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HS is challenging because it is a chronic, relapsing inflammatory disease with substantial physical, functional, and psychological consequences

Q1 What makes hidradenitis suppurativa (HS) such a challenging disease for patients and clinicians?

HS is challenging because it is a chronic, relapsing inflammatory disease with substantial physical, functional, and psychological consequences. Pain, drainage, odour, sleep disturbance, sexual-health concerns, and scarring can be profoundly disabling. Disease activity and irreversible structural damage often coexist, so controlling inflammation alone may not restore function, while surgery alone does not address the inflammatory drive. HS is also associated with a substantial comorbidity burden, including obesity and cardiometabolic risk, mental-health conditions, and immune-mediated inflammatory disease in selected patients. Effective care therefore requires early recognition, longitudinal assessment, and a multidisciplinary, patient-centred approach.

Q2 Why does diagnostic delay remain such a significant issue in HS?

HS is still frequently mistaken for recurrent infection, boils, or isolated abscesses. Lesions may be treated episodically without recognising the characteristic combination of typical morphology, intertriginous distribution, and recurrence. Public and professional awareness remains uneven, and some patients delay seeking care because of stigma, uncertainty, or the intimate location of lesions. Across studies, the average diagnostic delay remains approximately 7–10 years. This delay permits progression to tunnels and fibrosis, which are harder to reverse. Improving recognition and referral pathways in primary care, emergency medicine, gynaecology, surgery, and other front-line settings is essential.

Q3 How has our understanding and management of HS evolved in recent years?

Our understanding of this complex immune-mediated disease continues to deepen; while it involves intricate pathogenic interplays, this growing knowledge is providing us with an expanding therapeutic armamentarium to deliver truly appropriate and personalised care. We now recognise HS as an immune-mediated, follicular disorder, fundamentally transforming clinical practice. Crucially, no two patients are alike, and management must be tailored to the predominant lesion type, clinical phenotype, individual risk of progression, patient age, and coexisting comorbidities. We are also increasingly aware of the need to establish well-defined therapeutic goals and utilise workable, operational severity assessment scales to achieve disease control and accurately capture response to treatment, integrating bedside high-frequency cutaneous ultrasound whenever feasible. Non-draining but inflamed tunnels are clinically relevant and should be considered when assessing disease extent and severity. Ultrasound provides superior mapping of subclinical lesions, tunnels, and inflammatory

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extension, both in routine consultation and perioperatively. Consequently, management has evolved well beyond repeated antibiotic courses towards individualised, combined strategies, incorporating lifestyle interventions, hair-reduction measures, flare management, pain control, targeted systemic therapies, and timely procedural or surgical intervention as the standard of care. In many patients, combination treatment is the rule rather than the exception.

Q4 What factors are most important when individualising treatment?

I consider inflammatory burden, the presence and extent of tunnels and scarring, anatomical site, pain, flare frequency, and the patient's functional priorities to be most important. Comorbidities, smoking status, body weight, hormonal factors, reproductive plans, prior treatment exposure, and access to specialist surgery also influence the plan. Hurley stage alone is insufficient: it describes irreversible damage but is not a dynamic measure of inflammation. Active lesion counts and dynamic disease-activity scores should also be used together with validated patient-reported measures that capture the impact of disease on quality of life. Treatment targets should be agreed with the patient and reassessed using both clinical and patient-reported outcomes.

Q5 How should clinicians approach patients whose disease remains uncontrolled despite treatment?

The first step is to define why control has not been achieved. We should reassess phenotype, anatomical extent, adherence, dose and duration of previous treatments, treatment-limiting adverse events, and important differential diagnoses or coexisting disease. Ultrasound can be particularly useful when the clinical extent or presence of active tunnels is uncertain. Persistent disease should trigger therapeutic optimisation, timely switching where appropriate, and referral to an experienced multidisciplinary HS team. Crucially, persistent tunnels require a procedural or surgical plan; escalation of systemic therapy alone is often not enough.

Q6 What role does surgery have within the modern HS treatment pathway?

Surgery is a core component of HS care, not a final resort. De-roofing and other tissue-sparing procedures can be valuable for selected localised tunnels, whereas wider excision may be necessary for extensive, chronically damaged areas. The procedure, margins, reconstruction, and timing should be individualised. In my view, the best results come from combining surgery with systemic anti-inflammatory control, rather than treating them as competing options. Whenever feasible, surgery should be planned during a period of optimised inflammatory control, while recognising that persistent structural lesions may still require timely intervention. This integrated approach aims to preserve function, support healing, and reduce the risk of local recurrence.

> **The best results come from combining surgery with systemic anti-inflammatory control**

Q7 Which emerging therapies or developments are you most excited about?

The expanding therapeutic landscape is encouraging, particularly the validation of inflammatory pathways beyond tumour necrosis factor. Equally important are better treatment sequencing and combination strategies for patients who have both active inflammation and

structural disease. I am also interested in translational work linking clinical phenotypes with tissue, molecular, imaging, and microbiome data, with the aim of identifying biomarkers that can support more tailored treatment. Imaging is advancing rapidly, with high-frequency and ultrahigh-frequency ultrasound, Doppler, quantitative elastography, and handheld devices, alongside other emerging techniques, improving disease staging and longitudinal follow-up. Therapies targeting metabolic pathways are under investigation and attracting growing interest in the context of obesity and metabolic comorbidity, although HS-specific evidence remains limited. Laser and device-based approaches may have an adjunctive role in selected patients. Digital tools and AI could improve lesion quantification, tunnel mapping, and outcome capture, but they require robust prospective clinical validation and meaningful patient involvement. At an international level, there is ongoing work towards consensus, validated definitions of disease progression and its risk factors, as well as around the concepts of window of opportunity, disease interception, and disease modification, together with operative therapeutic outcomes such as minimal disease activity.

Q8 How can we better integrate management of HS-associated comorbidities and psychosocial burden into routine care?

Comorbidity assessment should be built into the initial consultation and reviewed over time. This includes cardiometabolic risk, smoking, inflammatory bowel disease, and musculoskeletal symptoms where clinically indicated, as well as depression, anxiety, sexual health, and sleep. Pain should be assessed and actively treated rather than accepted as inevitable. A multidisciplinary network is often more realistic than a single approach: dermatology can coordinate care with primary care, surgery, psychology, nurses, pain services, gastroenterology, rheumatology, and gynaecology or urology when needed. Patients should be directly asked about the issues that are most difficult for them.

Q9 What are the biggest unmet needs that research needs to address?

We need biomarkers that predict disease course, flares, remission, and treatment response, alongside validated algorithms for sequencing and combining medical and surgical treatment. Translational research should help us move towards genuinely tailored therapy rather than a one-size-fits-all approach. Trial endpoints must also capture what matters to patients: pain, drainage, function, flare burden, work productivity, and durable control, not only lesion counts, although these remain very important. We need stronger evidence in difficult anatomical sites, in advanced tunnel disease, and in groups historically under-represented in research. Finally, innovative techniques and implementation research are needed to reduce diagnostic delay and inequities in access to specialist care.

> **The ultimate aim is not simply fewer lesions; it is greater comfort, function, dignity, and quality of life**

Q10 Looking ahead, what would you most like to see change in HS care?

I would like HS to be recognised earlier and managed proactively, before chronic tunnels and disabling scarring develop. The priority should be to alter the life course of people living with HS: intercept progression where possible, achieve sustained control or minimal disease activity where realistic, and protect function and quality of life. Every patient should have access to a coordinated pathway that includes expertise in systemic treatment, surgery, wound care, pain, and psychological support. We should move from repeated crisis management to personalised, longitudinal care with clearly defined treatment goals. The ultimate aim is not simply fewer lesions; it is greater comfort, function, dignity, and quality of life.

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