

The Role of Chronic Inflammation in Hidradenitis Suppurativa Progression and Patient Outcomes

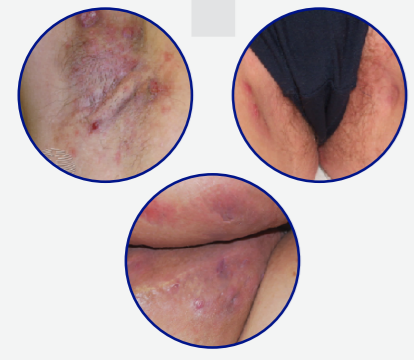
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What is HS?

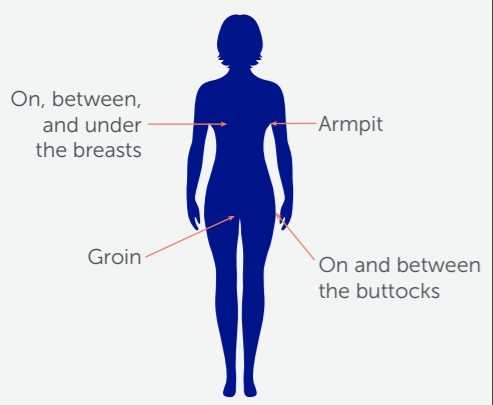
HS is a chronic, inflammatory, recurrent, and debilitating skin disease of the hair follicle¹

HS presents with painful, deep-seated, inflamed lesions in the intertriginous, apocrine gland-bearing areas of the body¹



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Most common locations of HS:^{1,3,4}

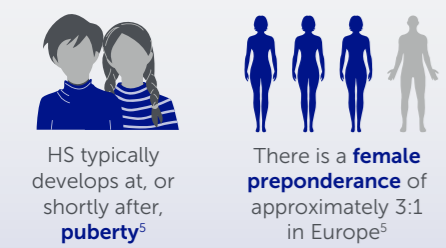


Diagnosis of HS is made by naked-eye clinical observation and symptom history^{3,4}

Criteria: Recurrent painful or suppurating lesions occurring more than twice in 6 months in one of the intertriginous areas of the body^{4,4}

Affected populations

Approximately **1 in 100** people in Europe have HS⁵



Chronic uncontrolled inflammation leads to tunnel formation, irreversible tissue destruction, and scarring^{6,7}

HS is a complex, multifactorial disease⁴

Although the pathophysiology of HS has not yet been fully elucidated, the TNF- α and IL-17 pathways have an important role, and synergise with many cytokines to sustain inflammation⁶

Compared with healthy controls:

- >40x** IL-17-producing CD4⁺ T cells⁶
- Elevated levels of IL-17A and IL-17F⁸
- 5x** TNF expression⁹

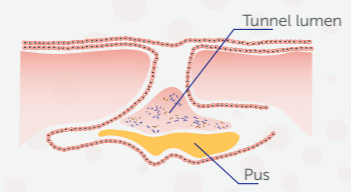
have been found in lesional tissue in patients with HS

Draining tunnels are a unique feature of moderate-to-severe HS and a source of inflammation, actively contributing to further disease progression^{6,10}

IL-17A and IL-17F activate tunnel-lining keratinocytes to release a range of chemokines, which drive dermal inflammation around tunnels and neutrophil influx into the tunnel lumen⁶

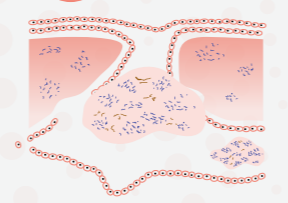
This positive amplification loop of inflammation contributes to irreversible tissue destruction and scarring⁶

5 Further rupture and tunnel formation



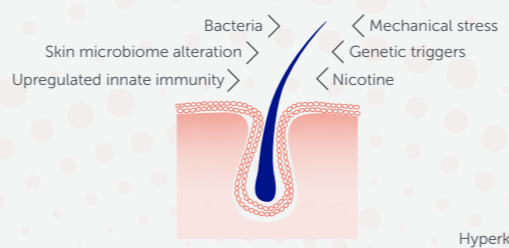
Whole regions become highly inflamed and tunnels form, which can leak purulent discharge. The skin thickens and fibrosis develops in the dermis and epidermis, leading to scarring. Tissue destruction promotes further chronic inflammation.

4 Rupture



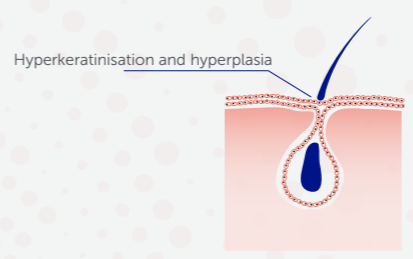
Hair follicle rupture releases the inflammatory contents into the dermis. Secondary bacteria may colonise lesions. Proinflammatory positive feedback cycle further increases inflammation.

1 Trigger



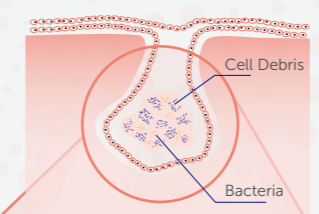
HS is triggered by genetic and/or environmental factors.

2 Follicular infundibulum occlusion



Hyperkeratinisation leads to bulb occlusion.

3 Bulb dilation



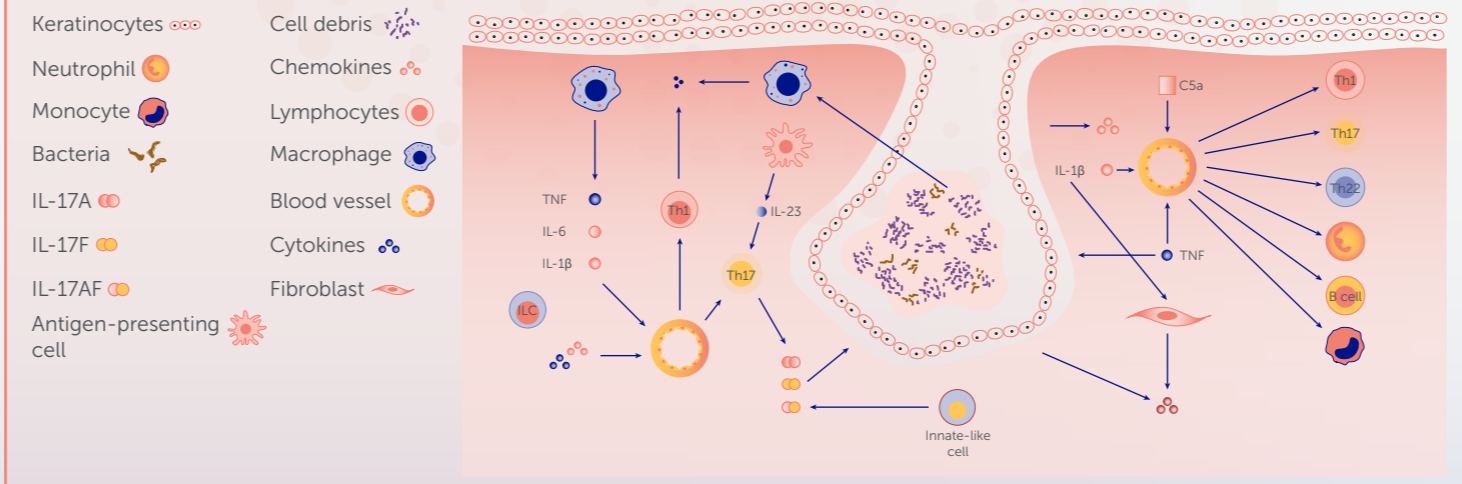
The hair is lost, and the occluded follicular bulb dilates. Immune cells are activated, producing and releasing inflammatory cytokines and chemokines.

Current proposed pathogenic mechanism of severe HS^{6,7}

Propagation of inflammation

Tissue destruction

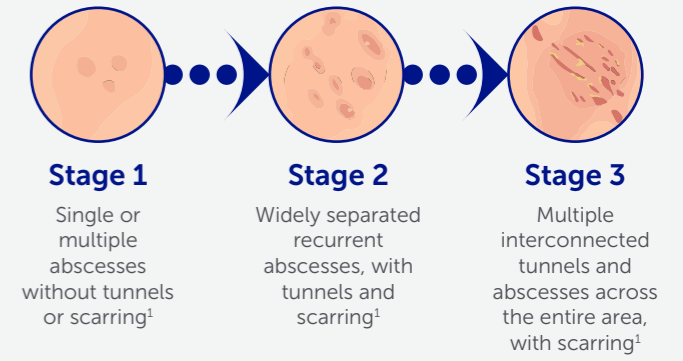
Inflammatory cascade



HS is a chronic and debilitating disease⁴

The Hurley staging system assesses the extent and type of lesions to indicate the severity of HS and direct treatment¹

Increasing severity, decreasing quality of life¹¹



Stage 3 HS has a profoundly negative impact on all aspects of a patient's life¹¹

- Many patients with HS report a very much or extremely impaired quality of life,¹² which declines with increasing Hurley stage¹¹
- Patients live daily with chronic pain, purulent discharge, restriction of mobility (due to interconnected tunnels and scarring), stigmatisation, impaired body image, sexual dysfunction, unemployment, and social isolation⁶
- The burden of HS leads to high rates of depression, anxiety, and suicide⁶
- Uncontrolled chronic inflammation can also result in systemic inflammation, and patients with HS have a high frequency of comorbidities, adding to the existing burden⁶
- Despite the extensive impact on patient quality of life, diagnostic delay is a serious and prevalent issue for patients with HS and is associated with severe disease and a higher prevalence of systemic comorbidities⁶

~7 YEARS The average diagnostic delay⁷

≥5 The average number of healthcare professionals visited before diagnosis¹³

Timely diagnosis of HS is essential for the initiation of appropriate treatment to minimise disease progression, irreversible tissue destruction, and disease burden⁶

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Abbreviations
C5a: complement component 5a; CD4⁺: cluster of differentiation 4; HS: hidradenitis suppurativa; IL: interleukin; ILC: innate lymphoid cell; Th: T helper cell; TNF: tumour necrosis factor.
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