the vertical direction with careful preservation of skin perforators, increases the mobility of surrounding tissue for local tissue transfer without creating a large continuous dead space. V-Y advancement flaps allow for movement of larger local flaps into the wound bed (Fig. 5). A variety of perforator flaps may be designed for advancement, rotation, or transposition into the defect. Superior and inferior gluteal artery perforator flaps provide coverage of larger wounds in gluteal, perianal, and perineal hidradenitis suppurativa lesions,[33] whereas thoracodorsal artery perforator flaps provide well-vascularized soft tissue for axillary hidradenitis suppurativa lesions.[34]

Anatomical Considerations

In axillary hidradenitis, primary closure is a viable option for the reconstruction of both smaller wounds and larger wounds in patients following weight loss, where excess local tissue can be used for wound closure.[28] If lesions are too large for staged primary closure, local perforator flaps may be considered, which provide well-vascularized tissue but do introduce bulk. If bulk is of concern, skin grafts provide excellent coverage of large wounds. Skin grafts are usually meshed to allow for drainage and draping over irregular surfaces and are commonly covered with a negative-pressure wound therapy device that is left in place for 5 to 7 days.[35]

Excision of lesions in the inguinal and perineal region requires careful dissection to preserve normal skin and structures. Reconstruction will depend on the availability of local tissue, with preference for primary closure of small lesions but effective alternatives including local soft-tissue movement with discontinuous undermining or tissue advancement, delayed primary closure with internal vacuum-assisted closure, or skin graft with negative-pressure wound therapy. [**See Video 4 (online)**, which shows staged management of penile, perineal, and scrotal hidradenitis with skin grafting and negative-pressure wound therapy in a 54-year-old man with a history of severe scrotal, penile, and perineal hidradenitis after limited scrotal resection. The patient presented with residual disease and lymphedema of the scrotum and penis. Wide local excision of hidradenitis of scrotum and penis and reconstruction with local tissue rearrangement, split-thickness skin grafting to the penis, and placement of irrigation negative-pressure wound therapy were performed. A urinary catheter was used given the location of the penile skin graft recipient site, which was treated with negative-pressure wound therapy. During the second stage, the negative-pressure wound therapy device was removed to reveal skin graft take to the penile shaft. Additional disease of the perineum and right groin were identified and excised. Layered closure was performed over a round closed-suction drain.]

In cases of scrotal hidradenitis, reused skin grafts harvested from the scrotum with meshing may eliminate the need for a secondary skin graft donor site. Perianal lesions are an anatomically challenging area for both excision and reconstruction. After ruling out any perianal fistulas, local flaps are typically used for reconstruction.

Gluteal hidradenitis often presents with deep interconnecting tracts that tunnel throughout the gluteal soft tissue, so wide excision and thorough elimination of these tracts are critical to prevent recurrence. Reconstructive options for large wounds include recycled skin graft[31]; delayed primary closure with internal vacuum-assisted

closure[28]; and local fasciocutaneous flaps, including posterior thigh V-Y advancement flaps, rotation flaps, or bilobed flaps.[26,36]

Breast hidradenitis that develops along the inframammary fold can be treated with wide local excision in conjunction with breast reduction. Lower abdominal hidradenitis often presents under the fold of the pannus, which can be treated with wide local excision and panniculectomy.

POSTOPERATIVE CARE

Multimodal therapy should continue postoperatively with adjunctive medical therapy. For advanced biologics, there is no clear consensus regarding withholding the biologic perioperatively. The standard practice at our institution is to withhold biologics for 1 week before surgery and for 2 to 3 weeks following surgery, but this may not be necessary. For more extensive excisions, intravenous antibiotics may be given postoperatively with subsequent transition to oral antibiotic therapy as guided by intraoperative culture data.

Postoperative wound care has been drastically changed with the advent of negative-pressure wound therapy. Hygiene should be maintained to protect the negative-pressure wound therapy device adhesive to ensure the sponge holds suction. In cases of perineal or gluteal hidradenitis suppurativa, an indwelling urinary catheter should be considered to protect the negative-pressure wound therapy seal. Bed rest is implemented only if there is concern for sheer forces on a skin graft with ambulation.

COMPLICATIONS

Complications of surgical treatment of hidradenitis suppurativa include infection, bleeding, suture dehiscence, nonhealing wound, scarring, contractures, and recurrence of diseased tissue.[7,37] Wound healing problems can be managed with local wound care or vacuum-assisted closure. Beyond factors such as malnourishment and vitamin deficiencies, many patients are obese and some may resume smoking, all of which impede effective wound healing. If wound healing difficulties are suspected to be because of the presence of high bacterial load or recurrent disease, intravenous antibiotics should supplement wound care.

Reported rates of recurrence are highly variable, with some reports of hidradenitis suppurativa patients having a recurrence rate of 100 percent when treated with incision and drainage alone.[21] Another systematic review found a lower recurrence rate of 22 percent for incision and drainage, compared to 27 percent for unroofing and 13 percent for wide excision.[38] The same review found that recurrence rate differed based on type of closure, with 15 percent for primary closure, 8 percent for flap closure, and 6 percent for skin graft closure. The anatomical location with the greatest risk of recurrence is the genital region [17 of 31 (55 percent)], followed by inguinal [37 of 94 (39 percent)], axillary [20 of 56 (36 percent)], and gluteal [17 of 65 (26 percent)] regions, with an overall average recurrence rate of 38 percent in cases managed with wide excision with secondary healing.[39] Although not without the risk of recurrence and other complications, it is our experience that wide surgical excision with reconstruction leaves most patients healed, with substantial reduction in their disease burden.

QUALITY OF LIFE

Hidradenitis suppurativa causes significant psychosocial and physical impairment in patients,[40] contributing to devastating effects on quality of life in personal and professional activities.[41] Reduced quality of life, as measured by the Dermatology Life Quality Index, was found to be strongly correlated to hidradenitis suppurativa clinical severity.[42,43] One retrospective review that examined patients with Hurley stage III disease undergoing wide excision with multimodal therapy found that nearly all patients reported restrictions on everyday life because of hidradenitis suppurativa [243 of 255 (95 percent)], 197 of 255 (77 percent) of whom classified their limitations as “very strong” or “strong.”[44] Ninety-seven percent of patients in this study reported impairment of everyday life and 60 percent of patients reported impairment in professional life. Eighty percent of patients reported that they were very satisfied (118 of 251) or satisfied (82 of 251) following surgical treatment, with two-thirds [169 of 249 (68 percent)] reporting satisfaction with their aesthetic result and 20 percent (49 of 249) classifying their aesthetic result as “very good.” Two hundred ten of 246 patients (85 percent) noted they would recommend surgical treatment to other patients similarly affected by hidradenitis suppurativa.

CONCLUSIONS

Hidradenitis suppurativa requires a multidisciplinary team approach and multimodal therapy for successful treatment. Surgical excision and reconstruction approaches depend on

hidradenitis suppurativa severity, size, degree of contamination, and anatomical location. Patients can greatly benefit from surgical treatment for this debilitating disease.

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