Biomedical Dermatology

RESEARCH Open Access

Hidradenitis suppurativa and acne vulgaris and conglobata—systematic review and meta-analysis

Kevin Phan[1,2*], Olivia Charlton[3] and Saxon D. Smith[3,4,5]

Abstract

Background: Hidradenitis suppurativa (HS) is a chronic inflammatory skin disorder which involves painful nodules and draining abscesses in flexural areas. Acne vulgaris and its more severe variants including acne conglobata and acne fulminans are also disorders involving the follicular unit. Given that follicular obstruction, dilatation and inflammation feature in both HS and acne vulgaris/conglobata, it has been suggested that HS is associated with acne vulgaris/conglobata.

Methods: The present systematic review and meta-analysis was performed according to recommended PRISMA guidelines. All eligible case-control studies comparing patients with HS vs non-HS were included in the present review. All studies must have included either the proportion of patients with acne vulgaris/conglobata in each group, or the summary effect size for association between HS and acne vulgaris/conglobata. The odds ratio (OR) was used as a summary statistic.

Results: From pooled unadjusted meta-analysis, we found a significantly higher proportion of patients with acne vulgaris/conglobata in HS cases compared to controls (OR 3.44, 95% CI 1.95–6.07, $P < 0.0001$, $I^2 = 100\%$). Pooled meta-analysis was also performed with adjusted effect sizes. This demonstrated that HS was significantly associated with acne vulgaris/conglobata after adjustment for potential confounders (OR 3.44, 95% CI 2.43–4.87, $P < 0.00001$, $I^2 = 99\%$).

Conclusions: In summary, a significant association was found between HS and acne vulgaris/conglobata. This has implications in terms of understanding the burden of disease on patient quality of life as well as consideration of optimal management strategies to target both disorders. Physicians taking care of patients with HS should be aware of this association.

Keywords: Hidradenitis suppurativa, Acne vulgaris, Acne conglobata, Androgenism, Acne

Introduction

Hidradenitis suppurativa (HS) is a chronic inflammatory skin disorder which involves painful nodules and draining abscesses in flexural areas, particularly the axillae, submammary areas, groin and perineum (Jemec 1988). HS can have a detrimental impact on quality of life, especially with the formation of fistulas, sinus tracts and scarring. These features result from obstruction and inflammation of the follicular unit (Yu and Cook 1990). There is increasing evidence that HS is a systemic inflammatory disorder, associated with other systemic disorders including cardiovascular disease, diabetes, metabolic syndrome and autoimmune disorders including thyroid disease (Dessinioti et al. 2014; Shlyankevich et al. 2014).

Acne vulgaris and its more severe variants including acne conglobata and acne fulminans are also disorders involving the follicular unit (White 1998). Mild disease is characterised by formation of inflammatory papules and pustules along with comedones in acne vulgaris, whereas more severe forms involve nodules and nodulocystic lesions. Given that follicular obstruction, dilatation and inflammation feature in both HS and acne vulgaris/

* Correspondence: kphan.vc@gmail.com
[1]Department of Dermatology, Liverpool Hospital, Liverpool, Sydney, Australia
[2]Southwest Sydney Clinical School, Liverpool Hospital, University of New South Wales (UNSW), Sydney, Australia
Full list of author information is available at the end of the article

conglobata, it has been suggested that HS is associated with acne vulgaris/conglobata (Wertenteil et al. 2019). Furthermore, both HS and acne vulgaris and its variants have similar underlying risk factors, including female gender predisposition and association with hyperandrogenism and polycystic ovarian syndrome (PCOS) (Perkins et al. 2012; Bhate and Williams 2013; Housman and Reynolds 2014). However, few studies have directly investigated the association between HS and acne vulgaris/conglobata.

To address current limitations in the evidence, we performed a systematic review and meta-analysis of available case-control studies investigation the relationship between HS and acne vulgaris/conglobata.

Methods

Search strategy

The present systematic review and meta-analysis was performed according to recommended PRISMA guidelines (Moher et al. 2009). As no human or animal subjects were involved in this study, ethics approval was not required. Electronic searches were performed using Ovid MEDLINE, PubMed, Cochrane Central Register of Controlled Trials (CCTR), Cochrane Database of Systematic Reviews (CDSR), ACP Journal Club, Database of Abstracts of Review of Effectiveness (DARE), EMBASE and PsycINFO from their dates of inception to 8 August 2018. To achieve maximum sensitivity of the search strategy and identify all studies, we combined the terms "hidradenitis suppurativa", "acne inversa", "verneuil", "acne conglobata", "acne vulgaris", as either keywords or MeSH terms (Additional file 1: Table S1). The reference lists of all retrieved articles were reviewed for further identification of potentially relevant studies. All identified articles were systematically assessed using the inclusion and exclusion criteria.

Selection criteria

All eligible case-control studies comparing patients with HS vs non-HS were included in the present review. All studies must have included either the proportion of patients with acne vulgaris/conglobata in each group, or the summary effect size for association between HS and acne vulgaris/conglobata. When institutions published duplicate studies with accumulating numbers of patients or increased lengths of follow-up, only the most complete reports were included for quantitative assessment at each time interval. All publications were limited to those involving human subjects. Language was not an exclusion criterion. Abstracts, case reports, conference presentations, editorials and expert opinions were excluded. Review articles were omitted because of potential publication bias and duplication of results.

Data extraction and quality assessment

All data were extracted from article texts, tables and figures. Data collected included study characteristics, the proportion of patients with acne vulgaris/conglobata in the HS cohort vs non-HS control cohort. If the proportion data was not available, then the effect size either in the form of odds ratio, relative risk or hazard ratio with 95% confidence interval was collected. Both unadjusted and adjusted effect sizes were pooled for meta-analysis. Discrepancies between the two reviewers were resolved by discussion and consensus. The strength of evidence for each outcome was evaluated using the Grading of Recommendations, Assessment, Development and Evaluations (GRADE) approach and presented as a summary of findings to identify the certainty of all pooled outcomes (Balshem et al, 2011). Each study was then assessed against the Newcastle-Ottawa Scale which accounted for criteria such as selection, comparability and outcome to evaluate the quality of its design.

Statistical analysis

The odds ratio (OR) was used as a summary statistic. In the present study, the random-effects model was tested, where it was assumed that there were variations between studies. The random-effects model was presented to take into account the possible clinical diversity and methodological variation between studies. χ^2 tests were used to study heterogeneity between trials. I^2 statistic was used to estimate the percentage of total variation across studies, owing to heterogeneity rather than chance, with values greater than 50% considered as substantial heterogeneity. I^2 can be calculated as $I^2 = 100\% \times (Q - \mathrm{df})/Q$, with Q defined as Cochrane's heterogeneity statistics and df defined as the degree of freedom Specific analyses considering confounding factors were not possible because raw data were not available. All P values were two-sided. All statistical analysis was conducted with Review Manager Version 5.3 (Cochrane Collaboration, Software Update, Oxford, UK).

Results

Search strategy

A total of 1271 references were identified from electronic database search using the search strategy in Additional file 1: Table S1. After application of inclusion and exclusion criteria, and filtering titles, abstracts and full texts, a final four case-control studies (Wertenteil et al. 2019; Ingram et al. 2018; Lee et al. 2018; Kimball et al. 2018) were included in the present systematic review and meta-analysis. Two studies (Vazquez et al. 2013; Ravn Jørgensen et al. 2019) were excluded as they did not include control groups to compare with HS. The included study characteristics and demographics of these studies are summarised in Table 1. There were a total of

Table 1 Characteristics of studies included in the present systematic review and meta-analysis. *HS* hidradenitis suppurativa, *n* number of cases, *ICD-10* 10th revision of the International Statistical Classification of Diseases and Related Health Problems, *SNOMED CT* SNOMED Clinical Terms

Author	Year	Study period	Country	Design	*n* (HS)	*n* (controls)	Data source	Selection of HS cases	Selection of controls	Matching	Mean age (years)	Females (%)
Ingram	2018	2013	UK	Retrospective case-control	69,842 (proxy)	93,869	Clinical Practice Research Datalink (CPRD)	Proxy cases: Based on clinical features and algorithm, confirmed by validation questionnaire	General population participants in the database without diagnosis of HS	1: 1 matched based on age, sex and registration at the same primary care practice, obesity, diabetes, smoking, comorbidities	–	–
					24,027 (physician-diagnosed)	93,869	Clinical Practice Research Datalink (CPRD)	Physician diagnosed cases: Codes M25y100 (hidradenitis) and M25y111 (hidradenitis suppurativa)	General population participants in the database without diagnosis of HS	1: 1 matched based on age, sex and registration at the same primary care practice, obesity, diabetes, smoking, comorbidities	–	–
Kimball	2018	1999–2014	USA	Retrospective case-control	2292 (mild HS)	2292	OptumHealth Care Solutions, Inc. database	At least two diagnoses for HS (ICD-9 code 705.83) recorded on different dates between January 1999 and March 2014	Database participants over the same study period without diagnosis for HS	1:1 matching based on age, sex, region of residence, healthcare plan	42.19 vs 42.19	74 vs 74
					3065 (severe HS)	3065	OptumHealth Care Solutions, Inc. database	HS severity based on predetermined clinical characteristics	Database participants over the same study period without diagnosis for HS	1:1 matching based on age, sex, region of residence, healthcare plan	42.19 vs 42.19	71 vs 71
Lee	2018	2007–2016	Korea	Retrospective case-control	28,516	142,580	Korean National Institute of Health database	Diagnosed by ICD-10 code I.729 between January 2007 and December 2016	Random selection of patients from a database without diagnosis of HS	1:5 matching based on age and sex. Adjustment for age, sex, socioeconomic status, diabetes, hypertension, hyperlipidaemia	33.6 vs 33.6	38.7 vs 38.7
Wertenteil	2018	2012–2017	USA	Retrospective case-control	48,050	16,899, 470	Explorys developed by International Business Machines (IBM) Corporation	SNOMED-CT term "hidradenitis", which has one-to-one mapping with the ICD-9 code for hidradenitis (705.83)	Database participants without diagnosis for HS	Adjustment for age, sex, obesity, smoking status, PCOS status	18-44y: 60 vs 39.6; 45-64y: 30.1 vs 33; ≥65y: 9.8 vs 27.4	74.4 vs 56.5

175,792 HS patients compared with 17,141,276 control cases.

Association between HS and acne vulgaris/conglobata

From pooled unadjusted meta-analysis, we found a significantly higher proportion of patients with acne vulgaris/conglobata in HS cases compared to controls (OR 3.44, 95% CI 1.95–6.07, $P < 0.0001$). There was significant heterogeneity noted ($I^2 = 100\%$, $P < 0.0001$) Fig. 1.

Pooled meta-analysis was also performed with adjusted effect sizes. This demonstrated that HS was significantly associated with acne vulgaris/conglobata after adjustment for potential confounders (OR 3.44, 95% CI 2.43–4.87, $P < 0.00001$). Significant heterogeneity was noted ($I^2 = 99\%$, $P < 0.00001$) Fig. 2.

Quality assessment

The certainties of the effect estimate for each outcome were deemed to be low-moderate, as per the GRADE criteria 16 due to serious risks of bias (Additional file 1: Table S1). According to the Newcastle-Ottawa Scale criteria (Wells et al. 2014), all studies were identified to be good quality (Additional file 1: Table S2).

Discussion

To our knowledge, this is the first meta-analysis investigating the association between HS and acne vulgaris/conglobata. We demonstrated from both unadjusted and adjusted meta-analysis that patients with HS were associated with a 3.4 higher odds of having acne vulgaris/conglobata compared to control cases.

Although there have been some published data looking at the association between HS and acne vulgaris/conglobata, this has predominantly been limited to cohort studies with controls. One of the earliest reports (Jemec 1988) prospectively compared 70 women with HS symptomatology vs 100 control women who were age-matched and healthy. The authors did not find any significant differences between the groups in terms of signs of hyperandrogenism or prevalence of acne vulgaris; however, this study was limited in that only females were included and only representative of a single centre experience. There are several cohort studies published reporting prevalence rates of acne vulgaris in HS of 33–45% (Vazquez et al. 2013; Mortimer et al. 1986; Brunsting 1939). Vazquez and colleagues (Vazquez et al. 2013) reported the first population study on this topic, based on the medical record linkage system of the Rochester Epidemiology Project. In their study, they reported a diagnosis of acne in 35.9% of males and 36.7% of females, with acne severity not correlated with HS severity. Mortimer and colleagues (Mortimer et al. 1986) studied 42 females with HS from a single institution, of which 19 (45%) had acne vulgaris. They also reported HS patients having higher total concentration of testosterone and free androgen index compared to healthy controls and proposed that these endocrine abnormalities may be the androgenic basis for the disease. HS patients with acne vulgaris has also been shown to have a lower mean age and age of onset of HS compared to HS patients who do not develop acne vulgaris (Ravn Jørgensen et al. 2019).

The burden of acne vulgaris and conglobata has not been extensively assessed in case-control studies. Wertenteil et al. (2019) performed a cross-sectional analysis comparing 48,050 HS cases and 16,899,470 non-HS controls. They reported an acne vulgaris prevalence of 16.4% in females compared to 11.8% in males. HS patients had a 4.5 times higher odds of having acne vulgaris compared to controls, and the prevalence was higher in females aged 18–44 years, non-whites, obese and patients with PCOS. Ingram et al. (2018) performed an analysis of the national United Kingdom Clinical Practice Research Datalink. The authors also reported a significant association between both proxy-diagnosed and physician-diagnosed HS with acne vulgaris, with odds of 2.17 and 1.77, respectively. However, the authors did not perform subgroup analysis to determine which type of HS patients were at higher risk of developing acne. The aforementioned studies have predominantly focused on Western populations, whereas few studies have focused on the epidemiology of HS in Asian

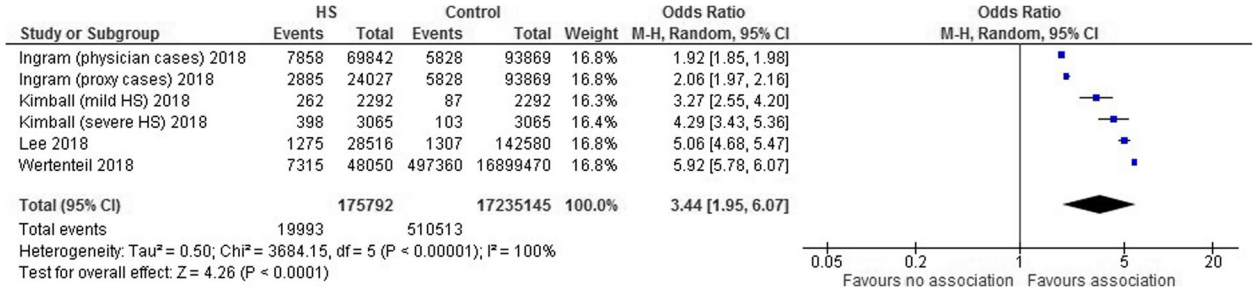

Fig. 1 Forest plot of unadjusted meta-analysis of case-control studies, with pooled effect sizes testing the association between hidradenitis suppurativa (HS) and acne vulgaris and conglobata

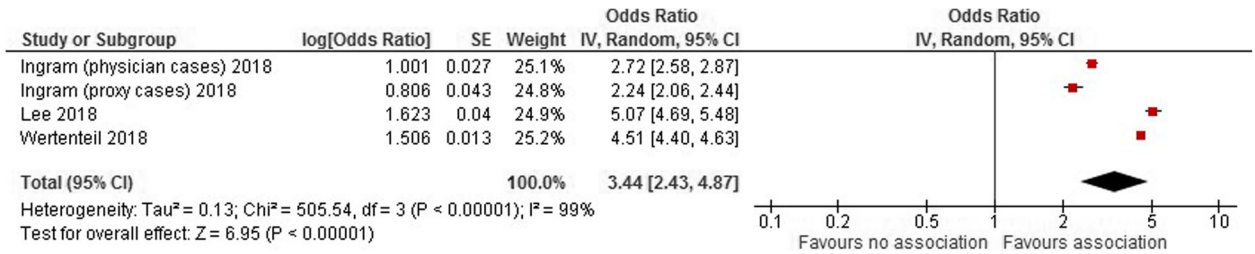

Fig. 2 Forest plot of adjusted meta-analysis of case-control studies, with pooled effect sizes testing the association between hidradenitis suppurativa (HS) and acne vulgaris and conglobata

populations. Lee et al. (2018) performed an analysis of HS and control cases in South Korean using the Korean National Health Insurance database. It was found that HS was also significantly associated with acne conglobata in this Asian population study. This suggests that the pathogenesis underlying the association between HS and acne vulgaris/conglobata may not be dependent on the underlying genetic or racial differences between Western and Asian populations.

There are multiple clinical features which are common to both the development of HS and acne vulgaris/conglobata, including follicular obstruction, dilatation, inflammation and rupture. Common pathological mechanisms, such as excessive activation of the innate immune system and increased cytokine and interleukin-1 release, may lead to neutrophil-rich cutaneous inflammation (Patel et al. 2015; Thein et al. 2004; Cugno et al. 2017). Interleuki-17 is another cytokine that has been shown to be overexpressed in both HS and acne vulgaris (Agak et al. 2014; Schlapbach et al. 2011). It plays a role in mobilising neutrophils to peripheral tissues, which may promote cutaneous neutrophil-driven inflammation. Interestingly, the above mechanisms are also integral in systemic inflammatory disorders including inflammatory arthropathies. Patients with HS-related arthritis may also experience other inflammatory skin diseases such as acne congloblata, dissecting cellulitis of the scalp and pilonidal sinus forming the follicular occlusion disorders as well as acne vulgaris and less commonly pyoderma gangrenosum, acral pustular psoriasis and even systemic amyloidosis (Salim et al. 2003; Richette et al. 2014; Girouard et al. 2012). The combination of inflammatory skin conditions and arthritis may form specific syndromes, including PAPASH (pyogenic arthritis, pyoderma gangrenosum, acne and hidradenitis suppurativa) and PASH (pyoderma gangrenosum, acne vulgaris, hidradenitis suppurativa) syndromes. It has been hypothesised that IL-1 plays a pathogenic role in these auto-inflammatory conditions, a hypothesis that has been strengthened by the improvement of symptoms in response to IL-1 or IL-1 receptor blockade (Leuenberger et al. 2016). There may also be underlying genetic risk factors that predispose some patients to the development of both acne and HS in PAPASH, including a mutation in the PSTPIP1 gene located on chromosome 15 which is thought to be responsible for activation and assembly of the inflammasome (Braun-Falco et al. 2012). PASH is typically associated with a NCSTN mutation, a gene coding for the transmembrane receptor involved in notch signalling. However, it was also recently associated with the PSTPIP1 gene mutation (Calderón-Castrat et al. 2016).

The present study is constrained by several limitations. There is a significant lack of available literature to date directly addressing the relationship between HS and acne vulgaris/conglobata, with only four case-control studies of adult cases identified. Of the included studies, only Wertenteil et al. (2019) performed a primary analysis of the prevalence of acne vulgaris amongst HS patients. There is also considerable heterogeneity in the source of cases/control cases amongst the studies, including variation in the racial distribution of included patients, and as such results should be interpreted with significant caution. Diagnostic criteria for HS cases also varied, with registry or large database studies being based on case retrieval by diagnostic code. Furthermore, not all included studies provided adjusted effect sizes for pooled meta-analysis. Given the limitations of the presented data in the included studies, we were unable to perform subgroup analysis to determine whether HS disease severity correlated with acne severity. We were also unable to perform meta-analysis to determine which subgroups of patients had the strongest association of HS with acne vulgaris/conglobata.

Conclusions

In summary, a significant association was found between HS and acne vulgaris/conglobata. This association remained significant even after adjustment of potential confounding factors. This has implications in terms of understanding the burden of disease on patient quality of life as well as consideration of optimal management strategies to target both disorders. Physicians taking care of patients with HS should be aware of this association.

Additional file

Additional file 1: Table S1. Ovid Medline Search strategy used for the present systematic review and meta-analysis. **Table S2.** GRADE assessment of primary outcomes to assess the level of evidence contributing to each outcome. OR, hazard ratio; CI, confidence interval. The overall quality score is determined based on the sum of the included domains. Type of evidence is based on design of the included studies. The study quality reflects the blinding and allocation, follow-up and withdrawals, sparsity of data, and methodological concerns. Consistency is graded based on heterogeneity of included population and study end points with respect to one another. Directness is graded based on generalizability of included results. The overall quality of results for each outcome can be considered high (≥ 4 points), moderate (3 points), low (2 points) or very low (≤ 1 point). **Table S3.** Newcastle Ottawa Scale for assessment of study quality. The criteria for quality assessment consist of eight criteria, which can be answered as either present/implied (*) or absent (.). The criteria, with maximum score in brackets, are: 1. Representativeness of Cohort (*), 2. Selection of Nonexposed (*), 3. Cohort Ascertainment of Exposure (*), 4.Outcome of Interest (*), 5.Comparability of Cohorts (**), 6. Assessment of Outcome (*), 7.Adequate Duration of Follow-up (*), 8.Adequate Follow-up of Cohort (*).Good quality: 3 or 4 stars in selection domain (Criteria 1-4) AND 1 or 2 stars in comparability domain (Criteria 5) AND 2 or 3 stars in outcome/exposure domain (Criteria 6-8). Fair quality: 2 stars in selection domain AND 1 or 2 stars in comparability domain AND 2 or 3 stars in outcome/exposure domain. Poor quality: 0 or 1 star in selection domain OR 0 stars in comparability domain OR 0 or 1 stars in outcome/exposure domain.

Acknowledgements
None declared.

Availability of data materials
Available in text and supplemental material

Authors' contributions
KP, OA and SS contributed to the study design, interpretation, drafting and final draft of the manuscript. KP contributed to the data extraction and statistical analysis. All authors read and approved the final manuscript

Funding
None

Ethics approval and consent to participate
Ethics waived for the present project as no patients or animals involved in the study.

Consent for publication
Not applicable as patients are not involved in the present study.

Competing interests
SS has received sponsorship from Abbvie for international conference attendance and honorarium from Abbvie for educational presentations and advisory board membership. SS is an investigator for clinical trials with UCB and Abbvie. All other authors declare that they have no conflicts of interest.

Author details
[1]Department of Dermatology, Liverpool Hospital, Liverpool, Sydney, Australia. [2]Southwest Sydney Clinical School, Liverpool Hospital, University of New South Wales (UNSW), Sydney, Australia. [3]Department of Dermatology, Royal North Shore Hospital, St Leonards, Sydney, Australia. [4]Northern Clinical School, Sydney Medical School, University of Sydney, Sydney, Australia. [5]The Dermatology and Skin Cancer Centre, Gosford and St Leonards, Sydney, Australia.

Received: 29 July 2019 Accepted: 20 September 2019
Published online: 27 December 2019

References
Agak GW, Qin M, Nobe J, et al. Propionibacterium acnes induces an IL-17 response in acne vulgaris that is regulated by vitamin A and vitamin D. J Invest Dermatol. 2014;134:366–73.
Balshem H, Helfand M, Schünemann HJ, et al. GRADE guidelines: 3. Rating the quality of evidence. J Clin Epidemiol. 2011;64:401–6.
Bhate K, Williams H. Epidemiology of acne vulgaris. Br J Dermatol. 2013;168: 474–85.
Braun-Falco M, Kovnerystyy O, Lohse P, Ruzicka T. Pyoderma gangrenosum, acne, and suppurative hidradenitis (PASH)–a new autoinflammatory syndrome distinct from PAPA syndrome. J Am Acad Dermatol. 2012;66:409–15.
Brunsting HA. Hidradenitis suppurativa; abscess of the apocrine sweat glands: a study of the clinical and pathologic features, with a report of twenty-two cases and a review of the literature. Arch Dermatol Syphilol. 1939;39:108–20.
Calderón-Castrat X, Bancalari-Díaz D, Román-Curto C, et al. PSTPIP 1 gene mutation in a pyoderma gangrenosum, acne and suppurative hidradenitis (PASH) syndrome. Br J Dermatol. 2016;175:194–8.
Cugno M, Borghi A, Marzano AV. PAPA, PASH and PAPASH syndromes: pathophysiology, presentation and treatment. Am J Clin Dermatol. 2017;18: 555–62.
Dessinioti C, Katsambas A, Antoniou C. Hidradenitis suppurrativa (acne inversa) as a systemic disease. Clin Dermatol. 2014;32:397–408.
Girouard SD, Falk RH, Rennke HG, Merola JF. Hidradenitis suppurativa resulting in systemic amyloid A amyloidosis: a case report and review of the literature. Dermatol Online J. 2012;18:2.
Housman E, Reynolds RV. Polycystic ovary syndrome: a review for dermatologists: part I. Diagnosis and manifestations. J Am Acad Dermatol. 2014;71:847.
Ingram J, Jenkins-Jones S, Knipe D, et al. Population-based Clinical Practice Research Datalink study using algorithm modelling to identify the true burden of hidradenitis suppurativa. Br J Dermatol. 2018;178:917–24.
Jemec G. The symptomatology of hidradenitis suppurativa in women. Br J Dermatol. 1988;119:345–50.
Kimball AB, Sundaram M, Gauthier G, et al. The comorbidity burden of hidradenitis suppurativa in the United States: a claims data analysis. Dermatol Ther (Heidelb). 2018;8:557–69.
Lee JH, Kwon HS, Jung HM, Kim GM, Bae JM. Prevalence and comorbidities associated with hidradenitis suppurativa: a nationwide population-based study. J Eur Acad Dermatol Venereol. 2018;32:1784–90.
Leuenberger M, Berner J, Di Lucca J, et al. PASS syndrome: an IL-1-driven autoinflammatory disease. Dermatology. 2016;232:254–8.
Moher D, Liberati A, Tetzlaff J, Altman DG. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. Ann Intern Med. 2009;151:264–9.
Mortimer PS, Dawber R, Gales MA, Moore RA. Mediation of hidradenitis suppurativa by androgens. Br Med J (Clin Res Ed). 1986;292:245–8.
Patel M, Cohen JM, Wright NA, et al. Epidemiology of concomitant psoriasis and hidradenitis suppurativa (HS): experience of a tertiary medical center. J Am Acad Dermatol. 2015;73:701–2.
Perkins AC, Maglione J, Hillebrand GG, Miyamoto K, Kimball AB. Acne vulgaris in women: prevalence across the life span. J Women's Health (Larchmt). 2012; 21:223–30.
Ravn Jørgensen AH, Ring HC, Thomsen SF. Acne vulgaris in patients with hidradenitis suppurativa. J Am Acad Dermatol. 2019;80:e129–30.
Richette P, Molto A, Viguier M, et al. Hidradenitis suppurativa associated with spondyloarthritis-results from a multicenter national prospective study. J Rheumatol. 2014;41:490–4.
Salim A, David J, Holder J. Dissecting cellulitis of the scalp with associated spondylarthropathy: case report and review. J Eur Acad Dermatol Venereol. 2003;17:689–91.
Schlapbach C, Hänni T, Yawalkar N, Hunger RE. Expression of the IL-23/Th17 pathway in lesions of hidradenitis suppurativa. J Am Acad Dermatol. 2011;65: 790–8.
Shlyankevich J, Chen AJ, Kim GE, Kimball AB. Hidradenitis suppurativa is a systemic disease with substantial comorbidity burden: a chart-verified case-control analysis. J Am Acad Dermatol. 2014;71:1144–50.
Thein M, Hogarth M, Acland K. Seronegative arthritis associated with the follicular occlusion triad. Clin Exp Dermatol. 2004;29:550–2.

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;133:97–103.
Wells G, Shea B, O'connell D, et al. The Newcastle-Ottawa Scale (NOS) for assessing the quality of nonrandomised studies in meta-analyses. Ottawa: Ottawa Hospital Research Institute; 2014.
Wertenteil S, Strunk A, Garg A. Overall and subgroup prevalence of acne vulgaris among patients with hidradenitis suppurativa. J Am Acad Dermatol. 2019;80:1308–13.
White GM. Recent findings in the epidemiologic evidence, classification, and subtypes of acne vulgaris. J Am Acad Dermatol. 1998;39(2 Pt 3):S34–7.
Yu CC, Cook MG. Hidradenitis suppurativa: a disease of follicular epithelium, rather than apocrine glands. Br J Dermatol. 1990;122:763–9.

Publisher's Note