

A 28-Year-Old Man With Draining Nodules and Cysts in His Axilla

Isabelle Moseley, BA¹ • Allison Robbins, MD² • Oliver J. Wisco, DO^{3,4}

A 28-year-old man with a medical history significant for mild hypertension and pre-obesity presents with bilateral draining nodules and cysts with sinus tract formation in the axillae (**Figure**). He has minimal involvement of the inguinal folds.

A full-body examination of the skin reveals no other similar lesions, and there is notable sparing of the oral mucosa and nails. No lymphadenopathy was observed. The lesions have been present since his early teenage years and seem to wax and wane but never fully resolve. They can be very sore and periodically have a foul odor.

Which of the following is the most likely diagnosis?

- A. Furunculosis
- B. Marjolin ulcer
- C. Hidradenitis suppurativa
- D. Intertrigo

Answer: C. Hidradenitis suppurativa

The answer is hidradenitis suppurativa (HS). HS is an inflammatory disorder characterized by recurrent skin disease affecting the follicular unit and apocrine glands and presenting with deep-seated nodules that expand to form abscesses.^{1,2}

Differential Diagnosis

Furunculosis is characterized by multiple furuncles, which are small, purulent abscesses typically caused by a bacterial infection of a hair follicle.^{1,3} When there are several adjacent lesions involved, it is called a carbuncle. These subcutaneous lesions with overlying erythema and edema may mimic the appearance of HS; however, a microbial agent can usually be identified with cultured swabs.³ Furthermore, unlike the bilaterally occurring



Figure. Axillary nodules and draining cysts

cutaneous lesions of HS, furuncles have a random distribution and are commonly accompanied by fever and lymphadenopathy.⁴

A step beyond the chronic inflammation of HS, Marjolin ulcers reflect the progression of a chronic, nonhealing lesion to malignant degeneration. Typically, Marjolin ulcers arise within longstanding scars (commonly burn scars) or chronic wounds or inflammatory lesions.⁵ They present visually as ulcerative, foul-smelling lesions with rapid growth and rolled elevated margins. Histopathologic examination usually reveals underlying squamous cell carcinoma, but other cell types, including basal cell carcinoma, have been described.⁵

Intertrigo is a superficial inflammatory dermatitis arising in intertriginous areas as a result of moisture, friction, and/or lack of ventilation.⁶ Like HS, intertrigo is commonly associated with obesity and

AFFILIATIONS:

¹Warren Alpert Medical School of Brown University, Providence, RI

²Department of Dermatology, Warren Alpert Medical School of Brown University, Providence, RI

³Director, Department of Cutaneous Oncology, Warren Alpert Medical School of Brown University, Providence, RI

⁴Department of Dermatology, Warren Alpert Medical School of Brown University, Providence, RI

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CORRESPONDENCE:

Isabelle Moseley, BA, Brown University, 339 Eddy Street, Providence, RI 02903
(isabelle_moseley@brown.edu)

presents as intertriginous erythematous scaly plaques. However, intertrigo is characterized by pruritic regions of erythema and peripheral scaling between skin folds, which can be distinguished from the deep abscesses and old scars present in HS.⁶ These are polymicrobial infectious lesions, with candida being the most common pathogen.

Discussion

The diagnosis of HS is clinical; histopathology and other laboratory findings can rule out other diagnoses but cannot confirm the presence of HS.¹ Bacterial cultures are often performed to exclude an infectious etiology but are not required for diagnosis.¹ Although a bacterial culture was not performed for this patient because of the quintessential clinical appearance, a sterile swab would better support a diagnosis of HS rather than an infectious process. Criteria for definitive diagnosis of HS include (1) characteristic lesions (nodules, abscesses, sinus tracts, scarring), (2) typical distribution at flexural sites (skin folds, such as the groin, axillae, and inframammary regions), and (3) history of chronicity and lesion recurrence (more than twice in 6 months).^{1,2,7} Patients are categorized into 3 groups of severity based on the Hurley staging system:¹

- Stage I (mild): abscess formation (single or multiple) but no sinus tracts or scarring
- Stage II (moderate): recurrent abscesses with tract formation and scarring or multiple widely separated lesions
- Stage III (severe): diffuse or near-diffuse lesions, or multiple interconnected abscesses and tracts

The prevalence of HS is estimated to be 0.1% in the United States.² HS is more than twice as common in women than in men and most commonly affects young adults and individuals aged 30 to 39 years.^{2,8} Characterized by often painful, purulent lesions, HS may significantly impair a patient's quality of life and if left untreated, may lead to debilitating long-term complications, including genitourinary strictures,

scarring, and squamous cell carcinoma.^{2,9}

HS represents one-fourth of the follicular occlusion tetrad, a symptom complex consisting of acne conglobata, dissecting cellulitis of the scalp, and pilonidal sinus in addition to HS.¹⁰ The precise pathogenesis of this group of diseases has not been elucidated, but a common pathological process initiated by follicular occlusion in apocrine gland-bearing areas has been suggested.¹⁰ The initial cause or primary event in follicular occlusion remains unclear but is likely multifactorial, involving genetics, immune dysregulation, hormonal fluctuation, and lifestyle risk factors, such as tobacco use and obesity.^{2,11} Occlusion of the follicular canal and dilation of the pilosebaceous unit results in rupture of the follicular wall.^{1,2} Follicular contents are thus expelled into the dermis, causing a chemotactic inflammatory response, resulting in an influx of inflammatory cells and abscess formation.^{1,2}

Because of its multifactorial pathogenesis, HS is extremely difficult to treat. Although HS is not an infectious process, antibiotics are a mainstay of treatment, utilized for their potent anti-inflammatory (rather than antimicrobial) action. Management commonly follows the treatment for acne, as HS is also a disorder of the follicular unit. Initial treatment starts with the management of acute symptoms, including pain and drainage, in addition to addressing chronic pathologies, such as tunneling, disfigurement, and progression.¹² Additionally, lifestyle modifications like weight loss and tobacco cessation can be useful interventions for treatment.¹³

Active inflammation may be managed with pharmacological treatment as a primary means of addressing all levels of disease activity. Topical agents, including chlorhexidine, benzoyl peroxide, or zinc pyrithione antimicrobial washes, are common first-line products used to treat HS. Placebo- and active-controlled trials have also provided support for the use of topical clindamycin, 1% solution twice daily with concomitant use of benzoyl peroxide.^{14,15} Intralesional triamcinolone, 40 mg/mL (0.2-2.0 mL) may be used to manage

painful active inflammatory lesions.

Systemic treatments include antibiotics, hormonal therapy, oral retinoids, and immunosuppressants. Typically, the first antibiotic used is doxycycline, 100 mg by mouth twice daily for 3 to 12 months.¹⁶ Combination therapy with clindamycin and rifampin is often prescribed for patients whose symptoms fail to respond to oral tetracyclines or as initial treatment for patients with a significant inflammatory burden.¹⁷ Moderate to severe cases of HS, such as our patient, may benefit from biologic therapy.¹⁸ Cytokines, including tumor necrosis factor alpha (TNF- α), interleukin (IL)-1 β , IL-17, and IL-23 have been implicated as drivers of inflammation in HS, and their targeted inhibition shows promise as a treatment for cases refractory to conventional therapy. In particular, TNF- α inhibitors infliximab and adalimumab are effective and well tolerated for HS, while further evaluation may be needed for other agents.¹⁸

Finally, surgical procedures may be utilized in patients who are not responsive to pharmacologic intervention, or in patients who are contained to one region. While a surgical procedure is typically not recommended, as it commonly worsens the underlying inflammatory pathophysiology, therapeutic procedures include incision and drainage, deroofting, excision, or laser therapy. HS can be a debilitating disease; therefore, awareness and early diagnosis and intervention may help to prevent the progression of disease to advanced and chronic stages.¹²

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