HSV-1 from HSV-2 has prognostic implications, because the latter causes more frequent genital recurrences.

Painless, nontender, indurated ulcers with firm, nontender inguinal adenopathy suggest primary syphilis. If results of dark-field examination and a rapid serologic test for syphilis are initially negative, presumptive therapy should be provided on the basis of the individual's risk. For example, with increasing rates of syphilis among MSM in the United States, most experts would not withhold therapy for this infection pending watchful waiting and/or subsequent detection of seroconversion. Repeated serologic testing for syphilis 1 or 2 weeks after treatment of seronegative primary syphilis usually demonstrates seroconversion.

"Atypical" or clinically trivial ulcers may be more common manifestations of genital herpes than classic vesiculopustular lesions. Specific tests for HSV in such lesions are therefore indicated (Chap. 216). Commercially available type-specific serologic tests for serum antibody to HSV-2 may give negative results, especially when patients present early with the initial episode of genital herpes or when HSV-1 is the cause of genital herpes (as is often the case today). Furthermore, a positive test for antibody to HSV-2 does not prove that the current lesions are herpetic, because nearly one-fifth of the general population of the United States (and no doubt a higher proportion of those at risk for other STIs) becomes seropositive for HSV-2 during early adulthood. Although even "type-specific" tests for HSV-2 that are commercially available in the United States are not 100% specific, a positive HSV-2 serology does enable the clinician to tell the patient that he or she has probably had genital herpes, should learn to recognize symptoms, and should avoid sex during recurrences. In addition, because genital shedding and sexual transmission of HSV-2 often occur in the absence of symptoms and signs of recurrent herpetic lesions, persons who have a history of genital herpes or who are seropositive for HSV-2 should consider the use of condoms or suppressive antiviral therapy, both of which can reduce the risk of HSV-2 transmission to a sexual partner.

Demonstration of *H. ducreyi* by culture (or by PCR, where available) is most useful when ulcers are painful and purulent, especially if inguinal lymphadenopathy with fluctuance or overlying erythema is noted; if chancroid is prevalent in the community; or if the patient has recently had a sexual exposure elsewhere in a chancroid-endemic area (e.g., a developing country). Enlarged, fluctuant lymph nodes should be aspirated for culture or PCR to detect *H. ducreyi* as well as for Gram's staining and culture to rule out the presence of other pyogenic bacteria.

When genital ulcers persist beyond the natural history of initial episodes of herpes (2–3 weeks) or of chancroid or syphilis (up to 6 weeks) and do not resolve with syndrome-based antimicrobial therapy, then—in addition to the usual tests for herpes, syphilis, and chancroid—biopsy is indicated to exclude donovanosis, carcinoma, and other nonvenereal dermatoses. If not performed previously, HIV serology should be standard because chronic, persistent genital herpes is common in AIDS.

TREATMENT ULCERATIVE GENITAL OR PERIANAL LESIONS

Immediate syndrome-based treatment for acute genital ulcerations (after collection of all necessary diagnostic specimens at the first visit) is often appropriate before all test results become available because patients with typical initial or recurrent episodes of genital or anorectal herpes can benefit from prompt oral antiviral therapy (Chap. 216); because early treatment of sexually transmitted causes of genital ulcers decreases further transmission; and because many patients do not return for test results and treatment. A thorough assessment of the patient's sexual-risk profile and medical history is critical in determining the course of initial management. The patient who has risk factors consistent with exposure to syphilis (e.g., a male patient who reports sex with other men or who has HIV infection) should generally receive initial treatment for syphilis. Empirical therapy for chancroid should be considered if there has been an exposure in an area of the world where chancroid occurs or if regional lymph node suppuration is evident. In resource-poor settings lacking ready access to diagnostic tests, this approach to syndromic treatment for syphilis and chancroid has helped bring these two diseases under control. Finally, empirical antimicrobial therapy may be indicated if ulcers persist and the diagnosis remains unclear after a week of observation despite attempts to diagnose herpes, syphilis, and chancroid.

PROCTITIS, PROCTOCOLITIS, ENTEROCOLITIS, AND ENTERITIS

Sexually acquired *proctitis*, with inflammation limited to the rectal mucosa (the distal 10–12 cm), results from direct rectal inoculation of typical STD pathogens. In contrast, inflammation extending from the rectum to the colon (*proctocolitis*), involving both the small and the large bowel (*enterocolitis*), or involving the small bowel alone (*enteritis*) can result from ingestion of typical intestinal pathogens through oral–anal exposure during sexual contact. Anorectal pain and mucopurulent, bloody rectal discharge suggest proctitis or protocolitis. Proctitis commonly produces tenesmus (causing frequent attempts to defecate, but not true diarrhea) and constipation, whereas proctocolitis and enterocolitis more often cause true diarrhea. In all three conditions, anoscopy usually shows mucosal exudate and easily induced mucosal bleeding (i.e., a positive "wipe test"), sometimes with petechiae or mucosal ulcers. Exudate should be sampled for Gram's staining and other microbiologic studies. Sigmoidoscopy or colonoscopy shows inflammation limited to the rectum in proctitis or disease extending at least up into the sigmoid colon in proctocolitis.

The AIDS era brought an extraordinary shift in the clinical and etiologic spectrum of intestinal infections among MSM. The number of cases of the acute intestinal STIs described above fell as high-risk sexual behaviors became less common in this group. At the same time, the number of AIDS-related opportunistic intestinal infections increased rapidly, many associated with chronic or recurrent symptoms. The incidence of these opportunistic infections has since fallen with increasingly widespread coverage of HIV-infected persons with effective antiretroviral therapy. Two species initially isolated in association with intestinal symptoms in MSM—now known as *Helicobacter cinaedi* and *H. fennelliae*—have both been isolated from the blood of HIV-infected men and other immunosuppressed persons, often in association with a syndrome of multifocal dermatitis and arthritis.

Acquisition of HSV, *N. gonorrhoeae*, or *C. trachomatis* (including LGV strains of *C. trachomatis*) during receptive anorectal intercourse causes most cases of infectious proctitis in women and MSM. Primary and secondary syphilis can also produce anal or anorectal lesions, with or without symptoms. Gonococcal or chlamydial proctitis typically involves the most distal rectal mucosa and the anal crypts and is clinically mild, without systemic manifestations. In contrast, primary proctitis due to HSV and proctocolitis due to the strains of *C. trachomatis* that cause LGV usually produce severe anorectal pain and often cause fever. Perianal ulcers and inguinal lymphadenopathy, most commonly due to HSV, can also occur with LGV or syphilis. Sacral nerve root radiculopathies, usually presenting as urinary retention, laxity of the anal sphincter, or constipation, may complicate primary herpetic proctitis. In LGV, rectal biopsy typically shows crypt abscesses, granulomas, and giant cells—findings resembling those in Crohn's disease; such findings should always prompt rectal culture and serology for LGV, which is a curable infection. Syphilis can also produce rectal granulomas, usually in association with infiltration by plasma cells or other mononuclear cells. Syphilis, LGV, and HSV infection involving the rectum can produce perirectal adenopathy that is sometimes mistaken for malignancy; syphilis, LGV, HSV infection, and chancroid involving the anus can produce inguinal adenopathy because anal lymphatics drain to inguinal lymph nodes.

Diarrhea and abdominal bloating or cramping pain without anorectal symptoms and with normal findings on anoscopy and sigmoidoscopy occur with inflammation of the small intestine (enteritis) or with proximal colitis. In MSM without HIV infection, enteritis is often attributable to *Giardia lamblia*. Sexually acquired proctocolitis is most often due to *Campylobacter* or *Shigella* species.