Prevalence, Risk Factors, and Comorbidities of Hidradenitis Suppurativa

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KEYWORDS

• Prevalence • Incidence • Hidradenitis suppurativa • Metabolic syndrome • Smoking • Malignancies • Spondylarthritis • Psychological comorbidities

KEY POINTS

- The true prevalence of hidradenitis suppurativa (HS) is challenging to estimate because it is often under diagnosed and misdiagnosed; the incidence seems to be increasing.
- HS is associated with a wide range of somatic comorbidities, from metabolic syndrome to rheumatologic conditions, as well as psychological comorbidities.
- The sum of somatic and psychological comorbidities places significant burden on HS patients beyond dermatologic symptoms.
- Treatment of HS needs to target dermatologic symptoms as well as possible comorbidities.

PREVALENCE AND INCIDENCE

As hidradenitis suppurativa (HS) has been an orphan disease for decades and subsequently a highly misdianogsed and underdiagnosed condition with a significant diagnose delay,[1,2] the true prevalence has been correspondingly challenging to estimate. Prevalences are reported as low as 0.00033% and as high as 4.1%.[3–8]

Prevalence estimates seem to fluctuate according to the nature of the study design, participants, and geography. A uniform pattern based on these methodologic differences present low prevalence rates in predominantly American studies performed on insurance databases, and contrasting higher prevalences in studies based on an HS diagnosis determined by physical examination or interviews/questionnaires.

In a health-insured–only US population of 15,054,519 participants, Cosmatos and colleagues[7] found an unadjusted prevalence of 0.053%. A similar low prevalence was found in the Massachusetts General Hospital Database.[9]

In contrast, a Danish study based on HS symptomatology in 100 females from the staff or patients referred to Department of Dermatology reported a prevalence of 4%.[5] A similar point prevalence of 4.1% was described in Danish study based on physical examination of 507 patients undergoing screening for sexually transmitted diseases.[4]

Questionnaires aimed at diagnosing HS using simple descriptions of symptoms, for example, boils and location of the boils, suggest a specificity and sensitivity of 82% to 97% and 90% to 97%, respectively.[6,10] A French population-based questionnaire study of 10,000 participants found a prevalence of 1%,[8] and a more recent Danish population study of 17,454 participants from the general population reported a prevalence of 2.1%.[6]

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The incidence of HS based on an American database has been suggested to be 6 per 100,000 person-years, and seem to be increasing.[11] Thus, an increase from 4.3 per 100,000 (during 1970–1979) to 9.6 per 100,000 (during 2000–2008) was noted.[11]

RISK FACTORS AND COMORBIDITIES

There is a considerable overlap between what are characterized risk factors and comorbidities. One possible definition of a risk factor is something that increases a person's chances of developing a disease, whereas a comorbidity may be defined as a coexisting medical condition or disease process, and may be categorized as psychological or somatic.

Age and Sex

The mean age of onset is the early 20s; however, HS has additionally been reported children and postmenopausal women.[8,12] Considerable literature state a decline in prevalence after the age of 55, which may reflect an age-related clinical burnout of the HS activity or hormonal changes.[6,8] Equivalently, the observation that the female:male ratio is 3:1 led to the theory of androgens playing a pathogenetic part in HS. However, investigations have failed to support this hypothesis.[5,13]

Obesity

A sizable body of literature demonstrates obesity as a paramount risk factor.[8,14–18] Recently, a cross-sectional hospital- and population-based study comparing 32 hospital-based HS subjects, 326 population-based HS subjects, and 14,851 controls (non-HS subjects) found an odds ratio (OR) for obesity (body mass index [BMI] $\geq$30 kg/m^2) of 6.38 (95% CI, 2.99–13.62) and 2.56 (95% CI, 2.00–3.28) for hospital- and population-based HS subjects, respectively, when compared with controls.[18] Correspondingly, this study found an OR for abdominal obesity of 3.62 (95% CI, 1.73–7.60) and 2.24 (95% CI, 1.78–2.82) for the hospital- and population-based HS subjects, respectively.[18] Additionally, 2 studies including 336 and 80 hospital-based HS patients compared with controls found an association of HS and BMI as well as abdominal obesity.[16,17] Ambiguous results have, however, been described.[8]

An association between BMI and the HS severity measurement Sartorius score was found in 251 HS patients implying a dose–response relationship.[15] Moreover, the higher OR reported for hospital HS subjects compared with HS subjects from the general population might reflect differences in HS severity, and therefore be supportive of the dose–response relationship.[18] Nonetheless, results remain inconsistent with regard to severity, and surprisingly some studies have reported no correlation between the severity or duration of HS and obesity.[14,16,18]

Interventional studies are limited. However, 1 study demonstrated that a weight reduction of 15% in patients with BMI of greater than 30 kg/m^2 ameliorates HS supporting a dose–response relationship.[19] Moreover, obesity was reported as a risk factor for recurrence after CO_2 laser treatment of HS patients.[20]

Some factors may aid the pathophysiologic mechanisms behind the association of obesity and HS. The adipose cells are considered an independent endocrine tissue capable of secreting proinflammatory cytokines, which may add to the chronic inflammatory state of HS. Furthermore, obesity may lead to large skin folds enhancing the warm, humid milieu and skin-to-skin contact making a mechanically dependent exacerbation or maintenance of the HS lesions.

Smoking

Various studies report an association between smoking and HS. Rates of smoking in HS patients have been noted from 42% up to 70% to 92%.[18,21,22] A German study found the odds of having HS to be 9.4 times greater in current smokers versus non/ex-smokers.[23] Additionally, a French population-based study described a link between HS and current smoking, but not prior smoking.[8] The association with smoking has also been related to clinical HS disease severity.[15] It has recently been proposed that nonsmoking and nonobesity is associated with a better chance of HS remission, and it has come to light that HS surgery combined with smoking cessation give rise to fewer or no lesions.[21] Further aiding the dose–response relationship is a study investigating the role of smoking and obesity concluding that the severity of HS is worse in previous smokers when compared with never-smokers.[15]

A relationship between cigarette smoking and inflammation has been established previously. Kurzen and colleagues[24] hypothesized on possible pathophysiologic mechanisms of smoking on HS, for example, modification in the microflora of the skin and a prolonged secretion of nicotine in sweat inducing tumor necrosis factor (TNF)-α release and follicular occlusion.

Genetics

There also seems to be a genetic component to HS, which is discussed in greater detail by