In recent decades the practice of medicine has drifted away from the performance of a physical examination during most patient encounters and evolved toward the more intensive use of history, imaging, and laboratory studies to guide management decisions. For example, it is common for a woman to present to an emergency department with abdominal or pelvic pain and undergo a computerized tomography scan before an abdominal and pelvic examination is performed. Some authorities believe that the trend to reduce the importance of the physical examination has gone way too far and resulted in a reduction in the quality of health care.1,2

Many skin diseases only can be diagnosed by having the patient disrobe and examining the skin. Gynecologists are uniquely positioned to diagnose important skin diseases because, while performing a reproductive health examination, they may be the first clinicians to directly examine the anogenital area and inner thighs. Skin diseases that are prevalent and can be diagnosed while performing an examination of the anogenital region include lichen sclerosus (LS) and hidradenitis suppurativa (HS). The prevalence of each of these conditions is in the range of 1% to 4% of women.3–5

Failure to examine the anogenital area and insufficient attention to the early signs of LS and HS may result in a long delay in the diagnosis.6 In 1 survey, of 517 patients with HS, there was a 7-year interval between the onset of the disease and the diagnosis by a clinician.7 Delay in diagnosis results in increased scarring, which makes it more difficult to effectively treat the disease. In this editorial, I will focus on the pathogenesis, diagnosis, and treatment of HS.

**Diagnosis, presentation, and staging**

Hidradenitis suppurativa (from the Greek, hidros means sweat and aden means glands) is a painful, chronic, relapsing, inflammatory skin disorder affecting the follicular unit. It is manifested by nodules, pustules, sinus tracts, and scars, usually in intertriginous areas. The diagnosis is made by history and physical examination. The 3 cardinal features of HS are 1) deep-seated nodules, comedones, and fibrosis; 2) typical anatomic location of the lesions in the axillae, inguino-crural, and anogenital regions, and 3) chronic relapsing course.8

Disease severity is often assessed using the Hurley staging system:
- stage I: abscess formation without sinus tracts or scarring (FIGURE, page 10)
- stage II: recurrent abscesses with tract formation and scarring, widely separated lesions
Multiple inflammatory nodules or fistulas, classified as Hurley stage I disease. 

Pathogenesis

The pathophysiology of HS is thought to begin with occlusion of the follicle, resulting in follicle rupture deep in the dermis, thereby triggering inflammation, bacterial infection, and scarring. Dermal areas affected by HS have high concentrations of cytokines, including tumor necrosis factor (TNF)-alpha, interleukin (IL)-1-beta, IL-23, and IL-32. Once HS becomes an established process, it is difficult to treat because the dermal inflammatory process and scarring provides a microenvironment that facilitates disease progression. Hence early detection and treatment may result in optimal long-term outcomes.

Treatment

Many recommended treatments for HS have not been formally tested in large randomized trials. A recent Cochrane review identified only 12 high-quality trials and the median number of participants was 27 per trial. Consequently, most treatment recommendations are based on expert opinion. Recommended treatments include smoking cessation, weight loss, topical and systemic antibiotics, antiandrogens, anti-inflammatory biologics (adalimumab and infliximab), and surgery. Smoking cessation and weight loss are strongly recommended in the initial treatment of HS. Bariatric surgery and significant postprocedure weight loss has been reported to cause a reduction in disease activity. Stage I management. For the initial treatment of stage I HS, clindamycin gel 1% applied twice daily to affected areas is recommended. Recommended oral antibiotic treatments include tetracycline 500 mg twice daily for 12 weeks or doxycycline 100 mg or 200 mg given daily for 10 weeks or clindamycin 300 mg twice daily plus rifampicin 600 mg once daily for 10 weeks. These antibiotics have both antimicrobial and anti-inflammatory effects.

Hormonal interventions that suppress androgen production or action may help reduce HS disease activity. For women with HS who also need contraception, an estrogen-progesterin contraceptive may help reduce HS disease activity in up to 50% of individuals. The 5-alpha reductase inhibitor finasteride, at high doses (5 to 15 mg daily), has been reported to reduce HS disease activity. Finasteride is a teratogen, and the FDA strongly recommends against its use by women. Spironolactone, an anti-mineralocorticoid and antiandrogen, at a dose of 100 mg daily has been reported to reduce disease activity in about 50% of treated individuals and is FDA approved for use in women. Among reproductive-age women, spironolactone, which is a teratogen, only should be prescribed to women using an effective form of contraceptive. HS is often associated with obesity and insulin resistance. Metformin 500 mg three times daily has been reported to decrease disease activity.

Stage II or III management. For Hurley stage II or III HS, referral to a dermatologist is warranted. There is evidence that too few people with HS are referred to a dermatologist. For severe HS resistant to oral medications, anti-TNF monoclonal antibody treatment with adalimumab (Humira) or infliximab (Remicade) is effective. Adalimumab is administered by subcutaneous injection and is US Food and Drug Administration (FDA)-approved to treat HS. Following a loading dose, adalimumab is administered weekly at a dose of 40 mg. Infliximab, which is not FDA approved to treat HS, is administered by intravenous infusion at a dose of 5 mg/kg at weeks 0, 2, and 6, and then every 8 weeks.

Surgical management. HS is sometimes treated surgically with...
laser destruction of lesions, punch debridement, or wide excision of diseased tissue.\textsuperscript{24,29} There are no high quality clinical trials of surgical treatment of HS. Punch debridement can be performed using a 5- to 7-mm circular skin punch to deeply excise the inflamed follicle. Wide excision can be followed by wound closure with advancement flaps or split-thickness skin grafting. Wound closure by secondary intention is possible but requires many weeks or months of burdensome dressing changes to complete the healing process. Recurrence is common following surgical therapy and ranges from 30\% with deroofing or laser treatment to 6\% following wide excision and skin graft closure of the wound.\textsuperscript{30}

Physical examination vital to early diagnosis

Delay in diagnosis of an active disease process has many causes, including nonperformance of a physical examination. In a web-based survey of physicians’ experiences with oversight related to the physical examination, 3 problems frequently reported were: nonperformance of any portion of the physical examination, failure to undress the patient to examine the skin, and failure to examine the abdomen and anogenital region in a patient with abdominal or pelvic pain.\textsuperscript{31} Over sights in the physical examination frequently caused a delay in diagnosis and treatment. With both LS and HS, patients may not recognize that they have a skin disease, or they may be embarrassed to show a clinician a skin change they have noticed. Early diagnosis and treatment are essential to achieving a good outcome and make a tremendous difference in the quality of life for the patient. Physical examination is a skill we have learned through diligent study and experience in practice. We can use these skills to greatly improve the lives of our patients.●

References


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