Title
An atlas of the morphological manifestations of hidradenitis suppurativa

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An atlas of the morphological manifestations of hidradenitis suppurativa

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Abstract

This article is dermatological atlas of the morphologic presentations of Hidradenitis Suppurativa (HS). It includes: superficial abscesses (boils, furuncles, carbuncles), abscesses that are subcutaneous and suprafascial, pyogenic granulomas, cysts, painful erythematous papules and plaques, folliculitis, open ulcerations, chronic sinuses, fistulas, sinus tracts, scrotal and genital lymphedema, dermal contractures, keloids (some that are still pitted with follicular ostia), scarring, skin tags, fibrosis, anal fissures, fistulas (i.e. circinate, linear, arcuate), scarring folliculitis of the buttocks (from mild to cigarette-like scarring), condyloma like lesions in intertrigous areas, fishmouth scars, acne inversa, honey-comb scarring, cribiform scarring, tombstone comedones, and morphia-like plaques. HS can co-exist with other follicular diseases such as pilonidal cysts, dissecting cellulitis, acne conglobata, pyoderma gangrenosum, and acanthosis nigricans. In sum, the variety of presentations of HS as shown by these images supports the supposition that HS is a reaction pattern.

HS is a follicular based diseased and its manifestations involve a multitude of follicular pathologies [1,2]. It is also known as acne inversa (AI) because of one manifestation that involves the formation of open comedones on areas besides the face. It is as yet unclear why HS is so protean in its manifestations. HS severity is assessed using the Hurley Staging System (Table 1). It also remains unclear why hidradenitis may remain limited to Hurley Stage 1, evolve to the more confluent (Hurley Stage 2), or progress even further to the fully confluent (Hurley Stage 3).

In addition, HS can be associated with other follicular based diseases such as pilonidal cysts (PCs) of the sacrum and buttocks, dissecting cellulitis (DC), and acne conglobata (AC), which usually involves the face, chest, When HS occurs with PCs, DC, and/or AC it is referred to as the follicular occlusion triad or tetrad [2]. HS can more rarely be associated with pyoderma gangrenosum (PG) or Crohn disease (CD), other inflammatory diseases of the skin that are not follicular. The reason for this is unclear [2]. What AC, DC, HS, CD, and PG share is that they occur in bacterially rich environments. HS probably occurs with acanthosis nigricans because many HS patients are obese [2]. This concurrence seems under reported.

Table 1 -- Hurley staging system for hidradenitis suppurativa

<table>
<thead>
<tr>
<th>Hurley stage</th>
<th>Extent of disease in tissue</th>
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<tbody>
<tr>
<td>1</td>
<td>Abscess formation (single or multiple) without sinus tracts and cicatization</td>
</tr>
<tr>
<td>2</td>
<td>One or more widely separated recurrent abscesses with tract formation and scars</td>
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<tr>
<td>3</td>
<td>Multiple interconnected tracts and abscesses throughout an entire area</td>
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HS is an inflammatory and scarring dermatosis. Recent efforts try and establish subtypes but a full understanding of the basis of HS, its subtypes, and clinical manifestations has yet to be established [3, 4]. In some cohorts genetic defects have been associated in up to 1/3 of cases with stage 2 and stage 3 HS [3]. These genetic defects are associated with more severe HS than sporadic HS, but generally do not define the clinical findings of HS [5]. The rate of genetic linkage to Stage 1 HS and phenotypic variation of Stage 1 HS remains undefined. Early skin pathology of HS arises in the follicle in all types [6, 7]. Table 2 summarizes the clinical manifestation of HS and associated pathologic states.

### Table 2 summarizes the clinical manifestation of HS and Associated Pathologic States

<table>
<thead>
<tr>
<th>Clinical Manifestations of HS</th>
<th>Associated Pathologic States and HS</th>
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<tbody>
<tr>
<td>Abscesses (boils, furuncles, carbuncles)</td>
<td>Acanthosis Nigricans</td>
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<tr>
<td>Acne vulgaris</td>
<td>Acne Conglobata</td>
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<tr>
<td>Acne inversa</td>
<td>Dissecting Cellulitis</td>
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<tr>
<td>Anal Fissures</td>
<td>Pilonidal cysts</td>
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<tr>
<td>Bilateral breast cellulitis</td>
<td>Pyoderma gangrenosum</td>
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<tr>
<td>Cellulitis</td>
<td>Crohn's Disease</td>
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<tr>
<td>Circinate scars (usually overlying fistulas)</td>
<td>Mammillary Fistula</td>
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<tr>
<td>Cigarette scarring of buttocks</td>
<td>Abscesses</td>
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<tr>
<td>Chronic sinuses</td>
<td>Components of the tetrad are in bold type</td>
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<td>Condyloma like lesion in intertrigous areas</td>
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<td>Cribriform scarring</td>
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<td>Cysts [36]</td>
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<td>Dermal contractures</td>
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<td>Divots</td>
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<td>Elephantiasis of scrotum</td>
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<td>Fibrosis</td>
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<td>Fistulas</td>
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<td>Fishmouth scars</td>
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<td>Folliculitis</td>
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<td>Friable tissue</td>
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<td>Honey Comb scarring</td>
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<td>Keloids</td>
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<td>Lymphangiectasias (genitals)</td>
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<td>Lymphedema (genitals)</td>
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<td>Morphea like with follicular ostia</td>
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<tr>
<td>Painful papules and plaques</td>
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<td>Pits</td>
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<td>Polypoid HS</td>
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<td>Proud Fresh</td>
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<td>Pyogenic granulomas [36]</td>
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<td>Open ulcerations</td>
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<td>Oval scars</td>
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<tr>
<td>Scarring</td>
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<td>Scarring folliculitis of the buttocks</td>
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<td>Skin tags</td>
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<td>Sinus Tracts</td>
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<tr>
<td>String or rope like contractures</td>
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<tr>
<td>Suprafascial and subcutaneous abscesses</td>
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<tr>
<td>Tombstone comedones</td>
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<td>Verrucous lymphostasis [22]</td>
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<td>Vulvar edema</td>
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The most common lesion of HS is the recurrent or chronic non-infectious abscess (boil, furuncles, carbuncles). The HS abscess initially appears as the inflammation of one follicle and spreads to adjacent follicles (Figure 1 and 2). This is facilitated by the
hyperkeratosis present in the upper 1/3 of the follicles affected by HS. With the follicle closed the contents of follicle cannot escape normally. Instead, abetted by inflammatory mediators, the follicles are joined subcutaneously. In other words, small tunnels are formed between the follicles; if these connections are extensive and large we call them fistulas.

One clue that an abscess is a non-infectious abscess of HS rather than an infection of *Staphylococcus Aureus* is the persistence of the abscess in the face of antibiotic treatment or drainage and the scarring and recurrent nature of the abscesses. Although antibiotics cure *Staphylococcus Aureus*, the combination of antibiotic such as clindamycin and rifampin only ameliorate the abscesses and disease process of HS. The course of HS parallels that of inflammatory diseases that degrade tissue, such as inflammatory arthritis (e.g. rheumatoid or psoriatic), rather than cutaneous psoriasis, which can be cleared with adequate therapy without tissue destruction. Kin to HS are discoid lupus and severe acne vugaris, which are other diseases that leave behind permanent scars even after the inflammation has passed. In rarer cases, HS abscesses of the buttocks can penetrate to the subcutaneous tissue and muscle (Figure 3). Sometimes the abscesses can become confluent, involve whole areas of groin, and evolve into keloids mixed with abscesses (Figure 4, 5). Figure 6 shows a persistent abscess on the abdomen of a patient and figure 7 shows a persistent abscess on the groin and legs of an HS patient. Often the abscesses of HS can have a keloidal appearance (Figure 8). Sometimes the abscess can totally efface the tissue of the axilla (Figure 9). Subcutaneous suprafasical abscesses of HS (Figure 10) can develop in particular on the flank; fluid can be aspirated from them with a wide bore needle. They may develop form inflammation and abscess in the axilla that somehow migrates downward.

![Figure 1. HS abscess of the buttocks with pointing of pustule ©Noah Scheinfeld](image-url)
Figure 2. HS abscess of the axilla ©Noah Scheinfeld

Figure 3. Abscess of buttocks with penetration to the subcutaneous tissue ©Noah Scheinfeld

Figure 4. Hurley Stage 2 abscesses in the groin of an HS patient ©Noah Scheinfeld
Figure 5. Fully Confluent Abscess HS Hurley Stage 3 in groin ©Noah Scheinfeld

Figure 6. Persistent abscesses that leak pus on an HS patient's abdomen ©Noah Scheinfeld

Figure 9. Confluent axillary HS abscesses with copious purulent discharge ©Noah Scheinfeld
Keloidal abscess

HS also resembles the scarring response of inflammation that leads to the formation of keloids. This can occur on any part of the body including the abdomen (Figure 10). Keloid can co-exist with abscesses (Figure 11) of the buttocks, axilla, and arm (Figure 12). The keloids can be similar to typical keloids or have a quality similar to cellulitis with erythema surrounding the keloidal abscesses (Figure 13). Abdominal keloidal abscess of leg and groin in different stages can be present (Figure 14). Keloids of the abdomen can have a static quality (Figure 15). They might develop of areas of oval erythema that are characteristic for HS (Figure 16) or form abscess similar to what HS induces in the axilla (Figure 2).
Figure 12. Persistent abscesses of the axilla evolving into linear lesion © Noah Scheinfeld

Figure 13. Abdominal keloidal abscess with erythema (cellulitis) surrounding the abscess © Noah Scheinfeld
Figure 14. Keloidal abscess of leg and groin in different stages ©Noah Scheinfeld

Figure 15. Persistent Abdominal keloids evolving with minimal inflammation ©Noah Scheinfeld
Pyogenic Granulomas in HS

Another common lesion of HS, although underreported, is the pyogenic granuloma. Such pyogenic granulomas can be isolated and sometimes confluent (Figure 17, Figure 18) in the axilla, the sacral area (Figure 19), and the groin (Figure 20). In HS pyogenic granulomas are not uncommon on the female breast (Figure 21). Sometimes red friable nodules are associated with HS that are not PG. These can appear as friable tissues (Figure 22) or raw proud flesh (Figure 23) rather than true pyogenic granulomas.
Figure 18. Axillary pyogenic granuloma ©Noah Scheinfeld

Figure 19. Sacral pyogenic granulomas of HS ©Noah Scheinfeld
Figure 20. Pyogenic granulomas of the groin isolated ©Noah Scheinfeld

Figure 21. Isolated pyogenic granuloma on female breast ©Noah Scheinfeld
The female breast in HS

The breast is a less frequent location of HS than the axilla or groin. It can involve the dorsal (Figure 19) and ventral sides of the breasts (Figure 20). The area between the breasts can also manifest with erosions, abscesses, and scarring (Figure 21 and Figure 22). Breast HS is notable in several respects. Fistulas can occur on the breast (mammillary fistula) leading one researcher to note that this finding should be a part of the tetrad. I disagree because this is much more uncommon than other members of the tetrad [8]. Another case noted bilateral HS presenting as bilateral breast cellulitis [9]. It is notable for the fact that surgery short of mastectomy can result in early recurrence [10, 11, 12]; breast HS has a 50% recurrence rate after surgery [13].
Figure 20. Scarring with abscess activity on the ventral part of the breast

Figure 21. Erosion with pyogenic granulomas/abscesses between breasts ©Noah Scheinfeld

Figure 22. Abscess on lower breast and pyogenic granuloma on upper breast ©Noah Scheinfeld
Another pattern of HS involves scarring folliculitis of the buttocks. The scarring folliculitis can be mild, moderate, or severe and can be independent to scarring folliculitis of the back (Figure 23, 24, 25). When it is severe this folliculitis manifests the appearance of cigarette burns (Figure 26).
Folliculitis and HS

HS can also manifest as folliculitis in other areas, particularly in intertrignous areas such as the pannus of the abdomen in obese patients (Figure 27). Such folliculitis is common on the legs of HS patients as well (Figure 28). It can occur on the abdomen even if the patient is not obese and a pannus is not present (Figure 29).
Figure 27. Folliculitis of the pannus of the abdomen in an obese HS patient ©Noah Scheinfeld

Figure 28. Folliculitis and inflammatory nodule on the leg of an HS patient ©Noah Scheinfeld
Acne Conglobata and HS

The inflammatory lesions of HS and AC are sometimes distinct and although the patient might have HS in intertrigous areas they can have AC on the back (Figure 30). Sometimes the morphology of HS and AC overlap making a true taxonomic distinction between them difficult (Figure 31, Figure 32). HS can be accompanied by scarring abscesses and folliculitis on the chest (Figure 33, 34). Often HS of the axilla and groin is accompanied by devastating inflammation of the back that results in scarring acne, nodules, and fibrosis (Figure 35, Figure 36, Figure 37). Some have described this pattern of HS as "worm eaten" [14].
Figure 31. Acne Conglobata of the Abdomen in HS patient ©Noah Scheinfeld

Figure 32. Acne Conglobata of the Abdomen in HS patient ©Noah Scheinfeld

Figure 33. Inflammatory lesions on the chest of an HS patient with axillary and gluteal involvement ©Noah Scheinfeld
Figure 34. Inflammatory lesions on the chest of an HS patient with axillary and gluteal involvement.

Figure 35. Back: Difficult to distinguish HS from AC ©Noah Scheinfeld
Rope-like scars, linear scars, oval scars

HS can produce rope-like scars, linear scars with an oval quality, and oval scars. Figure 36 shows a keloidal rope that follows the lines of tension and extends from posterior ear to the face. Figure 37 shows linear scars appearing in the inguinal folds and figure 38 shows a rope-like scar extending around the base of the right breast. Figures 39, 40, and 41 demonstrate oval scars following lines of skin tension.
Figure 36 Rope-like scar extending from ear to face

Figure 37. The intertrigous inflammation of HS follows the inguinal folds.
Figure 38. Rope-like inflammation following the submamillary fold

Figure 40. Oval and Linear oval scars of HS.
Figure 41. HS linear scars that follow the lines of skin tension

Figure 41. HS following the lines of skin tension; there is some rope-like quality to these scars
Another interesting presentation of HS in intertrigous areas is the appearance of condyloma-like lesions. This can occur in particular where the skin is opposed to other skin in the groin (Figure 42), the axilla (Figure 43), or even behind the ears (Figure 44)[9,10]. It should be recalled that some dermatoses such as amyloidosis, can mimic condyloma when appearing in intertriginous areas [17, 18, 19]. Vulvar lymphangiomata has also be noted to mimic condlyomata [20].

Figure 42. HS presenting as condyloma-like lesions in the groin ©Noah Scheinfeld

Figure 43. HS presenting as condyloma like lesion of the axilla©Noah Scheinfeld
Lesions around the ear emphasize the principle that HS merely needs follicular skin to develop. Auricular skin has other requisite qualities that can make HS develop e.g. bacteria, friction. The posterior ear is an area of occlusion, which can explain why HS manifests with abscesses and tissue distortion (Figure 45, 46, 47, 48). HS does not occur in the ear itself, which is attributable to the ear's paucity of hair follicles. Older people do grow hair out their ears, but this has not been linked to HS.
Figure 46. Preauricular abscess of the ear to medial to the lobule ©Noah Scheinfeld

Figure 47. Figure area behind ear with fibrosis, abscesses in preceding patient ©Noah Scheinfeld
Acne inversa as a presentation of HS

Acne inversa (AI) has been used by some as synonymous with HS. I have argued that it is not, but I think that acne inversa is one of many common presentations of hidradenitis [14]. HS is a follicular disease and one aspect is hyperkeratosis of the follicle. Sometimes the acne inversa is so mild as to be hardly noticed (Figure 49). This can occur on any area of HS, but is particularly seen in the central fold of the axilla (Figure 50, 51, 52), the groin, the upper inner thigh (Figure 53), or in scars of HS (Figure 54). A keratin plug develops, which mirrors the open comedo on the face, the so called "black head." Sometimes acne inversa can coexist with the abscesses of HS (Figure 55). Like the black heads (open comedones) of the face they can be extracted and have a propensity to recur.

Interestingly, when acne inversa occurs there is less of a propensity to form abscesses, pyogenic granulomas, and other inflammatory lesions of HS, but this is not a rigid rule. Acne inversa and HS-type abscesses can co-exist. One finding that helps to make the clinical diagnosis of HS is the presence of tombstone comedones i.e. a scar with black heads on one or both ends (Figure 56).
**Figure 49.** Early acne inversa of the axilla

**Figure 50.** Acne inversa ("black heads") of the axilla as a manifestation of HS

**Figure 51.** Acne inversa ("black heads") of the axilla as a manifestation of HS
Figure 52. Acne inversa of the axilla

Figure 53. Acne inversa ("black heads") of the thigh as a manifestation of HS ©Noah Scheinfeld
Figure 54. Acne inversa of the axilla on a kelodial base ©Noah Scheinfeld

Figure 55. Acne inverse at fold of axilla and abscess at bottom of axilla
Keloids

Keloids are very common in all stages of HS. They evolve from the abscesses and folliculitis of HS. Often abscesses, folliculitis, and keloids co-exist. In cases of stage 3 HS, they can be almost totally confluent in the groin (Figure 57) and the axilla (Figure 58, Figure 59). On rare occasions, HS can exhibit a morphea-like appearance with the difference being that the sclerotic plaques are still pitted with follicular ostia and raised keloidal scars (Figure 60). After multiple surgeries to remove HS and multiple recurrences, HS can occupy the whole buttock and lead to deformation of the buttock (Figure 61). The keloids and HS can extend down onto the thigh (Figure 62).

The keloids and abscesses of HS can occur in non-typical areas that include the abdomen (Figure 63). Sometimes, the reason for the formation of abscesses is that there is an overlying abdominal pannus, which causes occlusion and friction of the skin (Figure 64). Keloidal HS abscesses can also form on the chest, (Figure 65) abdomen (Figure 66), and neck (Figure 67). HS keloids can involve the vulva and legs and co-exist with abscesses (Figure 68). These keloids can take a variety of forms, although usually the typical shape of keloids is oval and can also occur on arms and axilla (Figure 69). A verrucous keloidal abscess of the buttock is one presentation of HS (Figure 70) [22]. Keloids can extend from the axilla onto the chest and arms (Figure 71). After multiple surgeries, HS can manifest as keloids in the area where the surgery was performed (Figure 72).
Figure 58. Keloids in the axilla from burnt-out HS abscesses in the area ©Noah Scheinfeld

Figure 59. Keloids of the axilla that are extending on to the chest ©Noah Scheinfeld
**Figure 60.** HS that has extended to the leg with morphea-like induration. The follicular ostia and raised keloidal scar show that this is HS. ©Noah Scheinfeld

**Figure 61.** HS involving the buttock that extends to the legs; abscesses are also present ©Noah Scheinfeld
Figure 62. Keloidal HS extending from groin to upper thighs ©Noah Scheinfeld

Figure 63. HS abscesses on the abdomen ©Noah Scheinfeld
Figure 64. Keloids that have developed under the breast

Figure 65. An isolated keloidal nodule of HS on the upper lateral chest
Figure 66. Isolated keloidal abscess of the abdomen; note the follicular ostia

Figure 67. Combination HS abscess and keloid of the neck
Figure 68. Keloidal abscesses of buttocks Hurley Stage 3 ©Noah Scheinfeld

Figure 69. Keloidal abscesses HS of the arm, chest and axilla ©Noah Scheinfeld
Figure 70. A verrucous keloidal abscesses HS of the buttock ©Noah Scheinfeld

Figure 71. HS in the axilla with some abscess activity note the extension of abscesses on to the lateral chest ©Noah Scheinfeld
Skin tags

One of the scarring responses of HS is the development of skin tags (Figure 51 and Figure 52). This is not a common phenomenon. One report has noted polypoid HS on the anal verge [23], but this finding is rare. It is interesting that skin tags can also develop in Crohn disease [24]. Their precise etiology is unclear but probably involves one of body’s responses to repetitive scarring activity.
HS and ulceration or erosions

While HS is a follicular disease and because follicles break down, they can leave behind erosions and ulcerations (Figure 52, Figure 53, Figure 54, Figure 55)[25]. Care must be taken when ulceration appears in the gluteal, anal, perianal, and perineal areas, in particular, if they have been present for more than 20 years because they might be squamous cell cancers [26]. Squamous cell cancers have not been reported in the axilla and flank. It is not clear if these ulcers and erosions evolve from abscesses or simply from a breakdown of the follicular unit.
Figure 53. Erosions in axilla with keloidal abscess ©Noah Scheinfeld

Figure 54. Erosion of the gluteal area with abscess formation and scarring on the buttocks ©Noah Scheinfeld
Acanthosis Nigricans and HS.

Although only a few reports have linked HS with acanthosis nigricans [27], I believe that this association is underreported. Many patients with HS are obese and likely type 2 diabetics. Obesity is one of the common associations of HS and so it is no surprise that HS can occur with acanthosis nigricans, particularly in the axilla (Figure 56, Figure 57).
Patterns of Scarring other than Keloids in Hidradenitis

There are a number of patterns of scarring in HS that are not keloidal. They include white stellate scars (Figure 58, Figure 59) and white atrophic skin. There may be breakdown on these atrophic areas. Sometimes white scars can develop on an erythematous base (Figure 60). In the end, the inflammation will burn out, leaving white atropic scars in its wake. On other occasions while the inflammation is still active, the skin underneath the stellate scars breaks down leaving stellate erosions (Figure 61).

Figure 57. Acanthosis nigricans of axilla with some abscess activity; the circinate scars imply that a fistula has formed under the skin ©Noah Scheinfeld

Figure 58. Abscess formation on the thigh with white stellate scar where abscess have resolved co-existing with area of HS keloidal/abscess. ©Noah Scheinfeld
Figure 59. White stellate scar where abscesses have resolved on the abdomen. ©Noah Scheinfeld

Figure 60. White groin scars on an active inflammed erythematous base ©Noah Scheinfeld
The Fistula

Fistulas are a common finding in Stage 2 and 3 HS. One writer has gone so far to clam that mammilary fistular should join the tetrad to become a pentad [28]. I disagree because this finding is uncommon. HS has a tendency to form fistulae owing to the fact that the hyperkeratosis of the follicle causes the follicular wall to burst and to spread its content and inflammatory mediators horizontally to join follicular units together. If you press down on one side, pus can come out centimeters away. The configuration of the fistula can be circinate (figure 62), diagonal (Figure 63), linear (Figure 64), or arcuate (Figure 65). either way simple abscess stand joined like so many stations on the Cross (note the out pouching on Figure 65). The crust on Figure 62 implied that fluid is still leaking out and even evaporating so that the osita of HS lesion is has dried material on top of it.
Figure 62. Circinate Scar overlies a fistulae of HS on torso/axilla ©Noah Scheinfeld

Figure 63. Diagonal Scared HS Fistula of the axilla ©Noah Scheinfeld

Figure 64. Linear scared HS fistula in the groin ©Noah Scheinfeld
Scars left after inflammation of HS has subsided.

Once the inflammation of the HS resolved after destruction of follicles, fibrosis forming a divot or depression can remain where a follicle had been. This likely relates to the type of inflammatory mediators and the timing. The divots and depressions are manifestation of burnt-out HS. These divots or depressed scars can take on a number of forms. Figure 65 represents a
combination of divot scars and active keloidal abscesses. Figure 66 demonstrates pitted scars of HS on buttocks with one active abscess. Figure 67 shows mostly quiescent HS with one divot present in the groin.

The destruction HS leaves behind can be more than just ice pick divots. Figure 68 shows depressions and pits on the scrotum with quiescent HS. Figure 69 combines active HS abscesses with pits (divots) in a burned out area of HS. Figure 70 shows divots, depressions, acne inversa, and abscesses co-existing. "Honeycomb" scarring (contiguous divot formation) can occur with HS, most commonly on the scrotum (Figure 71, Figure 72). Honeycomb scarring can be described by the word cribriform, which means pierced with holes, sieve-like divots. Anetoderma scarring on the abdomen in HS is shown in Figure 73. This followed an active case of HS folliculitis in the area.
Figure 67. Pitted scar of groin ©Noah Scheinfeld

Figure 68. Depressions and divots of the scrotum from burned-out HS ©Noah Scheinfeld

Figure 69. Pitted scars of groin co-existing with abscesses ©Noah Scheinfeld
Figure 69. Pitted scars of breast co-existing with acne inversa and abscesses ©Noah Scheinfeld

Figure 70. Pitted scars of breast co-existing with acne inversa and abscesses ©Noah Scheinfeld
Figure 71. Honeycomb scarring of the upper scrotum ©Noah Scheinfeld

Figure 72. Honeycomb scarring of scrotum generalized ©Noah Scheinfeld

Figure 73. Anteoderma scarring on the abdomen in HS

Figure 74. Anteoderma scarring on the abdomen in HS with active abscess
Hidradenitis can also be associated with anal fissures (Figure 76), but these can resolve with treatment, just as HS can respond to medical treatment [29].

Distortion of tissue architecture by HS

HS can destroy the normal architecture of the skin. Figure 73 shows how HS distorted the umbilicus, creating a fold where an abscess was and a scar is still apparent. HS and the abscesses that it causes can distort the normal appearance of the groin (Figure 74). Figure 75 shows how the combination of obesity and HS driven inflammation distortsthe tissue architecture (Figure 76).
Figures 77 and 78 show how the follicle, which is plugged by HS, can be degraded and be made flaccid under the barrage of inflammation. Clearly the pattern of HS follicular scarring is distinct and is different than the scarring of Favre-Racouchot. However, both can leave behind black heads and patulous follicles. Figure 80 shows how HS and pilonidal cysts distort the symmetry at the top of buttocks.

**Figure 73.** Distortion (fold addition) of the umbilicus by HS ©Noah Scheinfeld

**Figure 74.** Destruction groin architecture by HS which is still active ©Noah Scheinfeld

**Figure 75.** Active HS degrades tissue and leads to tissue distortion ©Noah Scheinfeld
Figure 76. Figure HS scarring degrades the flank tissue architecture; skin tag is present © Noah Scheinfeld

Figure 77. HS scarring degrades the tissue architecture of the skin and follows the skin lines © Noah Scheinfeld
The inflammation that HS engenders can lead to the development of a variety of tissue distortions. The scars can have an oval appearance, a linear appearance, a fishmouth appearance, or a pierced with holes appearance. Figure 79 shows a fishmouth keloidal type scar, which is also pitted on the groin and leg. Figure 80 shows an active abscess, depression, pits, brown scars, scars that resemble striae, wrinkling, and at least one pustule; these are all present in one small area of skin. Sometimes stage I HS burns itself out leaving only brown oval scars in its wake (Figure 78). The patient in Figure 78 had active HS at age 16 but lost weight and went into a permanent remission.
Edema and HS

HS can be present as lymphedema of the genital organs. One such presentation is scrotal elephantiasis secondary to hidradenitis suppurativa [30, 31]. Edema of the scrotum and penis is shown in Figures 80 and 81.

Lymphedema as a complication of hidradenitis suppurativa in three patients has been noted [32]. Hidradenitis suppurativa complicated by severe lymphedema and lymphangiectasias in a case that developed into squamous cell carcinoma has been noted. Although the precise mechanism by which HS leads to lymphedema is not known, it is postulated that the chronic, recurrent inflammation and scarring, which occurs in patients with HS, leads to the blockade/destruction of lymphatic drainage routes [33]. Vulvar edema can also occur in HS [34].
Figure 80. Scrotal edema in patient with Stage 3 HS ©Noah Scheinfeld

Figure 81. Scrotal edema in patient with Stage 3 HS penis enlarged ©Noah Scheinfeld
The follicular occlusion tetrad and PG

HS, AC, DC, and PNs have been grouped together to form the follicular occlusion tetrad. I have found that despite seeing hundreds of HS patients the occurrence of all four in one patient is rare. I note herein one patient who at one time or another did have all four: HS, AC, DC, and PNs (Figure 82, Figure 83, Figure 84, Figure 85). I have also seen a patient with pyoderma gangrenosum (one of HS more common associations) with stage 3 HS, so include excellent images of this patient in this atlas. Finally, I finish off this atlas with images of metastatic Crohn disease, which is an imitator of HS. This was differentiated from HS in a CD patient by the presence of neutrophils in its infiltrates.

Figure 82. Dissecting cellulitis in patient with follicular occlusion tetrad ©Noah Scheinfeld

Figure 83. Remnants of AC of face in patient with follicular occlusion tetrad ©Noah Scheinfeld
Figure 84. Scarring of back with some active pustules and nodules in patient with follicular occlusion tetrad ©Noah Scheinfeld

Figure 85. Somewhat active HS of the axilla in patient with follicular occlusion tetrad ©Noah Scheinfeld
Figure 86 Excision scar from removal of pilonidal cyst in patient with follicular occlusion tetrad ©Noah Scheinfeld

Figure 87 Stage 3 HS, which was accompanied by pyoderma gangrenosum ©Noah Scheinfeld
Figure 88 Stage 3 HS, which was accompanied by pyoderma gangrenosum ©Noah Scheinfeld

Figure 89 Stage 3 HS, which was accompanied by pyoderma gangrenosum ©Noah Scheinfeld
Figure 90. Pyoderma gangrenosum in patient accompanied by Stage 3 HS ©Noah Scheinfeld

Figure 91 Pyoderma gangrenosum in patient accompanied by Stage 3 HS ©Noah Scheinfeld
Conclusion

HS is a reaction pattern rather than a signal entity. Efforts have been made to parse the types of HS and in this schema there are 3 types of HS. These three phenotypes ("axillary-mammary", "follicular", and "gluteal") are shown in Table 5 [3]. I find this typing too narrow, based on the morphologies that I have outlined in this paper. I think that by having an atlas of the types of HS, it will assist creating a taxonomy of clinically useful phenotyping. In table 6, I categorize HS a little differently, more by its genetics and its clinical associations. To conclude, HS is dynamic protean disease. The full taxonomy needs more precise definition.

<table>
<thead>
<tr>
<th>Type 1</th>
<th>Type 2</th>
<th>Type 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Majority Women</td>
<td>Majority men</td>
<td>Between type 1 and Type 2 in sexual distribution</td>
</tr>
<tr>
<td>Axillary-mammary</td>
<td>Follicular</td>
<td>Gluteal involvement</td>
</tr>
<tr>
<td>Had a high probability of breast and armpit lesions (.74) and hypertrophic scars</td>
<td>Almost all had breast and armpit lesions (.96)</td>
<td>Breast involvement least</td>
</tr>
<tr>
<td>Higher History of Acne (21%)</td>
<td>History of acne (47%)</td>
<td>Low history of acne 16%</td>
</tr>
<tr>
<td>Comparable smoking to type 2</td>
<td>Comparable smoking to type 1</td>
<td>Highest rate of current smoking</td>
</tr>
<tr>
<td>Family history (29%)</td>
<td>Highest Family History (44%)</td>
<td>Familial disease 37%</td>
</tr>
<tr>
<td>Obesity more common</td>
<td>Obesity less common</td>
<td>Least Obesity</td>
</tr>
<tr>
<td>More severe diseases</td>
<td>More severe disease</td>
<td>Less severe diseases</td>
</tr>
</tbody>
</table>

Table 6 Way of classifying HS based on external factors

| HS isolated Stage 2 or Stage 3 | Genetic defect (up to 1/3 of cases) |
| HS isolated                    | Arthritis                         |
| HS with follicular occlusion usually not all 4 association are present in the same patient | One of more of the following pilonidal cysts, acne conglobata, dissecting cellulitis |
| HS                            | Crohn's disease associated        |

References


