

ORIGINAL ARTICLE



Clinical presentation and management outcome of hidradenitis suppurativa: a developing country's perspective

Muhammad Saaq

Department of Plastic Surgery and Burns, National Institute of Rehabilitation Medicine, Islamabad, Pakistan

ABSTRACT

Background: Hidradenitis suppurativa (HS) is a chronic inflammatory skin condition. The protracted and recurrent nature of the illness has serious physical and psychological repercussions for the unfortunate sufferers. Public awareness and understanding about the course and likely outcome would help to improve the overall outcome of management. The current study was conducted to determine the clinical presentation and management outcome of HS with a standard trimodal therapeutic approach.

Methods: This descriptive study was carried out at the National Institute of Rehabilitation Medicine, Islamabad, over a period of seven years. All patients who presented with HS during the study period were included. Non-consenting patients were excluded.

Results: Out of 31 patients, there were 22 males (70.96%) and 9 females (29.03%). The age range was 12-41 years, with a mean age of 27.93 ± 6.73 years. 90.32% of them were adults. The anatomic locales most commonly affected were the axillae (n=31; 100%), groins (n=9; 29.03%), and buttocks (n=5; 16.12%). 17 (54.83%) patients had stage III disease, whereas 8 (25.80%) patients had stage I disease, and 6 (19.35%) patients had stage II disease. Family history of HS was positive among 4 (12.90%) patients. The most common comorbidities identified included obesity (n=9; 29.03%), hypertension (n=2; 6.45%), and polycystic ovarian syndrome (PCOS) (n=2; 6.45%). The various surgical interventions instituted included incision and drainage (n=7; 22.58%), surgical deroofting (n=4; 12.90%), wide local excisions and reconstruction with skin grafts or local pedicled flaps (n=19; 61.29%). There was no local recurrence at two years following the radical excision and reconstruction; however, the emergence of new HS lesions at other sites was observed among 17.64% of these patients.

Conclusion: In the studied population, the disease was found to be significantly more prevalent among adult males. The majority of them had stage III disease. The most common associated comorbidities were obesity, hypertension, and PCOS. The majority of the patients were successfully managed with wide local excisions and reconstruction with skin grafts or local pedicled flaps.

KEYWORDS

Hidradenitis suppurativa; Wide local excision; Skin grafting; Local flaps; Apopilosebaceous unit; Apocrine glands; Hair follicle

ARTICLE HISTORY

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Introduction

Hidradenitis suppurativa (HS) is a chronic inflammatory ailment primarily involving the hair follicles. Its typical course is characterized by the formation of deep-seated nodules, painful abscesses, pus-draining fistulae, and sinus tracts involving the skin and subcutaneous tissue planes. Chronic inflammation leads to typical scarring and puckering of the skin. The axillae and groins represent the most commonly affected anatomic locales; however, it is not uncommon to find the involvement of other intertriginous body regions such as the inframammary folds, intergluteal sites, buttock regions, mons pubis, retro auricular areas, scalp and the nape of the neck. The condition affects approximately 1-4% of the global population. It considerably jeopardizes the quality of life of the sufferer. It is associated with serious physical and psychological repercussions for the sufferers [1-3].

The pathogenesis of HS is complex and continues to be explored. The inflammatory process is thought to be centered on follicular hyperkeratosis within the apo-pilosebaceous unit (Figure 1). This structural unit contains a hair follicle containing hair and the associated sebaceous and apocrine sweat glands. The sebaceous gland secretes sebum, whereas the apocrine gland secretes sweat through its short ducts that pass through the hair follicle near the skin surface. Initially, follicular hyperkeratosis occurs, resulting in plugging and dilation that eventually ruptures the follicle. It triggers the inflammatory process that leads to abscess formation and other manifestations of the HS disease. The emerging research is providing new insights into the involvement of cytokines in the pathogenesis of HS [3-6]. The current study was conducted to document the clinical presentation and management outcome of HS in our population.

*Correspondence: Dr. Muhammad Saaq, Department of Plastic Surgery and Burns, National Institute of Rehabilitation Medicine, Islamabad, Pakistan, e-mail: muhammadsaaq5@gmail.com

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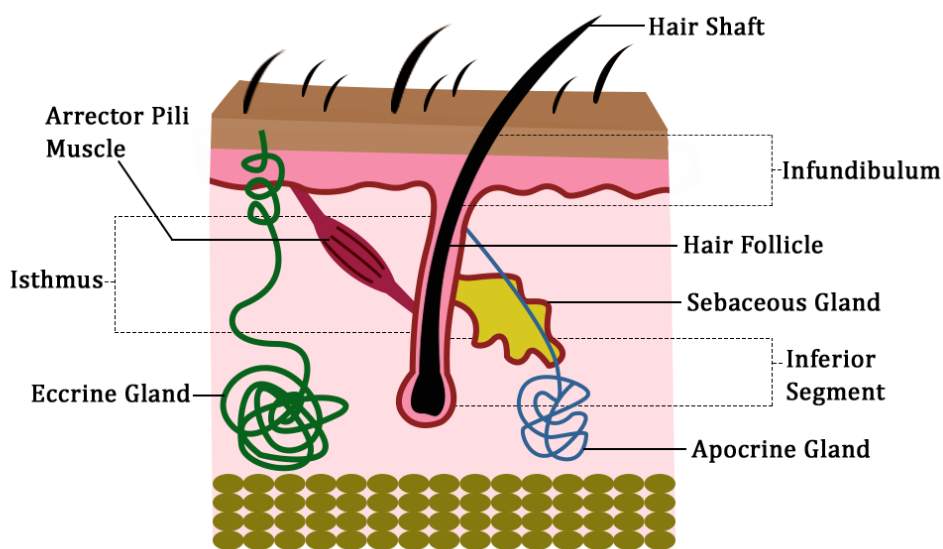


Figure 1. The Apo-pilosebaceous unit consists of a sebaceous gland and an apocrine sweat gland, both draining their secretions through their short ducts into the associated hair follicle, near the surface opening on epidermis. The arrector pili muscle connects the pilosebaceous apparatus to the epidermis.

Materials and Methods

This descriptive case series was carried out at the Department of Plastic Surgery, National Institute of Rehabilitation Medicine (NIRM), Islamabad, over a period of seven years. Written informed consent was taken from the patients. Being an observational study, it was performed in conformity to the Helsinki's declaration of 1975, as revised in 2008. Patient anonymity was ensured. Non-probability consecutive sampling was done. All patients who presented with HS during the study period were included. Non-consenting patients were excluded. The patients were initially assessed with history and detailed local examination, particularly of the intertriginous regions. Baseline investigations were performed to assess the general health and rule out any associated systemic derangements.

The HS was classified by employing the famous Hurley system of classification [7]. The following criteria were employed: 1) Stage 1 disease: Formation of single or multiple isolated abscesses. No formation of sinus tracts or presence of scarring; 2) Stage 2 disease: Formation of recurrent abscesses plus sinus tracts and scars which are separated from one another with intervening unaffected normal skin; 3) Stage 3 disease: Presence of more diffuse disease affecting an entire anatomic region, sparing no intervening normal skin. Typically, there will be multiple abscesses and sinus tracts, which will be interconnected.

Trimodal treatment protocol was employed for managing the patients. This three-pronged approach included educating the patients regarding the adoption of precautionary measures, medical management of the inflammatory flares and infection, and surgical therapy for any abscesses, septic nodules, fistulae, sinus tracts, and disfiguring overlying puckering skin. Most of the patients were managed with variable contributions from all three prongs of the treatment protocol, as dictated by the severity of their HS disease. The patients with extensive and recalcitrant lesions were managed with radical or wide local excision. The excision specimens were subjected to both histopathological and

bacteriologic examinations. The resultant defects were reconstructed with meshed skin grafts and local flaps. The reconstructions were performed either immediately (where the excisional wound was clean) or after an interval of temporization with vacuum-assisted therapy (VAC) dressings to eradicate the infection from the grossly contaminated excisional defect. The cases that underwent wide local excisions were follow-ups of two years to document any early recurrence of the disease.

The demographic profile of the patients, history of smoking, family history of the disease, duration of the HS, age of onset of the illness, any associated comorbid conditions, anatomic locales affected with the HS lesions, and type of surgical procedure undertaken were all recorded. The below figures show some illustrative cases of HS (Figures 2-11).



Figure 2. A male aged 17 years presented with a three-month history of abscess formation in both axillae. There were isolated abscesses on both axillary regions. In this clinical photograph, one can appreciate a single area of active inflammation and abscess formation in the distal part of the axilla. There is no sinus formation or scarring. These features exemplify the Hurley Stage I hidradenitis suppurativa.



Figure 3. A 19-year-old male presented with a two-year history of recurrent abscess formation in his axillae. The abscesses responded to a combination of surgical deroofing, topical antibiotics, and oral doxycycline therapy. In this clinical photograph, one can visualize scarred areas and healed sinus tracts which are clearly separated from one another with intervening normal cutaneous tissues. These features typify the Hurley Stage II hidradenitis suppurativa.



Figure 4. A 32-year-old male presented with a seven-year history of recurrent abscess formation and purulent discharge involving both axillary regions. In this clinical photograph, one can appreciate the diffuse affliction of the axillary region with the disease process. The skin of the entire anatomic region of the axilla is affected, and no skin is spared of the disease. There is active inflammation with the formation of multiple abscesses. The multiple sinus tracts are interconnected. These features are typical of the Hurley Stage III hidradenitis suppurativa.



(A)



(B)

Figure 5. A female aged 17 years had polycystic ovarian syndrome and associated cosmetically disfiguring acanthosis nigricans involving the axillae, groins, and nape of the neck. The groin regions were spared of any lesions. She presented with Hurley Stage II hidradenitis suppurativa involving the axillae (B) and nape of the neck (A). The clinical photograph (A) shows the involvement of the nape of the neck, which is a rare site of affliction with hidradenitis suppurativa. There were multiple discrete abscesses and septic nodules involving the region. The patient presented with Hurley Stage II disease.



Figure 6. A male aged 33 years presented with stage III disease involving the groins, suprapubic region, scrotum, and natal cleft areas in addition to the axillae.



Figure 7. A male aged 38 years who had hidradenitis suppurativa of nine years duration. He had active lesions involving the groin and perineal regions. There were multiple abscesses, septic nodules, and sinus tracts which were interconnected. These features reflect Hurley Stage III disease.



Figure 8. Clinical photograph of a patient with Hurley Stage III hidradenitis suppurativa, showing the pattern of involvement of the scrotum and perineum.

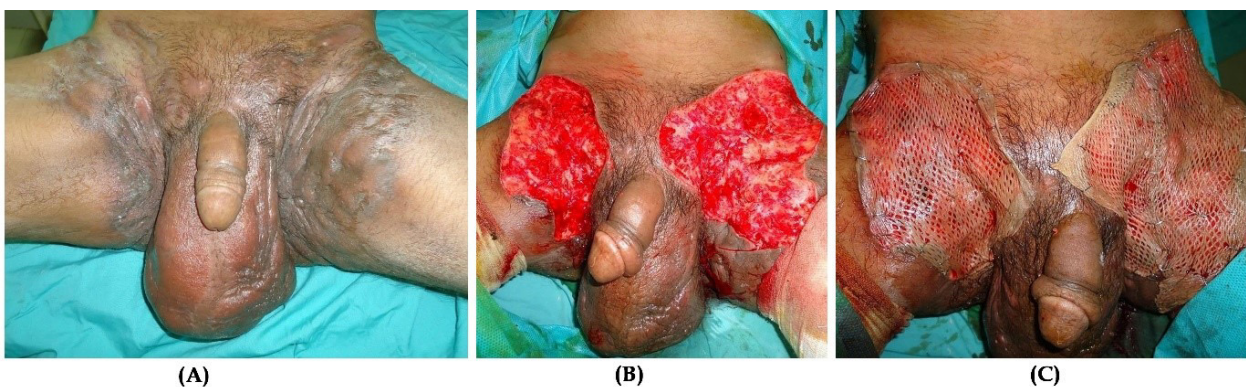


Figure 9. (A) A 41-year-old patient with Hurley Stage III hidradenitis suppurativa. One can appreciate the extensive involvement of the groin regions and the scrotum. (B) This clinical photograph shows the radical excision of the affected groin regions. The scrotal lesions were excised after an interval of three months. (C) The defect that resulted from radical excision of the groin regions is resurfaced with split-thickness meshed skin graft harvested from the thigh.

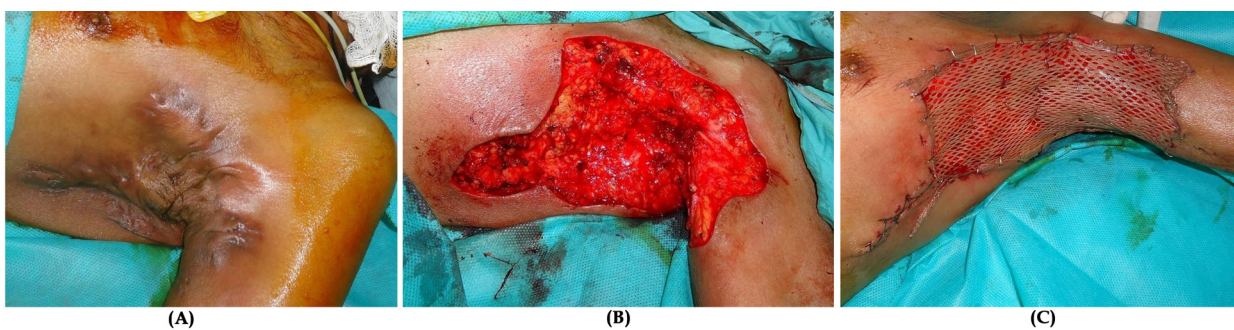


Figure 10. (A). Clinical photograph showing the characteristic appearance of axilla in Hurley Stage III hidradenitis suppurativa. (B) The clinical photograph shows radical excision of the affected axilla. (C) The resultant defect has been skin grafted with meshed split thickness skin graft harvested from the thigh.



Figure 11. Clinical photograph showing the most frequently employed flap for the axilla following radical resection of the affected axillary skin.

Statistical analysis

The data were analyzed through the Statistical Package for Social Sciences version 22. Descriptive statistics were used to calculate frequencies, percentages, means, and standard deviation. The numerical data, such as the patient's age, duration of the HS, and duration of HS, were expressed as Mean \pm Standard deviation. In contrast, the categorical data, such as the anatomic locales affected by the HS procedures performed, were expressed as frequency and percentages. The percentages of various variables were compared by employing the chi-square test, and a p-value of less than 0.05 was regarded as statistically significant.

Results

Of the 31 patients, there were 22 (70.96%) males, whereas 9 (29.03%) females. The age range was 12-41 years. The mean age of the patients was 27.93 ± 6.73 years. The majority of the (n=28; 90.32%) patients were adults, whereas 3 (9.67%) patients were in the pediatric age group. The mean duration of the disease was 5.12 ± 1.8 years. The mean age of disease onset was 23.70 ± 5.11 years. The majority of the patients (n=20; 64.51%) presented with over five years of history of the disease. The frequencies of

involvement of different anatomic locales were as follows: axillary regions (n=31; 100%); groin regions (n=9; 29.03%); buttocks (n=5; 16.12%); scrotum (n=4; 12.90%); mons pubis/vulva region (n=2; 6.45%); inframammary regions (n=2; 6.45%); scalp (n=2; 6.45%) and nape of neck (n=1; 3.22%).

According to the Hurley system of classification, 8 (25.80%) patients presented with stage I disease, 6 (19.35%) patients had stage II disease, and 17 (54.83%) patients had stage III disease. The comorbid conditions identified among the patients included obesity (n=9; 29.03%); hypertension (n=2; 6.45%); PCOS (n=2; 6.45%); acne vulgaris (n=2; 6.45%); (40.8%), pilonidal sinus (n=2; 6.45%); diabetes mellitus (n=1; 3.22%) and smoking (n=1; 3.22%). Family history of HS was positive among 4 (12.90%) patients.

The various surgical interventions performed included incision and drainage (n=7; 22.58%), surgical deroofting (n=4; 12.90%), wide local excisions and reconstruction with skin grafts or local pedicled flaps (n=19; 61.29%). There was no local recurrence at two years following the radical excision and reconstruction with skin grafts or flaps; however, the emergence of new HS lesions at other sites was observed among 17.64% of these patients.

Table 1. Summary of the various demographic and clinical variables of interest (n=31).

Variables	Percentage	p-value
Gender:		
Male (n=22)	70.96%	0.000*
Female (n=9)	29.03%	
Age:		
Up to 30 years (n= 21)	67.74%	0.001*
>30 years (n= 10)	32.25%	
Age group:		
Adults (n= 28)	90.32%	0.000*
Children (n=3)	9.67%	
Duration of HS:		
≥ 5 years (n=27)	87.09%	0.000*
<5 years (n=4)	12.90%	
Hurley stage of HS:		
Stage I (n= 8)	25.80%	
Stage II (n= 6) Stage III (n= 17)	54.83%	0.000*
Family history of HS:		
Present (n= 4)	12.90%	0.000*
Absent (n=27)	87.09%	
Obesity:		
Present (n=9)	29.03%	0.001*
Absent (n=22)	70.96%	
Hypertension:		
Present (n=2)	6.45%	0.000*
Absent (n=29)	93.54%	
PCOS:		
Present (n=2)	6.45%	0.000*
Absent (n=29)	93.54%	
Acne vulgaris:		
Present (n=2) Absent (n=29)	6.45%	0.000*
	3.54%	
Pilonidus sinus disease:		
Present (n=2)	6.45%	0.000*
Absent (n=29)	93.54%	
Diabetes mellitus:		
Present (n=1)	3.22%	0.000*
Absent (n=30)	96.77%	
Smoking:		
Present (n=1)	3.22%	0.000*
Absent (n=30)	96.77%	

* p-value significant ≤ 0.05 ; ** p-value insignificant ≥ 0.05

Discussion

The current study provides a comprehensive picture of the epidemiological landscape of HS in our population. Our institute deals with patients referred from across the country. We have no local prevalence data of HS in our country; however,

the condition is reported to affect 1-4% of the global population.

In the current study, the age range of the affected patients was 12-41 years. The published studies have reported variable age groups afflicted by HS in their studied populations. Slyper

M et al. reported their patients falling in the age range between 18-44 years [8]. Sayed CJ et al. reported the involvement of individuals aged between 18-40 years [9]. In the current study, male predominance was observed. Our finding conforms to most of the studies based on Asian populations. They all have also reported predominant affliction of the men with the disease [10-12]. In glaring contrast to the aforementioned finding, female predominance (with a female-to-male ratio of 3:1) has been reported in Europe and North America [13]. The standardized point prevalence in the United States is approximately 2.4-fold greater among women than men [14].

In this study, all the patients had involvement of their axillae regions. Both males and females had an equal percentage of the affliction of the axillary regions; however, involvement of the groins and buttock regions was observed among males only. Contrary to these observations of the current study, some studies, particularly from developed countries, have reported a different gender-based variation in the clinical presentation of HS. They have reported a predilection of women for more frequent affliction of their axillary regions compared to the matched population of men with HS. Likewise, the involvement of the sub-mammary regions is more frequent among women than men. Most studies have reported more frequent gluteal HS among men than women [15-18]. There is a need for in-depth research to explain these gender-based differences in the clinical presentation of HS in the Asian and Western populations. One possible contributing factor is the effect of androgens on the skin.

In this study, most of the patients availed the first plastic surgery consultation when they had the disease for more than five years. Even in developed countries such as the United States, patients often have a similar significant delay in seeking appropriate medical advice [19]. An average global diagnostic delay of 7-10 years is reported in this regard. The diagnostic delay may, on the one hand, unduly prolong the miseries of the patients, and on the other hand, it discourages the collection of robust clinical epidemiological data. As a result, many efforts have been made to evolve effective strategies for disease screening [20-24]. In our part of the globe, the issue of delayed presentation could stem from a variety of factors such as 1) low literacy rate, 2) lack of awareness about the protracted course of the disease, 3) geographic remoteness from the available healthcare facilities; and 4) social, cultural and economic hurdles in receiving adequate health care. Delayed presentation results in advanced disease and more jeopardized outcomes.

In this study, 29% of the patients were obese. Vazquez BG et al. from the US reported a 54.9% prevalence of obesity among their HS patients [16]. Other studies have reported obesity among 50-75% of their HS patients. The abundant fat content of the subcutaneous tissue planes predisposes to a pro-inflammatory response. It has been observed that obese individuals tend to have more severe HS disease [25,26]. Several studies have also demonstrated a strong association of HS with metabolic syndrome. This syndrome includes insulin resistance or diabetes mellitus; hypertension; dyslipidemia; and obesity [25,27,28].

In this study, two patients had polycystic ovarian syndrome (PCOS). Garg A et al. reported a 9.0% prevalence of PCOS among women with HS [29]. Several other studies have reported a similar association between HS and PCOS; however, Barth JH et al. did not find any evidence of biochemical

hyperandrogenism among women with HS compared to the matched population of controls [30]. It is not surprising that both HS and PCOS share closely resembling demographic features. The most notable sharing features include high body mass index (BMI) and metabolic syndrome. Both conditions also have similar response rates to anti-androgen therapy [30-33].

In this study, only one male patient was a cigarette smoker. Our observation contrasts with the published literature, which has quoted higher frequency rates of smoking among HS sufferers. Does smoking trigger the HS, or is it a kind of an addiction resort by the sufferers who otherwise attempt to satisfy their associated psychological distress? The exact answer is still unknown, and the literature has not taken up this question for a formal scientific inquiry. One postulation is that Nicotine may cause adverse events such as epidermal hyperplasia, follicular plugging, neutrophil chemotaxis, cytokine production by keratinocytes, and downregulation of the antimicrobial peptides. Smoking may also impede the healing of the active lesions by reducing the cutaneous blood supply by causing vasoconstriction, jeopardizing tissue oxygenation, and impairing the healing process. Smoking cessation is recommended as one of the many remedial measures for HS patients [34-38].

In this study, three patients belonged to the pediatric age group, and all were obese. HS has been traditionally considered to be rare before the onset of puberty; however, nowadays, we do come across children afflicted with HS. Pediatric HS presents a clinical spectrum of the disease, which is comparable to that among adults. Avoidance of high body mass index and other risk factors should be ensured among these unfortunate children. These measures will delay the disease's progression and reduce its severity [39,40]. In this study, four patients had a positive family history of the HS disease. Studies have reported a positive family history of HS among 1/3rd to 1/6th of their HS patients. Certain studies have even suggested the possibility of autosomal dominant inheritance patterns among HS patients [41-43].

In this study, we followed the time-tested traditional trimodal treatment protocol for addressing the HS. All HS patients should be offered general precautionary measures to reduce the rapid progression and severity of the disease. These included education and advice regarding weight reduction, maintaining good hygiene, particularly of the intertriginous regions of the body, cessation of smoking if applicable, control of diabetes mellitus, treatment of PCOS, antibiotics for disease flares, and minor drainage procedures for any abscesses or infected nodules. Various antibiotics have been employed; however, these only help to control the flares without eradicating the disease. The antibiotics may be administered as a combination of topical and systemic antibiotics. Systemic steroids may also be added to the antibiotic therapy to induce remission [1,44-53].

In this study, most patients presented with advanced-stage HS and were managed with wide local excision of their recalcitrant and extensive lesions. The resultant defects were reconstructed with skin grafts and local flaps. The advanced stage HS (i.e., Hurley stage III) usually requires management with wide local excision of the affected hair-bearing region, followed by reconstruction of the raw areas. Several published studies have reported similar wide local excisions as the

mainstay treatment for addressing the diffuse lesions of advanced-stage HS [49,54,55].

In recent years, some case series and case reports have reported useful effects of biologic therapy by employing newer targeted Therapeutics in managing the advanced stage of HS disease. It is an exciting development and offers a new ray of hope for this unfortunate population of HS patients; however, at this preliminary stage of testing, we cannot draw any valid conclusions about the efficacy of these agents [6,49,56-58].

Conclusions

In the studied population, the disease was significantly more prevalent among adult males. The majority of them had stage III disease. The most commonly associated comorbidities were obesity, hypertension, and PCOS. Most patients were successfully managed with wide local excisions and reconstruction with skin grafts or local pedicled flaps.

Informed consent

Patients provided informed consent for the inclusion of their photographs in the study.

Disclosure statement

No potential conflict of interest was reported by the author.

References

1. Sabat R, Jemec GBE, Matusiak L, Kimball AB, Prens E, Wolk K. Hidradenitis suppurativa. *Nat Rev Dis Primers*. 2020;6(1):18.
2. Jemec GBE, Kimball AB. Hidradenitis suppurativa: Epidemiology and scope of the problem. *J Am Acad Dermatol*. 2015;73(5 Suppl 1):S4-S7.
3. Saunte DML, Jemec GBE. Hidradenitis suppurativa: Advances in diagnosis and treatment. *JAMA*. 2017;318(20):2019-2032.
4. Hoffman LK, Ghias MH, Lowes MA. Pathophysiology of hidradenitis suppurativa. *Semin Cutan Med Surg*. 2017;36(2):47-54.
5. Vossen ARJV, van der Zee HH, Prens EP. Hidradenitis suppurativa: A systematic review integrating inflammatory pathways into a cohesive pathogenic model. *Front Immunol*. 2018;9:2965.
6. Markota Cagalj A, Marinović B, Bukvic Mokos Z. New and emerging targeted therapies for Hidradenitis suppurativa. *Int J Mol Sci*. 2022;23(7):3753.
7. Hurley HJ. Axillary hyperhidrosis, apocrine bromhidrosis, hidradenitis suppurativa and familial 459 benign pemphigus. Surgical approach. In: Roenigk, R RH, eds. *Dermatologic surgery, principles and practice*. 2nd ed. New York: Marcel Dekker; 1989:623-646.
8. Slyper M, Strunk A, Garg A. Incidence of sexual dysfunction among patients with hidradenitis suppurativa: a population-based retrospective analysis. *Br J Dermatol*. 2018;179(2):502-503.
9. Sayed CJ, Hsiao JL, Okun MM. Clinical epidemiology and management of hidradenitis suppurativa. *Obstet Gynecol*. 2021;137(4):731-746.
10. Omine T, Miyagi T, Hayashi K, Yamaguchi S, Takahashi K. Clinical characteristics of hidradenitis suppurativa patients in Okinawa, Japan: Differences between East Asia and Western countries. *J Dermatol*. 2020;47(8):855-862.
11. Lee JH, Kwon HS, Jung HM, Kim GM, Bae JM. Prevalence and comorbidities associated with hidradenitis suppurativa in Korea: a nationwide population-based study. *J Eur Acad Dermatol Venereol*. 2018;32(10):1784-1790.
12. Choi E, Cook AR, Chandran NS. Hidradenitis Suppurativa: An Asian perspective from a Singaporean institute. *Skin Appendage Disord*. 2018;4(4):281-285.
13. Ingram JR. The epidemiology of hidradenitis suppurativa. *Br J Dermatol*. 2020;183(6):990-998.
14. Garg A, Kirby JS, Lavian J, Lin G, Strunk A. Sex and age-adjusted population analysis of prevalence estimates for hidradenitis suppurativa in the United States. *JAMA Dermatol*. 2017;153(8):760-764.
15. Chu CB, Yang CC, Tsai SJ. Hidradenitis suppurativa: Disease pathophysiology and sex hormones. *Chin J Physiol*. 2021;64(6):257-265.
16. Vazquez BG, Alikhan A, Weaver AL, Wetter DA, Davis MD. Incidence of hidradenitis suppurativa and associated factors: a population-based study of Olmsted County, Minnesota. *J Invest Dermatol*. 2013;133(1):97-103.
17. Yang JH, Moon J, Kye YC, Kim KJ, Kim MN, Ro YS, et al. Demographic and clinical features of hidradenitis suppurativa in Korea. *J Dermatol*. 2018;45(12):1389-1395.
18. Hayama K, Fujita H, Hashimoto T, Terui T. Japanese HS Research Group. Questionnaire-based epidemiological study of hidradenitis suppurativa in Japan revealing characteristics different from those in Western countries. *J Dermatol*. 2020;47(7):743-748.
19. Nguyen TV, Damiani G, Orenstein LAV, Hamzavi I, Jemec GB. Hidradenitis suppurativa: an update on epidemiology, phenotypes, diagnosis, pathogenesis, comorbidities and quality of life. *J Eur Acad Dermatol Venereol*. 2021;35(1):50-61.
20. Saunte DM, Boer J, Stratigos A, Szepietowski JC, Hamzavi I, Kim KH, et al. Diagnostic delay in hidradenitis suppurativa is a global problem. *Br J Dermatol*. 2015;173(6):1546-1549.
21. Garg A, Neuren E, Cha D, Kirby JS, Ingram JR, Jemec GBE, et al. Evaluating patients' unmet needs in hidradenitis suppurativa: Results from the global survey of impact and healthcare needs (VOICE) project. *J Am Acad Dermatol*. 2020;82(2):366-376.
22. Riis PT, Andersen PL, Jemec GB. Arguments for a national questionnaire-based screening for hidradenitis suppurativa in Denmark. *Acta Dermatovenerol Alp Pannonica Adriat*. 2018;27(3):115-120.
23. Vinding GR, Miller IM, Zarchi K, Ibler KS, Ellervik C, Jemec GBE. The prevalence of inverse recurrent suppuration: a population-based study of possible hidradenitis suppurativa. *Br J Dermatol*. 2014;170(4):884-889.
24. Cazzaniga S, Naldi L, Damiani G, Atzori L, Patta F, Guidarelli G, et al. Validation of a visual-aided questionnaire for the self-assessment of hidradenitis suppurativa. *J Eur Acad Dermatol Venereol*. 2018;32(11):1993-1998.
25. Menter A. Recognizing and managing comorbidities and complications in hidradenitis suppurativa. *Semin Cutan Med Surg*. 2014;33(3 Suppl):S54-S56.
26. Riis PT, Saunte DM, Benhadou F, Marmol VD, Guillem P, El-Domyati M, et al. Low and high body mass index in hidradenitis suppurativa patients-different subtypes? *J Eur Acad Dermatol Venereol*. 2018;32(2):307-312.
27. Ergun T. Hidradenitis suppurativa and the metabolic syndrome. *Clin Dermatol*. 36(1):41-47.
28. Lim ZV, Oon HH. Management of hidradenitis suppurativa in patients with metabolic comorbidities. *Ann Dermatol*. 2016;28(2):147-151.
29. Garg A, Neuren E, Strunk A. Hidradenitis suppurativa is associated with polycystic ovary syndrome: A population-based analysis in the United States. *J Invest Dermatol*. 2018;138(6):1288-1292.
30. Barth JH, Layton AM, Cunliffe WJ. Endocrine factors in pre- and postmenopausal women with hidradenitis suppurativa. *Br J Dermatol*. 1996;134:1057-1059.
31. Ingram JR, Jenkins-Jones S, Knipe DW, Morgan CLI, Cannings-John R, Piguet V. Population-based clinical practice research datalink study using algorithm modeling to identify the true burden of hidradenitis suppurativa. *Br J Dermatol*. 2018;178(4):917-924.
32. Kimball AB, Sundaram M, Gauthier G, Guerin A, Pivneva I, Singh R, et al. The comorbidity burden of hidradenitis suppurativa in the United States: a claims data analysis. *Dermatol Ther (Heidelb)*. 2018;8(4):557-569.
33. Phan K, Charlton O, Smith SD. Hidradenitis suppurativa and diabetes mellitus: updated systematic review and adjusted meta-analysis. *Clin Exp Dermatol*. 2019;44(4):e126-e132.
34. Akdogan N, Alli N, Uysal PI, Topcuoglu C, Candar T, Turhan T. Visfatin and insulin levels and cigarette smoking are independent

- risk factors for hidradenitis suppurativa: a case-control study. *Arch Dermatol Res.* 2018;310(10):785-793.
35. Sartorius K, Emtestam L, Jemec GBE, Lapins J. Objective scoring of hidradenitis suppurativa reflecting the role of tobacco smoking and obesity. *Br J Dermatol.* 2009;161(4):831-839.
 36. Acharya P, Mathur M. Hidradenitis suppurativa and smoking: A systematic review and meta-analysis. *J Am Acad Dermatol.* 2020;82(4):1006-1011.
 37. Deilhaes F, Rouquet RM, Gall Y, Aquilina C, Paul C, Konstantinou MP. Profile of smoking dependency in hidradenitis suppurativa patients and smoking cessation outcomes. *J Eur Acad Dermatol Venereol.* 2020;34(12):e790-e791.
 38. Mokos ZB, Mise J, Balic A, Marinovic B. Understanding the relationship between smoking and hidradenitis suppurativa. *Acta Dermatovenerol Croat.* 2020;28(1):9-13.
 39. Garcovich S, Fania L, Caposiena D, Giovanardi G, Chiricozzi A, De Simone C, et al. Pediatric hidradenitis suppurativa: A cross-sectional study on clinical features and treatment approaches. *J Cutan Med Surg.* 2022;26(2):127-134.
 40. Micheletti RG. Hidradenitis suppurativa: current views on epidemiology, pathogenesis, and pathophysiology. *Semin Cutan Med Surg.* 2014;33(3 Suppl):S48-S50.
 41. Vinkel C, Thomsen SF. Hidradenitis suppurativa: Causes, features and current treatments. *J Clin Aesthet Dermatol.* 2018;11(10):17-23.
 42. Constantinou CA, Fragoulis GE, Nikiphorou E. Hidradenitis suppurativa: infection, autoimmunity or both? *Ther Adv Musculoskelet Dis.* 2019;11:1759720X19895488.
 43. Knaysi GA Jr, Cosman B, Crikelair GF. Hidradenitis suppurativa. *JAMA.* 1968;203(1):19-22.
 44. Zouboulis CC, Bechara FG, Dickinson-Blok JL, Gulliver W, Horváth B, Hughes R, et al. Hidradenitis suppurativa/acne inversa: A practical framework for treatment optimization - systematic review and recommendations from the HS ALLIANCE working group. *J Eur Acad Dermatol Venereol.* 2019;33(1):19-31.
 45. Farrell AM, Randall VA, Vafaee T, Dawber RP. Finasteride as a therapy for hidradenitis suppurativa. *Br J Dermatol.* 1999;141(6):1138-1139.
 46. Danby FW, Hazen PG, Boer J. New and traditional surgical approaches to hidradenitis suppurativa. *J Am Acad Dermatol.* 2015;73(5 Suppl 1):S62-S65.
 47. Ortiz CL, Castillo VL, Pilarte FS, Barraguer EL. Experience using the thoracodorsal artery perforator flap in axillary hidradenitis suppurativa cases. *Aesthetic Plast Surg.* 2010;34(6):785-792.
 48. Soldin MG, Tulley P, Kaplan H, Hudson DA, Grobelaar AO. Chronic axillary hidradenitis: the efficacy of wide excision and flap coverage. *Br J Plast Surg.* 2000;53(5):434-436.
 49. Falola RA, DeFazio MV, Anghel EL, Mitnick CDB, Attinger CE, Evans KK. What heals hidradenitis suppurativa: Surgery, immunosuppression, or both? *Plast Reconstr Surg.* 2016;138(3 Suppl):219S-229S.
 50. Prens E, Deckers I. Pathophysiology of hidradenitis suppurativa: An update. *J Am Acad Dermatol.* 2015;73(5 Suppl 1):S8-S11.
 51. Rambhatla PV, Lim HW, Hamzavi I. A systematic review of treatments for hidradenitis suppurativa. *Arch Dermatol.* 2012;148(4):439-446.
 52. Bettoli V, Join-Lambert O, Nassif A. Antibiotic treatment of hidradenitis suppurativa. *Dermatol Clin.* 2016;34(1):81-89.
 53. Scheinfeld N. Hidradenitis suppurativa: A practical review of possible medical treatments based on over 350 hidradenitis patients. *Dermatol Online J.* 2013;19(4):1.
 54. Gierek M, Ochała-Gierek G, Kitala D, Łabuś W, Bergler-Czop B. Surgical management of hidradenitis suppurativa. *Postepy Dermatol Alergol.* 2022;39(6):1015-1020.
 55. Manfredini M, Garbarino F, Bigi L, Pellacani G, Magnoni C. Hidradenitis suppurativa: Surgical and postsurgical management. *Skin Appendage Disord.* 2020;6(4):195-201.
 56. Kimball AB, Jemec GBE, Alavi A, Reguiui Z, Gottlieb AB, Bechara FG, et al. Secukinumab in moderate-to-severe hidradenitis suppurativa (SUNSHINE and SUNRISE): week 16 and week 52 results of two identical, multicentre, randomised, placebo-controlled, double-blind phase 3 trials. *Lancet.* 2023;401(10378):747-761.
 57. Ruggiero A, Martora F, Picone V, Marano L, Fabbrocini G, Marasca C. Paradoxical hidradenitis suppurativa during biologic therapy, An emerging challenge: A systematic review. *Biomedicines.* 2022;10(2):455.
 58. Chen SX, Greif C, Gibson RS, Porter ML, Kimball AB. Advances in biologic and small molecule therapies for hidradenitis suppurativa. *Expert Opin Pharmacother.* 2022;23(8):959-978.