Hidradenitis Suppurativa: Inside and Out

Sharmila Patil, Astuty Apurwa, Nitin Nadkarni, Shweta Agarwal, Parag Chaudhari, Manjyot Gautam

Abstract

Hidradenitis suppurativa is a chronic, disabling, suppurative disease characterized by deep tender subcutaneous nodules; complicated by fibrosis and extensive sinuses affecting primarily the apocrine gland bearing areas. It affects all races in early 20s with greater prevalence seen in women (3 to 5:1). The estimated disease prevalence is 1 - 4 %. The disease is speculated to be caused by follicular structural abnormalities with associated risk factors as smoking, obesity, positive family history and shaving. Certain co-morbidities can also be seen such as inflammatory bowel disease, spondyloarthropathies, epithelial tumors, pyoderma gangrenosum etc. Treatment modalities include counseling of the patient to lose weight if obese, to wear loose clothes, stop smoking and maintain good hygiene. Topical antibiotics, like 1% clindamycin, have shown to give good results along with benzoyl peroxide wash. Orally cocktail of antibiotics can be given, though biologicals remain the best treatment option. Surgical excision can be done in later stages and in recalcitrant cases.

KEY WORDS: Acne inversa, acne triad, adalimumab, Tapocrine gland, hidradenitis suppurativa, Hurley's staging, metformin, Sartorius system

History

Hidradenitis suppurativa (HS), first described by Velpeau in 1839 is eponymously associated with Verneuil. Pilsbury in 1956 described it as a component of the “acne triad.” In 1975, Plewig and Kligman modified the triad to tetrad by adding pilonidal sinus to it and gave the name “acne inversa.” It has also been called as apocrine acne, apocrinitis, fox den disease, pyoderma fistulans significa, Velpeau’s disease, and Verneuil's disease.

Introduction

HS is a chronic, disabling, and suppurative disorder characterized by deep tender subcutaneous nodules complicated by fibrosis and extensive sinuses mainly affecting apocrine gland-bearing areas.

Epidemiology

Most of the studies on HS are from Western sources and Indian or Asian studies are not available. The point prevalence of HS in the USA is 4%, while the 1-year prevalence is around 1%.

It begins after puberty affecting predominantly women, but a study from Korea suggested a male preponderance.

Etiopathogenesis

There are multiple theories regarding the etiology.

The first theory suggests that it is a disease of the apocrine gland, but histopathologically, it has been shown to involve the pilosebaceous unit (hence also called acne inversa). The primary cause is said to be follicular structural abnormality leading to secondary inflammatory reactions. There is an alteration of terminal follicular epithelium with dilatation and distortion of the upper infundibular tract which occludes the affected hair follicles leading to subsequent bacterial infection.

Alteration in the antibacterial, antifriction, endocrine and inflammatory modulatory function of the sebaceous gland is speculated to lead to further cascade of events seen in HS.

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 4.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms. For reprints contact: reprints@medknow.com

How to cite this article: Patil S, Apurwa A, Nadkarni N, Agarwal S, Chaudhari P, Gautam M. Hidradenitis suppurativa: Inside and out. Indian J Dermatol 2018;63:91-8.

Received: July, 2016. Accepted: January, 2018.
The third theory suggests hormonal influence, as HS has been associated with polycystic ovarian syndrome (PCOS), irregular menstruation, hirsutism, premenstrual flare, acne, high total testosterone concentration, and high free androgen index. Furthermore, the reduction is seen in disease after menopause and during pregnancy.

The fourth theory suggested immunological dysregulation by upgrading of toll-like receptors (TLR). There is exaggerated expression of TLR-2 in macrophages and dendritic cells infiltrating the dermis.

The fifth theory suggested exaggerated formation of free oxygen radicals from neutrophils.

Clinical Features

The primary sites are apocrine gland rich areas such as axillae, perianal, and perineum. It may also affect the neck, retroauricular area, adjacent scalp, back, buttocks, scrotum or labia, and inframammary or mammary region in women. Multiple sites can be involved. In women, inquinal and mammary regions are more commonly affected while in men, anogenital region is more affected.

The primary lesion starts as blind, erythematous, subcutaneous, firm, tender nodule approximately of 0.5–1.5 cm, often preceded by pruritus and discomfort. In the beginning, it can readily be confused with an infected sebaceous cyst or a furuncle. Polymorphous comedones are characteristically present in the lesional and perilesional areas. The nodules can remain dormant for few days to months or form abscess which in few hours to days breaks through the skin to form sinuses yielding malodorous purulent or seropurulent discharge. There can be subcutaneous extension leading to formation of indurated plaques and thick linear bands. The abscess can also ulcerate, burrow, and perforate the neighboring structures. There can be episodes of acute cellulitis, fever, and toxicity intermittently.

The lesions often do not subside completely; there is always indolent inflammation and drainage. They subside with fibrosis and band-like scars which restrict the mobility of limbs, and activity of the patient.

There are multiple scoring systems devised to evaluate the severity and stages of the disease; the most common being Hurley's staging system, Modified Sartorius scoring, Hidradenitis suppurativa severity index, Hidradenitis suppurativa physical global assessment scale and Acne inversa severity index [Tables 1-4].

Hurley’s Staging System:

- Stage I: Solitary or multiple isolated abscess formation without scarring and any sinus tract formation [Figure 1]
- Stage II: Recurrent abscess, single or multiple widely separated lesions with formation of sinus tracts and cicatrizion [Figure 2]
- Stage III: Diffuse and broad involvement across a regional area with multiple interconnected sinus tracts and abscess [Figure 3].

In 1989, Hurley proposed a severity classification. The Hurley classification is not quantitative and has its own limitations. It can be used to determine the severity but is not suitable for monitoring the efficacy of interventions in clinical trials.

Histology

Early lesions show follicular hyperkeratosis, plugging, and dilatation. The follicular epithelium may proliferate or get destroyed.

Eventually, perifolliculitis develops surrounded by extensive mixed infiltrate. Abscess formation leads to the destruction of pilosebaceous unit and later other cutaneous appendages.

The abscess extends deeper into the subcutaneous tissue forming draining sinus tracts which are lined by the epidermis.

### Table 1: Modified Sartorius Scoring/Modified Hidradenitis Suppurativa Score

| Characteristic of HS lesion | Point |
|----------------------------|-------|
| Anatomical region          |       |
| Any                        | 3     |
| None                       | 0     |
| Type of lesion             |       |
| Fistula                    | 4     |
| Nodule                     | 1     |
| Abscess                    | 1     |
| Scar                       | 1     |
| Other                      | 1     |
| Total area involved (cm)   |       |
| <5                         | 2     |
| 5-10                       | 4     |
| >10                        | 8     |
| Are all lesions totally separated by normal skin? | 0, 6 |
| Yes                        | 0     |
| No                         | 6     |
| Total                      | -     |

### Table 2: Hidradenitis Suppurativa Severity Index

| Score | Number of sites | BSA (%) | Number of lesions | Number of dressing changes | Pain (VAS) |
|-------|-----------------|---------|-------------------|---------------------------|------------|
| 0     | 0               | 0       | 0                 | 0                         | 0          |
| 1     | 1               | 1       | 1-2               | 1                         | 1          |
| 2     | 2               | 2-3     | 2-3               | 1                         | 2-4        |
| 3     | 3               | 4-5     | 4-5               | >1                        | 5-7        |
| 4+    | >4              | >5      | >5                | >5                        | 8-10       |

VAS: Visual analog scale, BSA: Body surface area
Healing occurs with extensive fibrosis.

**Risk Factors**

Smoking, obesity, female sex, lithium therapy, and genetic proneness to acne are some of the predisposing factors.

The risk of developing HS and the severity of the disease is 9.4 times higher in current smoker than in nonsmoker or ex-smokers. However, association of smoking has not been found to affect the course of the disease.\(^{(23)}\)

The risk of developing HS increases by 1.2 times for every unit increase in BMI and has been found to be more severe in obese than in underweight patients. Few studies have also shown higher prevalence of metabolic syndrome in HS patients.\(^{(13)}\) Not only that, mechanical stress occurring in axillae and groin due to continuous pressure and friction has also been considered as a contributing factor.

A positive family history has also been seen in more than 40% with autosomal dominant inheritance.\(^{(11)}\) Initial studies showed the involvement of chromosome 1p21.1–1q25.3 but has not been established.\(^{(25)}\)

CARD15 polymorphism, HLA-B, and HLA-DR type association have also been excluded.\(^{(26)}\)

Shaving of prone areas has been associated with earlier onset of the disease.

No association with cosmetics or oral contraceptive pills has been found.

**Comorbid Conditions**

HS has been shown to be associated with inflammatory bowel disease, spondyloarthropathy, epithelial tumors, and pyoderma gangrenosum.\(^{(27,28)}\)

Inflammatory bowel disease and spondyloarthropathy have been reported as the most frequently associated comorbid condition raising suspicion of shared pathogenesis and also that all three of them respond to tumor necrosis factor-\(\alpha\) (TNF-\(\alpha\)) inhibitor.\(^{(29)}\)

There is an increase in the formation of epithelial and nonmelanoma skin cancers in patients of HS almost by 50%. Patients with HS also show higher chances of developing buccal cancer, liver carcinoma, or squamous cell carcinoma.\(^{(10,31)}\)

HS shows association with pyoderma gangrenosum and has been shown to share common etiology involving cytokine dysregulation.

Few studies also show association with pityriasis rubra pilaris, acanthosis nigricans, and steatocystoma multiplex as they show common pathogenesis of follicular occlusion.

**Impact on Quality of Life**

The disease impairs severely the quality of life of patients due to its chronic, debilitating, painful nature, malodorous discharge, and healed fibrotic bands leading to restriction in mobility. The patient is often embarrassed due to the malodor and soaking of clothes.\(^{(32-34)}\) There is fall in self-worth, stigmatization leading to higher depression score than other dermatological disorders.

On an 11-point scale, in which 0 represents no pain and 10 represents the worst imaginable pain, patients of HS describe their pain in the range of 4–10 which is mostly

| Table 3: Hidradenitis Suppurativa Physician Global Assessment Score |
|-----------------------|---------|-------------------|
| Score | Ratings | Description |
| 0     | Clear   | No abscesses, no draining fistulas, no nodules |
| 1     | Minimal | No abscesses, no draining fistulas, no inflammatory nodules, presence of non-inflammatory nodules |
| 2     | Mild    | No abscess or draining fistulas and <5 inflammatory nodules OR Single abscess or draining fistula and no inflammatory nodules |
| 3     | Moderate| No abscess or draining fistulas and <5 inflammatory nodules OR Single abscess or draining fistula in the presence of inflammatory nodules OR Between 2 and 5 abscesses or draining fistulas with or without inflammatory nodules, up to 10 |
| 4     | Severe  | Between 2 and 5 abscesses or draining fistulas with or without inflammatory nodules that greater than 10 |
| 5     | Very severe | More than 5 abscesses or draining fistulas |

| Table 4: Acne Inversa Severity Index |
|-------------------------------------|
| AISI | If observed | Multiplied by overall number of sites where the lesions occur |
|------|-------------|--------------------------------------------------|
| Comedonic lesion | 1 point | |
| Abscess/inflammatory lesion | 2 points | |
| Sinus tract | 3 points | |
| Keloid, fibrotic adherence | 4 points | |
| Fibrosclerotic inflammatory plaque | 5 points | |
| Illness-VAS (pain discomfort-disability) | 0-10 | |
| Total | | |

AISI: Acne Inversa Severity Index, VAS: Visual analog scale
hot, burning, pressure, stretching, cutting, sharp, taut, splitting, gnawing, pressing sore, throbbing or aching.\[35\\]

The average visual analog scale (VAS) pain score is 4.2 and the mean dermatology life quality index ranges around 10 which indicates substantial disease-specific impairment of quality of life and is greater than other chronic dermatological disorders such as alopecia, acne, psoriasis, and vascular abnormalities of the face.\[32\\]

Karl Marx, the father of communism is considered to have suffered from HS. His thought process was majorly influenced by the incapacitating pain that came with the disease. Not only did it contribute to his poverty but also made him depressed and psychologically violent. Hence, it is considered by many that HS shaped the theory of communism in the 20th century (http://genevadermatology.ch/karl-marx-and-hidradenitis-suppurativa-verneuils-disease/).

**Complications**

The most common complication is anemia, rarely there is deposition of amyloid in the tissues.

If the early stages of the disease goes unrecognized in cases of perianal hidradenitis, it can extend up to anus and rectum leading to fistula formation and colitis.\[36\\]

In case of perigenital hidradenitis, it most likely distorts the external genitalia in both men and women.

In cases with deep penile lesions, there can be urethral fistula formation. Furthermore, in recalcitrant lesions, development of squamous cell carcinoma also has been reported.

**Differential Diagnosis**

In the early stages, HS can be mistaken for furuncles, infected sebaceous cysts, and epidermal cysts.

In later stages, with draining lesions, it has to be differentiated from scrofuloderma, lymphogranuloma venereum, actinomycosis, vegetating pyoderma gangrenosum, ulcerative colitis, and Crohn's disease.

Early diagnosis and treatment of HS is required to prevent the disease associated morbidities and to maintain the quality of life of the patient.

**Investigations**

Patient of HS needs to be thoroughly evaluated and a battery of investigations is required to be done which are as following:

1. Complete blood count
2. Erythrocyte sedimentation rate
3. C-reactive protein
4. Liver and renal function tests
5. Serum iron and ferritin
6. Fasting and postprandial blood sugar
7. Bacteriological analysis of the discharge
8. Body mass index calculation.

**Treatment**

The treatment modality of HS includes general measures, topical treatment, oral treatment, and surgery.

**General Measures**

The patients are advised to lose weight if they lie in the criteria of being overweight or obese. If they are smokers, cessation of the habit is advised. Patients are asked to wear lose fitting clothes to prevent friction and frequent changing to prevent soakage due to discharge and good personal hygiene has to be maintained; antibiotic soaps can be used to bath. The axillary hair should be clipped and not shaved and use of depilatories and deodorants is discouraged.

**Topical Treatment**

There are arrays of topical treatment available which are mostly used as an adjuvant or prophylactically.

During active inflammatory stage, warm normal saline compresses and Burow's solution (1:40) for 30 min have been shown to give relief from pain.

Frequent cleansing with povidone iodine or 5% benzoyl peroxide is followed by topical antibiotics.

Topical clindamycin (10 mg/ml) BD for 3 months or more has shown to be effective against anaerobic organisms. It can be used in milder cases of pediatric HS or as adjuvant with oral treatment.\[37\\]

Topical 15%–20% azelaic acid BD has been used in children with oral erythromycin or oral isotretinoin (0.7–1 mg/kg) with good results showing reduction and no recurrence.\[38\\]

Topical resorcinol 15% has been effective in reducing pain of the nodules assessed by VAS in Stage I and II HS in adults. It is not advisable to use it in children even in low concentration due to chances of resorcinol poisoning.\[39\\]

Adapalene (0.3%), silver sulfadiazine, intralesional triamcinolone (5 mg/ml), intramuscular human immunoglobulin, botulinum toxin A, and perilesional granulocyte-macrophage colony-stimulating factor have been used in different studies showing mixed efficacy, but further trials are required to prove their results.

Photodynamic therapy with aminolevulinic acid along with blue light therapy has been shown to be effective in one pilot study.\[40\\]

Cryotherapy can be used in cases with persistent and painful nodules, but patients have to be warned about the prolonged healing time, ulceration or risk of infections.\[41\\]
Carbon dioxide laser excision (10–15 W) followed by split skin grafting can be done.\(^{[42]}\)

Smooth beam laser (a 450 nm diode laser), Nd-YAG laser, Pulsed dye laser have been partially effective in one report.\(^{[43]}\)

EMLA should not be used in HS as it causes barrier deficiency and runs a chance of developing methemoglobinemia.

**Oral Treatment**

**Antibiotics**

The bacterial culture shows polymicrobial flora with up to 5 different species at least. The most common organism isolated is coagulase-negative staphylococci which is found in almost every 4\(^{th}\) subject mostly in the perineum and is responsible for slow, sub-acute evolution.\(^{[44-46]}\)

The second most common organism is *Staphylococcus aureus* with *Proteus mirabilis* which is responsible for follicular inflammation and necrosis. The high prevalence of *S. aureus* is also linked to smoking as nicotine favors its growth and colonization.

The third largest group of bacteria includes intestinal flora such as *Escherichia coli*, *Klebsiella* sp., *Enterococcus faecalis*, and *Pantoea agglomerans* seen slightly more in the perineum region.

In light of these findings, following few cocktail of oral wide spectrum antibiotics may be used.\(^{[47]}\)

Initial therapy should be started with penicillin with β-lactamase inhibitor (amoxicillin with clavulanic acid) or fluoroquinolones. The carbapenems have been found to be most effective but is not more commonly used due to parenteral administration.

As the second step, clindamycin 300 mg BD with rifampicin 300 mg BD for over 2–3 months has been shown to induce remission in 8 out of 14 patients.

As the third step, combination of rifampicin 600 mg with moxifloxacin and metronidazole can be used.

Tetracycline 500 mg BD has been found to be almost completely useless in advance cases, but it can be used for long-term maintenance therapy.

**Corticosteroids**

In severe flares, oral prednisolone (0.5 to 1 mg/kg) is given daily for 10–14 days and is gradually reduced as inflammation reduces.\(^{[48]}\)

For local and recalcitrant lesions, intraleosional triamcinolone acetonide 40 mg/ml can be given.

**Oral Retinoids**

About 50% of adult patients show partial response to isotretinoin therapy (high doses of 2 mg/kg body weight/day) if used for 1 year or longer. It can also be used before surgical procedures are to be undertaken to reduce the staging of the disease.\(^{[49,50]}\)

In children, the dosage of isotretinoin is 0.7–1 mg/kg body weight/day and has been effectively used with 15% azelaic acid.
If patients are not responding to isotretinoin, acitretin can be used which has been found to be more effective.[51]

**Antiandrogens**

Occasionally few female patients with underlying PCOS have been reported to respond to cyproterone acetate (50 mg/day) alone or when given in combination with ethinylestradiol (50 mcg).[52] Spironolactone and finasteride (5 mg/d) have also led to a significant response in a few patients.[53]

**Metformin**

As hyperinsulinemia is a fundamental disturbance in PCOS, use of insulin-sensitizing drugs such as metformin was introduced. It improves glucose utilization and also has antiandrogenic effects and is now used as first-line treatment of PCOS. It induces good remission of HS with minimal side effects.[54]

**Zinc**

Zinc gluconate 90 mg/day has been used in a pilot study and has shown partial and complete remission in patients not responding to other modalities, with minor gastrointestinal side effects.[55]

**Cyclosporine**

Cyclosporine (6 mg/kg/day) given for over 5–30 weeks has been found to be effective in patients not responding to antibiotics.[56]

**Dapsone**

Dapsone 100 mg/day has been shown to help occasionally.

**Biologics**

Due to the limitations of the above methods, biologics have emerged as a useful tool in treating Hurley stage II and III or recalcitrant HS.

Pro-inflammatory cytokines IL-1β, TNF-α, and IL-10, IL-12, IL-23 are significantly elevated in lesional HS skin and correlate with the severity of the disease. The following biologics have been used in HS.

Adalimumab: It is an anti-TNF-α monoclonal antibody used in the treatment of moderate-to-severe HS and refractory HS. It is administered in the dose of 40 mg subcutaneous injection every other week or every week.[57,58]

Infliximab: It is an anti-TNF-α chimeric monoclonal antibody. The dosage is 5mg/kg given at 0, 2, 6 weeks and have shown improvement even in recalcitrant HS. The fourth infusion in 10th week can be given if required.[59,60]

Anakinra: It is an IL-1 receptor antagonist which can be given in the dose of 100 mg daily subcutaneously for 12 weeks. It has been used for Grade II and III HS with mixed response.[51]

Efalizumab: It is a recombinant humanized monoclonal antibody which binds to the CD11a subunit of lymphocyte function-associated antigen 1. Its dosage is 1 mg/kg/week for 3 months subcutaneously. It was associated with fatal brain infections and was withdrawn from the market in 2009.[62]

Ustekinumab: It is an IL-12/23 inhibitor which can be used as 3–45 mg subcutaneous injections at 0, 1, and 4 months in recalcitrant HS.[63]

**Surgical Treatment**

Incision and drainage of the abscesses and fistulas are usually futile. However, when the medical therapies are rendered indolent or when recurrence occurs, operative excision remains as the only curative modality. Wide local excision with skin grafting, skin flap transfer, and primary closure are commonly done. However, fasciocutaneous or musculocutaneous flaps show lesser recurrence rate. Reconstruction of the defects with flaps may prevent contractures and bad scarring, but local flaps might possess the risk of carrying the same affected skin and lead to recurrences. Skin grafts may lead to contractures and extensive scarring but is acceptable in the gluteal or groin regions as they can be covered easily and it does not cause motion restriction. Another method used is the deroofing technique which maximizes preservation of surrounding healthy tissue and is associated with cosmetically acceptable scars.[64] Granulocyte-macrophage colony-stimulating factor injections given with surgical procedure has been reported to give excellent results. Along with the above, liposuction of the affected areas has emerged as a new modality of treatment in young patients. Only one area is treated at a time and recurrences can occur as it does not remove all the tissue.

**Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

**Financial support and sponsorship**

Nil.

**Conflicts of interest**

There are no conflicts of interest.

---

### What is new?

The advent of biologicals has improved the quality of life of patients with HS.

### References

1. Velpeau A. Aissele. In: Bechet Jeune Z, editor. Dictionnaire de
2. Verneuil AS. Studies on skin tumors; Some diseases of the sweat glands. Arch Med Gen (in French) 1854;94:693-705.

3. Pillsbury DM, Shelley WB, Klüggmann AM, editors. Bacterial infections of the skin. In: Dermatol. 1st ed. Philadelphia: Saunders WB; 1956. p. 482-4.

4. Gerd P, Albert MK, editors. Acne conglobata. In: Acne: Morphogenesis and Treatment. Berlin: Springer-Verlag; 1975. p. 168-203.

5. HS-USA: What is Hidradenitis Suppurativa?. Archived from the original; 17 June, 2013. [Last retrieved on 2013 Jul 20].

6. Jemec GB, Heidenheim M, Nielsen NH. The prevalence of hidradenitis suppurativa and its potential precursor lesions. J Am Acad Dermatol 1996;35:191-4.

7. von der Werth JM, Williams HC. The natural history of hidradenitis suppurativa. J Eur Acad Dermatol Venereol 2000;14:389-92.

8. Canoui-Poitrine F, Revuz JE, Wolkenstein P, Viallette C, Gabison G, Pouget F, et al. Clinical characteristics of a series of 302 French patients with Hidradenitis Suppurativa, with an analysis of factors associated with disease severity. J Am Acad Dermatol 2009;61:51-7.

9. Jemec GB, Heidenheim M, Nielsen NH. Hidradenitis suppurativa – Characteristics and consequences. Clin Exp Dermatol 2009;34:388-94.

10. Jemec GB. Clinical practice. Hidradenitis suppurativa. N Engl J Med 2012;366:158-64.

11. van der Zee HH, Laman JD, Boer J, Prens EP. Hidradenitis suppurativa: Viewpoint on clinical phenotyping, pathogenesis and novel treatments. Exp Dermatol 2012;21:735-9.

12. von der Werth JM, Williams HC. The natural history of hidradenitis suppurativa. J Eur Acad Dermatol Venereol 2000;14:389-92.

13. König A, Lehmann C, Rompel R, Happle R. Cigarette smoking as a triggering factor of hidradenitis suppurativa. Dermatology 1999;198:261-4.

14. Kutzner H, Wurzel RM, Wolff HH. Are acrosyringia involved in the pathogenesis of hidradenitis suppurativa (acne inversa)? Dermatol Morphogenesis and Treatment. Berlin: Springer-Verlag; 1975. p. 168-203.

15. Jemec GB, Heidenheim M, Nielsen NH. Hidradenitis suppurativa – Characteristics and consequences. Clin Exp Dermatol 1996;21:419-23.

16. Wolkenstein P. Quality of life in hidradenitis suppurativa. In: Jemec GB, Revuz J, Leyden J, editors. Hidradenitis Suppurativa. Berlin, Germany: Springer; 2006. p. 116-9.

17. Sartorius K, Emtestam L, Jemec GB, Lapins J. Objective scoring of hidradenitis suppurativa reflecting the role of tobacco smoking and obesity. Br J Dermatol 2009;161:831-9.

18. Jemec GB, Heidenheim M, Nielsen NH. Hidradenitis suppurativa – Characteristics and consequences. Clin Exp Dermatol 1996;21:419-23.

19. Wolkenstein P, Loundou A, Barrau K, Auquier P, Revuz J. Quality of Life Group of the French Society of Dermatology, et al. Quality of life impairment in hidradenitis suppurativa: A study of 61 cases. J Am Acad Dermatol 2007;56:621-3.

20. Smith HS, Chao JD, Teitelbaum J. Painful hidradenitis suppurativa. Clin J Pain 2010;26:435-44.

21. Kutzner H, Wurzel RM, Wolff HH. Are acrosyringia involved in the pathogenesis of “dyshidrosis”? Am J Dermatopathol 1986;8:109-16.

22. Jemec GB, Wendelboe P. Topical clindamycin versus systemic tetracycline in the treatment of hidradenitis suppurativa. J Am Acad Dermatol 1998;39:971-4.

23. Stojkovic-Filipovic JM, Gagic-Veljis MD, Nikolic M. Prepubertal onset of hidradenitis suppurativa in a girl: A case report and literature review. Indian J Dermatol Venereol Leprol 2015;81:294-8.

24. Jemec GB, Resorcinol peels as a possible self-treatment of painful nodules in hidradenitis suppurativa. Clin Exp Dermatol 2010;35:36-40.

25. Strauss RM, Pollock B, Stables GI, Goulden V, Cunliffe WJ.
Photodynamic therapy using aminolaevulinic acid does not lead to clinical improvement in hidradenitis suppurativa. Br J Dermatol 2005;152:803-4.

41. Bong JL, Shalders K, Saikian E. Treatment of persistent painful nodules of hidradenitis suppurativa with cryotherapy. Clin Exp Dermatol 2003;28:241-4.

42. Lapins J, Marcusson JA, Emtestam L. Surgical treatment of chronic hidradenitis suppurativa: CO2 laser stripping-secondary intention technique. Br J Dermatol 1994;131:551-6.

43. Downs A. Smoothbeam laser treatment may help improve hidradenitis suppurativa but not hailey-hailey disease. J Cosmet Laser Ther 2004;6:163-4.

44. Sartorius K, Kiliasl H, Oprica C, Sullivan A, Lapins J. Bacteriology of hidradenitis suppurativa exacerbations and deep tissue cultures obtained during carbon dioxide laser treatment. Br J Dermatol 2012;166:879-83.

53. Farrell AM, Randall VA, Vafaei T, Dawber RP. Finasteride as a therapy for hidradenitis suppurativa. Br J Dermatol 1999;133:371-6.

56. Gupta AK, Ellis CN, Nickoloff BJ, Goldfarb MT, Ho VC, Rocher LL, et al. Oral cyclosporine in the treatment of inflammatory and noninflammatory dermatoses. A clinical and immunopathologic analysis. Arch Dermatol 1990;126:339-50.

57. Blanco R, Martínez-Taboada VM, Villa I, González-Vela MC, Fernández-Llaca H, Agudo M, et al. Long-term successful adalimumab therapy in severe hidradenitis suppurativa. Arch Dermatol 2009;145:580-4.

58. Ravat F, O'Reily D, Handfield-Jones S. Recalcitrant hidradenitis suppurativa treated with adalimumab. J Am Acad Dermatol 2005;52:569.

60. Sullivan TP, Welsh E, Kerdel FA, Burdick AE, Kirsner RS. Infliximab for hidradenitis suppurativa. Br J Dermatol 2003;149:1046-9.

62. Tzanetakou V, Kanni T, Giatrakou S, Katoulis A, Papadavid E, Netea MG, et al. Safety and efficacy of anakinra in severe hidradenitis suppurativa: A Randomized clinical trial. JAMA Dermatol 2016;152:52-9.

63. Gulliver WP, Jemec GB, Baker KA. Experience with ustekinumab for the treatment of moderate to severe hidradenitis suppurativa. J Eur Acad Dermatol Venereol 2012;26:911-4.