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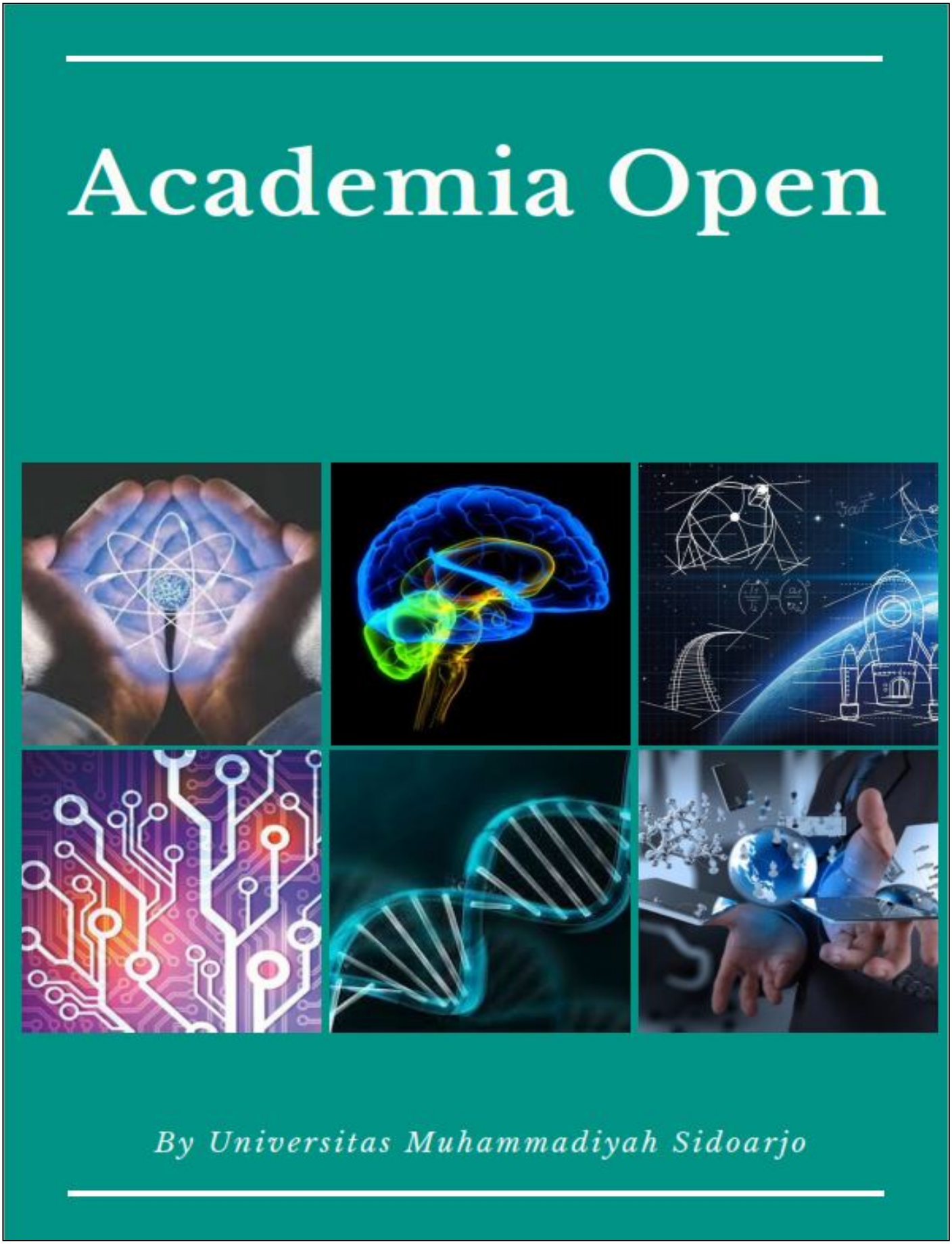

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A Review of Hidradenitis Suppurativa (HS)

Tinjauan tentang Hidradenitis Suppurativa (HS)

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Abstract

Background: Hidradenitis suppurativa (HS) is a chronic, inflammatory skin disease of the hair follicle, affecting intertriginous areas such as the axillae, perianal, and inguinal regions. It is more prevalent in females, with peak onset between ages 18 and 29. **Specific Background:** HS, characterized by nodules, abscesses, and sinus tract scarring, uses the Hurley staging system for disease severity classification, but its limitations exist due to its static clinical features. **Knowledge Gap:** Although several treatment modalities exist, ranging from antibiotics to surgery, there is no definitive therapy, and the pathogenesis remains poorly understood, complicating early diagnosis and management. Moreover, international diagnostic delays of 8-12 years further hinder patient outcomes. **Aims:** This study aims to explore the limitations of current classification and treatment approaches, while emphasizing the importance of prevention strategies and timely diagnosis to mitigate HS severity. **Results:** The Hurley classification, though widely used, fails to account for disease progression or quality of life. Current treatment options depend on disease severity, including topical and systemic antibiotics, hormonal therapies, biologics, and surgical interventions. Prevention strategies, such as laser hair removal and lifestyle modifications (e.g., smoking cessation, weight management), are essential to reducing disease impact. **Novelty:** This research highlights the under-explored role of early prevention and lifestyle interventions in managing HS and reducing progression through Hurley stages. **Implications:** Enhanced diagnostic tools, combined with early intervention strategies and a holistic approach to patient care, are necessary to improve clinical outcomes, reduce diagnostic delays, and minimize HS-related morbidity. Further research is needed to better understand the genetic and environmental factors contributing to HS pathogenesis.

Highlights:

1. HS peaks in females aged 18-29 with chronic skin inflammation.
2. 8-12 years diagnostic delay worsens disease severity and treatment.
3. Prevention: laser hair removal, lifestyle changes reduce HS progression.

Keywords: Hidradenitis suppurativa, Hurley staging, inflammatory skin disease, prevention, diagnostic delays

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Introduction

Hidradenitis suppurativa from the Greek hidros means sweat and Aden means glands [1]. Is a chronic, inflammatory skin disease of the hair follicle characterized by the improvement of frequent inflammatory nodules, abscesses, sinus tracts and scarring, involving the intertriginous regions, such as axillary, perianal and inguinal areas [2]. This disease known as a as acne inversa, and it is debilitating inflammatory skin disorder [3,4]. The retard in diagnosis of this disease can be 12 years or longer [5]. Rapid recognition and start of treatment can decrease the risk of HS progression to debilitating end-stage disease. Because of the associated pain, malodorous dis¬charge, and scarring that makes the psychosocial effect of HS is overwhelming. HS is associated with greater impair¬ment of quality of life and professional action than other chronic skin conditions such as psoriasis [6,7]. The real mechanism for pathogenesis of HS is unknown but believed to be a combination of environmental and genetic factors [8,9] with resulting immune dysregulation, follicular obstruction inflammation and secondary infection and tissue destruction [10]. HS divides into three phenotypes according to distribution of lesions: axillary-mammary, follicular, and gluteal. The axillary-mammary phenotype is found in approximately half of HS cases, predominantly in young women, is in the breast region and underarm and characterized by hypertrophic scarring [11].

The follicular form (occurring mainly in men) is often involving the ears, legs, chest, and back as well as the breast and axilla. This type also shows the highest chance for finding follicular lesions on histopathology among the three HS subtypes. The gluteal subtype presents in folliculitis in the gluteal areas and follicular papules. This phenotype is most related to smoking and low body mass index [11].

Females are more likely to develop HS than males. A study in the United States found that the frequency of HS was twice as high among females than among males; the highest age-specific prevalence was among patients between the ages of 18 and 29 years. The beginning of disease symptoms mostly occurs between puberty and age 40, topping in the second or third decade of life [12]. The higher incidence of this disease was also detected in patients with spondylarthritis, and the onset of HS symptoms was linked with increased (SpA activity) [13, 14].

Methods

Clinical Staging of HS

The most generally used system is the Hurley staging classification system [15]. The Hurley system is used to divide patients with HS into three disease severity stages:

Stage I: abscesses creation (single or multiple) without sinus tracts and scarring. This stage is the most common, affecting 68% of patients.

Stage II: Recurrent abscesses with sinus tracts and scarring, (single or multiple) broadly separated lesions. Stage II occurs in 28% of patients.

Stage III: Diffuse or near-diffuse involvement, (multiple interconnected tracts and abscesses across the whole area), and stage III occurs in 4% [16].

The Hurley classification is suitable for quick classification of HS severity, but it has serious limitations [16]. This staging system has three categories only, based on static disease features, and only accounts for the clinical presentation, other more progressive systems exist to categorize treatment efficiency and quality of life deficiency [15,16].

Prevention

Prevention is main for patients with HS; secondary prevention targets to reduce influence of the disease or injury that has already occurred [17,18], which includes prevention of the infundibulofolliculitis from becoming a deep folliculitis, nodule, as well as hurley stage development. Suitable treatment with quick diagnosis can reduce the severity of pain, lesions, and scarring. Internationally, HS patients continue to experience considerable diagnostic delays of (eight to twelve years) after beginning of initial symptoms [18,19]. Patients should avoid known causes, including stress, smoking, obesity, mechanical irritation, sweating, and poor hygiene [18]. Hair shaving creates ingrown hair shafts, which may lead to folliculitis in mechanically stressed areas in skin folds, thus initiating the inflammatory force of HS [18,20]. Thus, shaving along with any other mechanical stress must be avoided in patients with HS. Hair removing by laser is quite useful in both the prevention and treatment of HS [18].

Result and Discussion

Diagnosis

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Research on HS shows that a subclinical inflammatory state precedes the first visible marks, such as hyperkeratosis of the infundibulum, resulting in follicular plugging [21-24]. A histopathological study of early HS lesions exposed perifollicular inflammation and infundibulofolliculitis as the primary events in HS [25]. HS remains diagnostically challeng-ing. It is often misdiagnosed as sores or furunculosis on initial presentation and can take twelve years or more from onset of symptoms to HS diagnosis [5].

The three important clinical features that support a diag¬nosis of HS are typical lesions, typical locations, and relapses and chronicity [3,28]. Added elements to be elicited on history involves family history of HS, initial onset of symptoms, and gastroin¬testinal symptoms given the connotation with Crohn dis¬ease [27]. Physical checkup should measure the severity and extent of HS and characterize the lesions. The Hurley staging system is com¬monly used for evaluating HS severity [6,28,29].

The initial diagnosis might include measuring complete blood count, inflammatory markers and routine chemistries. In confident cases, suspected perianal HS should be biopsied to rule out malignancy and Crohn disease. Skin biopsy is rarely indicated unless the diagnosis is unclear or there is a need to rule out other disorders, especially lesions suggestive of squamous cell carcinoma. Wound cultures can be considered in lesions with indication of secondary infection [3, 28].

Treatment

Currently no therapy for HS, available treatment options depend on the severity of the disease [30]. The current treatment modalities include topical and systemic antibiotics, hormonal therapy, anti-inflammatory agents, biologics, and surgical interferences, such as marsupialization and excision of the affected skin. Not every patient is a candidate for surgical interventions, which have been revealed to significantly improve quality of life, and the efficiency of systemic therapies is limited in a significant population of patients, leading to a need for a better knowledge of the underlying pathophysiology of this disease [31-33].

Discussion

The pathophysiology oh HS is thought to be started by blockage of hair follicles, followed by follicle rupture and a subsequent inflammatory cascade [34]. A cooperated skin barrier may also play a role in HS, as an damaged epidermis cannot function correctly. Notch signaling plays a role in epidermal immunity and inflammation. Impaired notch signaling may interrupt apocrine gland homeostasis, affecting the skin barrier and potentially motivating inflammatory mediators [35].

It is important considered when dealing with complications of HS. The initial workup might include measuring complete blood count, routine chemistries, and inflammatory markers. In some cases, suspected perianal HS should be biopsied to rule out malignancy and Crohn disease. Skin biopsy is rarely showed unless the diagnosis is uncertain or one needs to rule out other disorders, especially lesions suggestive of squamous cell carcinoma. Wound cultures can be considered in lesions with substantiation of secondary infection [2, 28]. The most effective systemic therapies employed for HS is biologics and immunomodulators. Adalimumab and infliximab are biologics that have been shown to improve skin symptoms significantly [36]. When clindamycin , rifampicin combination therapy, and adalimumab were used to treat patients with HS, all three treatments decreased the mean modified Sartorius score [37,38].

Numerous treatment modalities have been recommended for HS including medical and surgical interferences. In mild to moderate HS, topical and systemic antibiotics including rifampicin, clindamycin and tetracycline tend to be the first line treatments [39]. The basis for antibiotic therapy in HS is based on the indication that bacteria is linked to the pathogenesis of the disease. The skin microbiome of HS patients with and without lesions, have demonstrated the presence of Staphyloccus lugdunesis, polymorphous anaerobic flora and skin commensals, including prophyromonas and poptoniphilus species, as the most commonly observed bacteria [2,40]. Antibiotic combinations were developed on a basis of prevention of resistance seen with monotherapy and in addition to their anti-inflammatory effects their extensive coverage for this broad spectrum of bacteria present in HS lesions [41,42].

The treatment of HS remains a serious worry for physicians. Several studies are investigating effective and tolerable treatment options for HS patients to advance HS management and reduce the risk of comorbidities. These options include local excision, deroofing and curettage, laser ablation (CO2), laser hair removal, and even radiation [43].

Conclusion

HS Is a chronic, inflammatory skin disease associated with the quality of life. The quite mechanism for pathogenesis of HS is unknown but believed to be a combination of genetic and environmental factors. HS remains diagnostically challeng¬ing. It is often misdiagnosed as sores or furunculosis on initial presentation and can take twelve years or more from onset of symptoms to HS diagnosis. Females are more likely to develop HS than males. Patients with HS should avoid known causes, including stress, smoking, obesity, mechanical irritation, sweating, and poor hygiene. Full encology of this disease requires numerous researches and in vitro studies.

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References

1. . C. Elisabete and B. Helena, "Managing Hidradenitis Suppurativa," 2019. DOI: 10.36959/446/38.
2. . H. C. Ring et al., "The Follicular Skin Microbiome in Patients With Hidradenitis Suppurativa and Healthy Controls," JAMA Dermatology, vol. 153, no. 9, p. 897, Sep. 2017, doi: 10.1001/jamadermatol.2017.0904.
3. . G. B. E. Jemec, "Hidradenitis Suppurativa," New England Journal of Medicine, vol. 366, no. 2, pp. 158–164, Jan. 2012, doi: 10.1056/nejmcp1014163.
4. . K. Sellheyer and D. Krahl, "'Hidradenitis Suppurativa' is Acne Inversa! An Appeal to (Finally) Abandon a Misnomer," International Journal of Dermatology, vol. 44, no. 7, pp. 535–540, Dec. 2004, doi: 10.1111/j.1365-4632.2004.02536.x.
5. . A. Mebazaa and R. Ben Hadid, "Hidradenitis Suppurativa: A Disease With Male Predominance in Tunisia," PubMed, Dec. 2009. [Online]. Available: https://pubmed.ncbi.nlm.nih.gov/20043054/.
6. . A. Alikhan, P. J. Lynch, and D. B. Eisen, "Hidradenitis Suppurativa: A Comprehensive Review," Journal of the American Academy of Dermatology, vol. 60, no. 4, pp. 539–561, Apr. 2009, doi: 10.1016/j.jaad.2008.11.911.
7. . Ł. Matusiak, A. Bieniek, and J. C. Szepietowski, "Hidradenitis Suppurativa Markedly Decreases Quality of Life and Professional Activity," Journal of the American Academy of Dermatology, vol. 62, no. 4, pp. 706-708.e1, Apr. 2010, doi: 10.1016/j.jaad.2009.09.021.
8. . M. Napolitano et al., "Hidradenitis Suppurativa: From Pathogenesis to Diagnosis and Treatment," Clinical Cosmetic and Investigational Dermatology, vol. 10, pp. 105–115, Apr. 2017, doi: 10.2147/ccid.s111019.
9. . L. R. Paus et al., "What Causes Hidradenitis Suppurativa?," Experimental Dermatology, vol. 17, no. 5, pp. 455–456, May 2008, doi: 10.1111/j.1600-0625.2008.00712_1.x.
10. . E. Prens and I. Deckers, "Pathophysiology of Hidradenitis Suppurativa: An Update," Journal of the American Academy of Dermatology, vol. 73, no. 5, pp. S8–S11, Nov. 2015, doi: 10.1016/j.jaad.2015.07.045.
11. . F. Canoui-Poitrine et al., "Identification of Three Hidradenitis Suppurativa Phenotypes: Latent Class Analysis of a Cross-Sectional Study," Journal of Investigative Dermatology, vol. 133, no. 6, pp. 1506–1511, Jun. 2013, doi: 10.1038/jid.2012.472.
12. . A. Garg, J. Lavian, G. Lin, A. Strunk, and A. Alloo, "Incidence of Hidradenitis Suppurativa in the United States: A Sex- and Age-Adjusted Population Analysis," Journal of the American Academy of Dermatology, vol. 77, no. 1, pp. 118–122, Jul. 2017, doi: 10.1016/j.jaad.2017.02.005.
13. . K. Kridin, E. Shavit, G. Damiani, and A. D. Cohen, "Hidradenitis Suppurativa and Rheumatoid Arthritis: Evaluating the Bidirectional Association," Immunologic Research, vol. 69, no. 6, pp. 533–540, Aug. 2021, doi: 10.1007/s12026-021-09221-4.
14. . A. Rondags, S. Arends, F. R. Wink, B. Horváth, and A. Spoorenberg, "High Prevalence of Hidradenitis Suppurativa Symptoms in Axial Spondyloarthritis Patients: A Possible New Extra-Articular Manifestation," Seminars in Arthritis and Rheumatism, vol. 48, no. 4, pp. 611–617, Feb. 2019, doi: 10.1016/j.semarthrit.2018.03.010.
15. . Y. Yao and S. F. Thomsen, "The Role of Interleukin-17 in the Pathogenesis of Hidradenitis Suppurativa," PubMed, Jul. 2017. [Online]. Available: https://pubmed.ncbi.nlm.nih.gov/29469692/.
16. . H. H. Van Der Zee and G. B. E. Jemec, "New Insights Into the Diagnosis of Hidradenitis Suppurativa: Clinical Presentations and Phenotypes," Journal of the American Academy of Dermatology, vol. 73, no. 5, pp. S23–S26, Nov. 2015, doi: 10.1016/j.jaad.2015.07.047.
17. . C. C. Zouboulis, Q. Ju, and L.-H. F. Xiang, "Editorial — The 3rd International Conference on Sebaceous Gland, Acne, Rosacea and Related Disorders," Dermato-Endocrinology, vol. 9, no. 1, p. e1386419, Jan. 2017, doi: 10.1080/19381980.2017.1386419.
18. . H. Kurzen and M. Kurzen, "Secondary Prevention of Hidradenitis Suppurativa," Dermatology Reports, vol. 11, no. 2, Oct. 2019, doi: 10.4081/dr.2019.8243.
19. . R. Sabat et al., "Acne Inversa/Hidradenitis Suppurativa: Ein Update," Der Hautarzt, vol. 68, no. 12, pp. 999–1006, Nov. 2017, doi: 10.1007/s00105-017-4082-5.
20. . J. Boer, M. Nazary, and P. T. Riis, "The Role of Mechanical Stress in Hidradenitis Suppurativa," Dermatologic Clinics, vol. 34, no. 1, pp. 37–43, Jan. 2016, doi: 10.1016/j.det.2015.08.011.
21. . G. Kelly, C. M. Sweeney, A. Tobin, and B. Kirby, "Hidradenitis Suppurativa: The Role of Immune Dysregulation," International Journal of Dermatology, vol. 53, no. 10, pp. 1186–1196, Jun. 2014, doi: 10.1111/ijd.12550.
22. . E. Prens and I. Deckers, "Pathophysiology of Hidradenitis Suppurativa: An Update," Journal of the American Academy of Dermatology, vol. 73, no. 5, pp. S8–S11, Nov. 2015, doi: 10.1016/j.jaad.2015.07.045.
23. . G. Kelly et al., "Dysregulated Cytokine Expression in Lesional and Nonlesional Skin in Hidradenitis Suppurativa," British Journal of Dermatology, vol. 173, no. 6, pp. 1431–1439, Nov. 2015, doi: 10.1111/bjd.14075.
24. . J. Molnar et al., "Hidradenitis Suppurativa and 1-Carbon Metabolism: Role of Gut Microbiome, Matrix Metalloproteinases, and Hyperhomocysteinemia," Frontiers in Immunology, vol. 11, Aug. 2020, doi: 10.3389/fimmu.2020.01730.
25. . J. Boer and G. B. E. Jemec, "Mechanical Forces and Hidradenitis Suppurativa," Experimental Dermatology, vol. 30, no. 2, pp. 212–215, Nov. 2020, doi: 10.1111/exd.14234.
26. . Here’s your reference list formatted in IEEE style, with titles and journal names in Title Case, and non-Latin letters converted to Latin letters. I’ve also completed any missing details based on available information.

27. . F. M. S. Al-Ali, D. M. Al-Jabri, M. D. Al-Balushi, and H. A. Al-Mamari, “Hidradenitis Suppurativa (Or Acne Inversa) With Autosomal Dominant Inheritance Is Not Linked to Chromosome 1p21.1-1q25.3 Region,” Exp. Dermatol., vol. 19, no. 9, pp. 851–853, Aug. 2010, doi: 10.1111/j.1600-0625.2010.01088.x.
28. . H. H. Zee, K. Winter, C. J. Woude, and E. P. Prens, “The Prevalence of Hidradenitis Suppurativa in 1093 Patients With Inflammatory Bowel Disease,” Br. J. Dermatol., vol. 171, no. 3, pp. 673–675, Jul. 2014, doi: 10.1111/bjd.13002.
29. . C. C. Zouboulis et al., “European S1 Guideline for the Treatment of Hidradenitis Suppurativa/Acne Inversa,” J. Eur. Acad. Dermatol. Venereol., vol. 29, no. 4, pp. 619–644, Jan. 2015, doi: 10.1111/jdv.12966.
30. . R. Alhusayen and N. H. Shear, “Pharmacologic Interventions for Hidradenitis Suppurativa,” Am. J. Clin. Dermatol., vol. 13, no. 5, pp. 283–291, Oct. 2012, doi: 10.2165/11631880-000000000-00000.
31. . T. Shih, K. Lee, J. R. Seivright, D. R. De, V. Y. Shi, and J. L. Hsiao, “Hyperhidrosis Treatments in Hidradenitis Suppurativa: A Systematic Review,” Dermatol. Ther., vol. 35, no. 1, pp. 1–10, Nov. 2021, doi: 10.1111/dth.15210.
32. . S. S. Jafari, E. Knüsel, S. Cazzaniga, and R. Hunger, “A Retrospective Cohort Study on Patients With Hidradenitis Suppurativa,” J. Am. Acad. Dermatol., vol. 78, no. 1, pp. 25–29, Jan. 2018.
33. . S. M. S. Jafari, R. E. Hunger, and C. Schlapbach, “Hidradenitis Suppurativa: Current Understanding of Pathogenic Mechanisms and Suggestion for Treatment Algorithm,” Front. Med., vol. 7, p. 68, Mar. 2020, doi: 10.3389/fmed.2020.00068.
34. . M. Gierek, D. Kitala, W. Łabuś, K. Szyluk, P. Niemiec, and G. Ochała-Gierek, “Impact of Hidradenitis Suppurativa Surgical Treatment on Health-Related Life Quality,” J. Clin. Med., vol. 11, no. 15, p. 4327, Jul. 2022, doi: 10.3390/jcm11154327.
35. . A. S. L. E. de Oliveira, G. Bloise, G. Moltrasio, C. Coelho, A. Agrelli, A. Moura, R. Tricarico, P. M. Jamain, S. Marzano, A. V. Crovella, and S. et al., “Transcriptome Meta-Analysis Confirms the Hidradenitis Suppurativa Pathogenic Triad: Upregulated Inflammation, Altered Epithelial Organization, and Dysregulated Metabolic Signaling,” Biomolecules, vol. 12, no. 1371, 2022, doi: 10.3390/biom12101371.
36. . N. Tanaka, A. Fujioka, S. Tajima, A. Ishibashi, S. Hirose, “Elafin Is Induced in Epidermis in Skin Disorders With Dermal Neutrophilic Infiltration: Interleukin-1 Beta and Tumour Necrosis Factor-Alpha Stimulate Its Secretion In Vitro,” Br. J. Dermatol., vol. 143, pp. 728–732, 2000, doi: 10.1046/j.1365-2133.2000.03802.x.
37. . A. Morita, H. Takahashi, K. Ozawa, S. Imafuku, N. Takekuni, K. Takahashi, T. Matsuyama, Y. Okubo, Y. Zhao, S. Kitamura, and et al., “Long-Term Analysis of Adalimumab in Japanese Patients With Moderate to Severe Hidradenitis Suppurativa: Open-Label Phase 3 Results,” J. Dermatol., vol. 48, no. 1, pp. 3–13, 2021, doi: 10.1111/1346-8138.15530.
38. . C. Marasca, M. C. Annunziata, A. Villani, S. Volpe, D. Marasca, and G. Fabbrocini, “Adalimumab Versus Rifampicin Plus Clindamycin for the Treatment of Moderate to Severe Hidradenitis Suppurativa: A Retrospective Study,” J. Drugs Dermatol., vol. 18, no. 4, pp. 437–438, 2019.
39. . O. Join-Lambert, F. Ribadeau-Dumas, V. Jullien, M.-D. Kitzis, J.-P. Jais, H. Coignard-Biehler, H. Guet-Revillet, P.-H. Consigny, M. Delage, M. Nassif, and et al., “Dramatic Reduction of Clindamycin Plasma Concentration in Hidradenitis Suppurativa Patients Treated With the Rifampin-Clindamycin Combination,” Eur. J. Dermatol., vol. 24, no. 1, pp. 94–95, Jan. 2014.
40. . D. M. L. Saunte and G. B. E. Jemec, “Hidradenitis Suppurativa: Advances in Diagnosis and Treatment,” JAMA, vol. 318, no. 20, pp. 2019–2032, Nov. 2017, doi: 10.1001/jama.2017.18282.
41. . H. Guet-Revillet, J. P. Jais, M. N. Ungeheuer, H. Coignard-Biehler, S. Duchatelet, M. Delage, and et al., “The Microbiological Landscape of Anaerobic Infections in Hidradenitis Suppurativa: A Prospective Metagenomic Study,” Clin. Infect. Dis., vol. 65, no. 2, pp. 282–291, Jul. 2017, doi: 10.1093/cid/cix296.
42. . S. Pradhan, B. Madke, P. Kabra, and A. L. Singh, “Anti-Inflammatory and Immunomodulatory Effects of Antibiotics and Their Use in Dermatology,” Indian J. Dermatol., vol. 61, no. 5, pp. 469–481, Oct. 2016, doi: 10.4103/0019-5154.190972.
43. . C. Marasca, P. Tranchini, V. Marino, M. C. Annunziata, M. Napolitano, D. Fattore, and et al., “The Pharmacology of Antibiotic Therapy in Hidradenitis Suppurativa,” Expert Rev. Clin. Pharmacol., vol. 13, no. 5, pp. 521–530, May 2020, doi: 10.1080/17512433.2020.1766785.
44. . M. J. Diaz, S. Aflatooni, P. Abdi, R. Li, M. R. Anthony, S. Neelam, C. Farkouh, J. T. Tran, S. Svoboda, M. Forouzandeh, and et al., “Hidradenitis Suppurativa: Molecular Etiology, Pathophysiology, and Management—A Systematic Review,” Curr. Issues Mol. Biol., vol. 45, pp. 4400–4415, 2023, doi: 10.3390/cimb45050280.

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