Chapter 7
Vulvovaginal Dermatoses, Lesions, and Masses

Paula C. Brady and Natasha R. Johnson

Differential Diagnosis

Infectious

Herpes simplex virus (HSV)
Herpes zoster, also called shingles
Syphilis
Chancroid
Granuloma inguinale, also called donovanosis
Lymphogranuloma venereum (LGV)
Candida
Folliculitis/impetigo
Group A β-hemolytic streptococcus

(continued)

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P.C. Brady, *Handbook of Consult and Inpatient Gynecology*,
DOI 10.1007/978-3-319-27724-0_7

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Scabies
Human Immunodeficiency Virus (HIV)
Tuberculosis

Noninfectious

Allergic or irritant contact dermatitis
Hidradenitis suppurativa
Lichen planus
Lichen sclerosis
Lichen simplex chronicus
Lipschutz ulcers
Post viral genital ulcers (aphthous ulcers)
Atrophic vaginitis
Inflammatory bowel disease
Pyoderma gangrenosum
Stevens-Johnson syndrome (SJS)
Toxic epidermal necrolysis (TEN)
Erythema multiforme
Bullous pemphigoid
Mucous membrane pemphigoid
Pemphigus vulgaris
Linear immunoglobulin A (IgA) dermatosis
Paraneoplastic pemphigus
Psoriasis
Eczema
Vulvar malignancy
Hematologic malignancy
Behçet's syndrome
Periodic fever, aphthous stomatitis, pharyngitis, and cervical adenitis (PFAPA)

Inguinal or Perineal Abscess

Chancroid
Lymphogranuloma venereum (LGV)
Hidradenitis suppurativa
Bartholin's gland or Skene's glands abscesses, urethral diverticulum

Bullae/Blisters/Desquamation

Stevens-Johnson syndrome (SJS)
Toxic epidermal necrolysis (TEN)
Erythema multiforme
Bullous pemphigoid
Mucous membrane pemphigoid
Pemphigus vulgaris
Linear IgA dermatosis
Paraneoplastic pemphigus
Allergic or irritant contact dermatitis

Ulcers/Erosions/Crusted Lesions

Herpes simplex virus (HSV)
Herpes zoster, also called shingles
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Chancroid
Granuloma inguinale, also called donovanosis
Lymphogranuloma venereum (LGV)
Candida
Allergic or irritant contact dermatitis
Lichen planus
Lichen sclerosis
Lichen simplex chronicus

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Lipschutz ulcers
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Linear IgA dermatosis
Paraneoplastic pemphigus
Psoriasis
Eczema
Vulvar malignancy
Hematologic malignancy
Behçet's syndrome
Periodic fever, aphthous stomatitis, pharyngitis, and cervical adenitis (PFAPA)
HIV
Tuberculosis

Fissures

Candida
Group A β-hemolytic streptococcus
Scabies
Allergic or irritant contact dermatitis
Hidradenitis suppurativa
Lichen planus
Lichen sclerosis

(continued)

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Lichen simplex chronicus
Eczema
Desquamative inflammatory vaginitis (DIV)
Inflammatory bowel disease

Perineal Masses

Bartholin's gland cyst/abscess
Skene's gland cyst
Urethral diverticulum
Müllerian and Gartner (mesonephric) duct cyst
Canal of Nuck cyst
Epidermoid inclusion cyst
Condyloma acuminata
Acrochordons (skin tags)
Less common: lipomas, neurofibromas, vulvovaginal endometriosis, and vulvar leiomyomas

When You Get the Call Ensure the patient is in a private room on bed or stretcher equipped for gynecologic exams (i.e., with stirrups).

When You Arrive Review the patient's vital signs, making note of signs of systemic illness, including fever or tachycardia.

History

The history of the present illness is crucial when approaching perineal lesions, in order to determine exposures to sexually transmitted infections, irritants, or any other instigating factors, including trauma. Review the evolution of the current lesion, including whether onset was rapid or gradual, and whether an ulceration began as a blister or was preceded by tingling,

burning, pruritis, or pain. Ask patients to describe symptoms currently accompanying the chief complaint, including itching, pain, purulent drainage from a lesion, or vaginal discharge.

Review whether the patient has already tried any treatments, which may lend insight into the original etiology or suggest a secondary overlying contact dermatitis if topical treatments have been applied. Inquire regarding pubic hair removal practices, as hair removal may introduce contact irritants and infection. Review whether the patient uses scented soaps, douches, lubricants for intercourse, condoms, or spermicides. Review whether the patient has new sexual partners, partners who have had similar lesions, or recent travel.

A review of systems should include constitutional complaints such as fever, myalgias, malaise, preceding or concomitant infections, or complaints in any other organ systems (including pharyngitis or gastrointestinal complaints). Review whether the patient has, or has ever had, oral or eye ulcerations.

A full sexual history is vital, including the gender and number of sexual partners, types of sexual contact (oral, vaginal, anal), and any history of sexually transmitted infections. High-risk sexual behaviors or drug or alcohol use may indicate higher risk of exposure to sexually transmitted infections.

A full medical history should be obtained, including any current immunosuppression, HIV, or autoimmune or inflammatory bowel disease. A family history should also include inflammatory bowel disease, Behçet's syndrome, or other autoimmune diseases.

Physical Examination

As indicated, examine for patient's mouth for gingival inflammation and lesions of the buccal mucosa, and note any other skin findings on extensor or flexor surfaces, intertriginous regions, and the hands and feet.

A physical exam of the vulva and vagina should begin with observation, carefully noting the anatomic location of any

lesions and relation to other structures. The distribution of erythema or other skin changes should be noted, as well as the presence or absence of normal vulvar architecture (such as shortening or loss of labia minora and adhesion of the clitoral hood to the glans) and change in pigmentation (hypo- or hyperpigmentation). Lesions should be described in detail, including size, whether lesions (vesicles, ulcers) are single or multiple, and accompanying erythema and tenderness. Note whether masses, cysts, or abscesses are painful, erythematous, mobile, fluctuant, or firm and whether any purulent material can be expressed. Assess for lymphadenopathy, including painful fluctuant enlargement of lymph nodes called buboes, particularly in the inguinal region [1]. Of note, local and systemic infections, such as lower extremity infections, can also lead to inguinal lymphadenopathy. An adult with vulvar ulcerations should also have a speculum exam to assess whether the ulcerations are also present in the vagina, which often require additional, specific management.

Diagnosis

Diagnostic testing must be tailored to the specific cause. The symptoms of vaginal discharge and/or odor and the presence of abnormal discharge on examination warrant pH testing, preparation of a wet mount (microscopic evaluation of vaginal discharge prepared with normal saline and 10 % potassium hydroxide, separately), and vaginal culture. Elevated pH (greater than 4.5) may be seen in patients with bacterial vaginosis, trichomoniasis, and atrophic vaginitis [2]. Testing for suspected sexually transmitted infections—whether serologic or from the lesion—is dictated by presentation, prevalence of disease, and the patient's risk factors. Sexually transmitted infections resulting in genital lesions are detailed below.

For any ulcerations—suspected lichen planus, lichen sclerosis, malignancy, or other dermatologic conditions including blistering diseases or pyoderma gangrenosum—biopsy should be considered. Recurrent ulcerations require more

in-depth workup for autoimmune causes such as inflammatory bowel disease or Behçet's syndrome. Dermatology consultation should be considered for extensive ulcerations and desquamation.

Management

General symptom management applies to several pathologies. In general, erosions should be managed with sitz baths (sitting in warm water for 5–10 min, two to three times per day) with application of topical lidocaine 2 % jelly for analgesia and/or plain petrolatum as a barrier [3]. Patients should be counseled to avoid contact irritants, including scented soaps, and to wash the vulva gently with water, without soap. If patients report pruritis, they may use hydroxyzine or other sedating antihistamine as a sleep aid and to limit scratching.

Management approaches for specific etiologies—infectious and noninfectious—of vulvovaginal lesions are detailed below. Management of traumatic vulvovaginal injuries, including straddle injuries and vulvar hematomas, are discussed in Chap. 11, Vulvovaginitis and Vaginal Bleeding in Pediatric and Adolescent Patients.

Infectious

In general, when infectious causes of genital lesions are suspected, the World Health Organization offers guidelines for management of vulvovaginal ulcerations designed to avoid delays in treatment, particularly useful in low-resource settings lacking extensive diagnostic resources.

For patients with genital ulcers, the WHO recommends treatment for syphilis and chancroid, in addition to offering treatment for HSV-2 in areas in which prevalence is 30 % or more [1]. Depending on local disease prevalence, treatment for granuloma inguinale and/or lymphogranuloma venereum

is recommended as well. Any fluctuant lymph nodes should be aspirated. Patients' symptoms should be reassessed in 7 days or less. Partner treatment should be addressed, and HIV testing should be offered when relevant and available. Patients with genital buboes without ulcers should be treated for chancroid and lymphogranuloma venerum.

Treatment of sexual partners will be reviewed for each infection below. In general, if an infectious etiology of genital lesions is suspected, patients should avoid unprotected intercourse until all symptoms and lesions have resolved or 1 week after single-dose treatment [1].

Herpes Simplex Virus (HSV)

Herpes simplex virus is a chronic infection that cannot be cured, but can be managed with antiviral medications. Two serotypes are most clinically relevant: HSV-1 classically causes oral lesions, while HSV-2 is associated with anogenital lesions. Increasingly, HSV-1 is also associated with anogenital lesions [4]. Patients present with vesicles that progress to painful ulcerations, dysuria, dyspareunia, and bleeding and can have inguinal lymphadenopathy (Fig. 7.1).

Analysis of data from the National Health and Nutrition Examination Surveys (NHANES) from 2007 to 2010 reported a prevalence of HSV-2 among women aged 14–49 years in the United States of 20.4 %; the prevalence is 49.5 % among non-Hispanic black or African American women [6].

In patients presenting with ulcerations suspicious for HSV, cell culture and polymerase chain reaction (PCR) are preferred for diagnosis of HSV. Viral culture can be obtained by swabbing the base of an unroofed vesicle [7]. Culture, however, has low sensitivity, particularly in the diagnosis of recurrent lesions. PCR has much higher sensitivity than culture, with more rapid results and less sensitivity to collection and transport conditions [8]. PCR can also differentiate between HSV serotypes [4]. Direct fluorescent antibody (DFA) testing or enzyme-linked

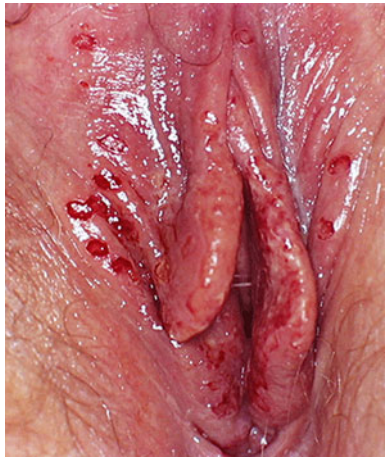

Fig. 7.1 Vulvar herpes simplex (Reprinted from Danby and Margesson [5], with permission of John Wiley & Sons, Inc.)

immunosorbent assays (ELISA) are also available, generating fast results, with moderate sensitivity [8]. Tzanck smears, in which the lesion base is scraped and examined for cytologic changes, lack sensitivity and specificity and cannot distinguish between HSV and varicella-zoster virus (VZV), which causes varicella (chickenpox) and herpes zoster (shingles) [9, 10].

Serologic testing is commonly performed to assess for HSV-specific glycoproteins, allowing for differentiation between HSV serotypes [4]. Serology, however, cannot reliably differentiate between past and present infections; IgM, which may be used as a marker of an acute infection for many other viruses, may be present even in recurrent HSV episodes [11]. Serologies may be clinically useful in symptomatic patients with negative HSV cultures or patients with recurrent lesions requiring diagnostic confirmation. HSV serologies can also be used to assess patients with HIV, at risk for acquiring HIV, or to assess the sexual partner of a patient with known herpes [4].

For primary (first-ever) HSV outbreaks, recommended treatment regimens are (1) acyclovir, 400 milligrams (mg) PO every 8 h for 7–10 days; (2) acyclovir 200 mg PO five times per day for 7–10 days; (3) famciclovir, 250 mg PO every 8 h for 7–10 days; or (4) valacyclovir 1 g PO every 12 h for 7–10 days [4].

For adults not on suppressive therapy with an episode of recurrence of genital herpes, the recommended regimen is (1) acyclovir 400 mg PO every 8 h for 5 days, (2) acyclovir 800 mg PO every 12 h for 5 days, (3) acyclovir 800 mg PO every 8 h for 2 days, (4) famciclovir 125 mg PO every 12 h for 5 days, (5) famciclovir 1000 mg PO every 12 h for 1 day, (6) famciclovir 500 mg once, followed by 250 mg every 12 h for 2 days, (7) valacyclovir 500 mg PO every 12 h for 3 days, or (8) valacyclovir 1000 mg PO once per day for 5 days.

Suppressive therapy reduces outbreaks by 80 % [12]. The regimen to prevent recurrent outbreaks in adults is (1) acyclovir 400 mg PO every 12 h, (2) famciclovir 250 mg PO every 12 h (3) valacyclovir 500 mg PO once daily, or (4) valacyclovir 1 g orally once daily [4].

Initial outbreaks in children should be treated with acyclovir, 40–80 mg per (kg) per day divided into 3–4 doses, administered for 5–10 days (maximum 1 g per day) [13]. In patients over 12 years of age, acyclovir is instead dosed as 1000–1200 mg/day divided into 3–5 doses for 7–10 days. Recurrent infections in patients over 12 years can also be treated with acyclovir: (1) 1000 mg divided into 5 doses for 5 days, (2) 1600 mg divided into 2 doses for 5 days, and (3) 2400 mg divided into 3 doses for 2 days [13].

IV acyclovir can be used for patients with severe outbreaks, particularly disseminated HSV. In adults, the recommended IV regimen is 5–10 mg/kg every 8 h until clinical improvement is obtained, followed by oral therapy for at least 10 days total [4]. For children over 12 years of age, IV acyclovir is dosed as 15 mg/kg per day divided into three doses for 5–7 days [13].

For symptomatic relief, patients may need to urinate into a warm water bath. Urinary retention may require temporary

Foley catheter placement. Topical, oral, and IV analgesics may be required for adequate pain control.

Counseling a patient diagnosed with HSV is crucial. Sexual transmission can occur during asymptomatic periods, though transmission is more common when patients have prodromal symptoms or lesions [12]. Prophylaxis and condom use may reduce, but not eliminate, the risk of transmission. Infection with HSV-2 may increase the risk of contracting HIV.

Pregnant women with partners known to have HSV should be counseled to avoid exposure to HSV in their third trimester (either intercourse with a patient with HSV-2 or genital HSV-1 or receptive oral sex with a partner with oral HSV-1). Suppressive therapy should be prescribed for pregnant women with genital HSV from 36 weeks of gestation onward [14]. The risk of neonatal infection should be explained.

Sex partners should be offered counseling and evaluation and can be offered HSV type-specific serologic testing.

Herpes Zoster

Zoster, also called shingles, represents a reactivation of the varicella-zoster virus, which causes a diffuse vesicular rash ("chickenpox") when a patient is first infected and remains latent in sensory ganglia roots, often reactivating in the context of immunosuppression or advancing age [15]. Zoster appears in a dermatomal distribution, rarely on the vulva, as vesicular lesions that break to form a crust (Fig. 7.2) [5]. Patients may report prodromal symptoms of pain or burning before lesions appear.

The diagnosis is usually clinically apparent; however, if necessary, PCR can be performed from lesion scrapings [16]. Direct fluorescent antibody (DFA) testing can also be performed from lesion scrapings, with rapid results but less sensitivity than PCR. Serology is less helpful, as IgM may represent a primary infection, reinfection, or reactivation [15].

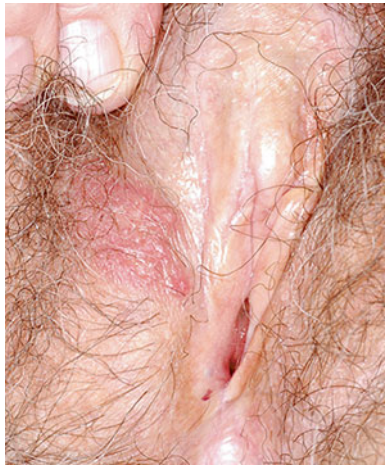

FIG. 7.2 Vulvar herpes zoster (Reprinted from Danby and Margesson [5], with permission of John Wiley & Sons, Inc.)

Recommended treatments, which should be initiated within 72 h of symptom onset, are (1) acyclovir 800 mg PO five times daily for 7–10 days, (2) famciclovir 500 mg PO three times daily for 7 days, or (3) valacyclovir 1000 mg three times daily for 7 days [15].

Patients with zoster, particularly those over age 50, are at risk of developing post-herpetic neuralgia. Antiviral medications reduce the risk of post-herpetic neuralgia, but additional medications may further mediate this risk and reduce pain [15]. Patients should be assessed for contraindications to any of these medications before they are started. Gabapentin 300 mg PO at bedtime or 100–300 mg PO every 8 h can be added and uptitrated to a total dose of 3600 mg per day; the major side effect is sedation. Tricyclic antidepressants such as nortriptyline can be initiated at a dose of 25 mg PO at bedtime and uptitrated by 25 mg every 2–3 days to a daily maximum of 150 mg. Tricyclic antidepressants are associated with sedation, constipation, dry mouth, and urinary retention; a starting dose of 10 mg can be considered in elderly patients. A course of oral prednisone may also

address pain; a recommended regimen is 60 mg PO daily for 7 days and then tapered.

Syphilis

Syphilis is a sexually transmitted infection caused by *Treponema pallidum*, a spirochete (spiral-shaped bacterium). The primary phase is usually characterized by a single painless ulcer (chancre) of the mouth, anus, cervix, or labia, accompanied by nontender lymphadenopathy [17]. The second stage of syphilis, which may overlap with the first, consists of a skin rash (often on the feet and hands), large raised lesions in the mouth, underarm, or groin called condyloma lata, fever, lymphadenopathy, pharyngitis, or myalgias [18]. Involvement of the central nervous system ("neurosyphilis") can occur at any stage. Syphilis is termed "latent" when a patient presents without symptoms but has positive serologic testing; early latent is defined as latency of less than 1 year. One-third of untreated cases will progress to involvement of the cardiovascular system and/or development of granulomatous lesions of any organ system, though the skin, skeletal system, and liver are the most common sites [17, 18].

Congenital syphilis is increasingly uncommon due to prenatal screening; however this should also be considered in infants presenting low birth weight, jaundice, anemia, and hepatosplenomegaly [19]. With regard to genital dermatologic findings, infants with congenital syphilis may present with papules and deep fissures in the perianal region. Infants may also have diffuse red papulo-squamous rashes, particular on the buttocks, palms, and soles, which may develop into bullae. The infant and mother should have serologic screening if congenital syphilis is suspected.

The definitive method of diagnosis for syphilis is darkfield examination and/or PCR of lesion material, which may not be available in all settings [18]. Serologic screening for syphilis typically begins with nontreponemal testing: rapid plasma reagin (RPR) or venereal disease research laboratory

(VDRL) [17]. These tests may result in false positives in patients with HIV, autoimmune disease, intravenous drug use, pregnancy, and older age [18].

Positive nontreponemal testing is confirmed with enzyme immunoassays or more specific treponemal testing with a fluorescent treponemal antibody–absorbed (FTA-ABS) test or the *T. pallidum* particle agglutination (TP-PA) test. Conversely, some centers have begun screening with treponemal tests, which, if positive, should be confirmed with a nontreponemal test. Of note, both treponemal and nontreponemal tests may not be reactive in early primary infection.

Examination of CSF is recommended in any patients with neurologic or ocular symptoms, for the diagnosis of neurosyphilis. Patients with confirmed syphilis should be tested for HIV.

Consider medicine or infectious disease consultation for management of syphilis treatment. Primary, secondary and early latent syphilis should be treated with one dose of benzathine penicillin G 2.4 million units intramuscularly (IM). Alternatives for penicillin allergies are (1) doxycycline 100 mg PO twice daily for 14 days, (2) tetracycline 500 mg PO four times daily for 14 days, and (3) ceftriaxone 1–2 g IM or IV daily for 10–14 days, though data is more limited for this last option [4]. Pregnant women must be treated with penicillin, even if desensitization is needed for penicillin allergy. Patients require serologic follow-up; nontreponemal titers are expected to fall fourfold in 6–12 months.

Infants and children diagnosed with syphilis should be managed by pediatric infectious disease specialists and should be evaluated for sexual abuse [4]. In children, primary, secondary, or early latent syphilis of less than 1 year is treated with benzathine penicillin 50,000 units/kg IM in a single dose (maximum 2.4 million units) [4]. In pediatric patients, latent syphilis of greater than 1 year or of unknown duration is treated with benzathine penicillin 50,000 units/kg IM for three doses at 1 week intervals (maximum total 7.2 million units). The management of more advanced syphilis—latent

and tertiary (neurosyphilis)—in children and adults will not be covered here.

Sexual partners are thought to be exposed only if the infected patient has mucocutaneous lesions, however partners exposed at any stage should be evaluated [4]. Sexual partners exposed within 3 months of the patient's diagnosis of primary, secondary, or early latent syphilis or late latent syphilis with high nontreponemal titers (>1:32) should be treated for presumed early syphilis even if serologies are negative. Partners exposed more than 90 days before the patient's diagnosis of primary, secondary, or early latent syphilis should be tested or simply treated if testing is not available. Long-term partners of patients with late latent syphilis should be tested and treated accordingly.

Chancroid

Chancroid is a sexually transmitted infection caused by *Haemophilus ducreyi*, a gram-negative bacterium [20]. It is more common in developing countries and in males. Patients may present with significant, painful lymphadenopathy, which become fluctuant, called buboes [1]. Sharply delineated painful ulcers develop, with little surrounding erythema; multiple small ulcers may coalesce into a single large ulcer. The annual incidence rate in the United States has been falling over time; only four cases were reported in the United States in 2013 [21].

An accurate diagnosis of chancroid relies on culture of *H. ducreyi* from the lesion. In addition, aspiration and culture of suppurative lymph nodes should be considered. Since the organism can be difficult to grow, the use of selective and supplemented media is necessary [20]. PCR can also be used and is more sensitive, but is not widely available [22]. For therapeutic purposes, large fluctuant buboes can be aspirated or incised and packed for symptomatic relief [23].

The Centers for Disease Control and Prevention (CDC) recommend four possible regimens: (1) azithromycin (1 g

PO in a single dose), (2) ceftriaxone (250 mg IM in one dose), (3) ciprofloxacin (500 mg PO every 12 h for 3 days), or (4) erythromycin base (500 mg PO every 8 h for 7 days) [4]. Ciprofloxacin should not be used in pregnancy. Children weighing less than 45 kg should be treated with (1) ceftriaxone (50 mg/kg IM up to 250 mg) in a single dose or (2) azithromycin (20 mg/kg PO up to 1 g) in a single dose [13]. Erythromycin is an alternative option; ciprofloxacin is not approved by the US Food and Drug Administration for people younger than 18 years of age.

Patients should be seen within a week to ensure symptom improvement, though complete healing of the ulcer may take longer. If symptoms have not improved, consider coinfection with another sexually transmitted infection or HIV, nonadherence with treatment, or bacterial resistance [4]. Sexual partners should be treated if they had contact with a patient infected with chancroid within the 10 days before the patient developed symptoms [4].

Granuloma Inguinale

Granuloma inguinale, also called donovanosis, is caused by the gram-negative bacterium *Klebsiella granulomatis* [4]. Occurring rarely in the United States, infections are most commonly identified in Papua New Guinea, parts of southern Africa, India, French Guyana, Brazil, and Australia [24].

Genital and perineal ulcers are slowly progressive, painless, and highly vascular, without associated lymphadenopathy ("pseudobuboes"); subcutaneous granulomas may be present. Extensive fibrosis can result in adhesions and sinus tracts [13]. The infection can extend into the pelvis, with dissemination to the intraabdominal organs, bones, or mouth.

K. granulomatis is difficult to culture [4]. The diagnosis can be made by visualization of dark-staining Donovan bodies on tissue crush preparation or histologic examination of biopsy specimens [4]. No FDA-cleared molecular tests for the detec-

tion of *K. granulomatis* DNA exist. *Haemophilus ducreyi* cultures should be obtained to exclude chancroid.

First-line treatment is azithromycin, 1 g orally once per week or 500 mg daily for at least 3 weeks and until all lesions have completely healed [1, 4]. Alternatives include the following, all for 3 weeks or until lesions have completely healed: (1) doxycycline (100 mg PO every 12 h), (2) ciprofloxacin (750 mg PO every 12 h), (3) erythromycin base (500 mg PO every 6 h), or (4) trimethoprim-sulfamethoxazole (one double-strength tablet (160/800 mg) PO every 12 h) [4]. Erythromycin and azithromycin are the first-line treatment in pregnancy. Children less than 45 kg can receive azithromycin (20 mg/kg PO in a single dose, maximum 1 g). Of note, doxycycline is not approved for children less than 8 years [13]. Gentamicin can be added to adult and pediatric regimens if improvements are not noted within the first few days of treatment.

The utility of empiric partner treatment in the absence of symptoms is unclear. Individuals who had sexual contact within 60 days of a patient's onset of symptoms should be examined [4].

Lymphogranuloma Venereum (LGV)

LGV is caused by *Chlamydia trachomatis* serotypes L1, L2, and L3 [25]. Once rare, LGV is becoming more common internationally and often coincides with HIV infection [26]. Patients most often present with an ulcer at the inoculation site. Patients subsequently develop tender lymphadenopathy, which may become fluctuant, called buboes [4]. Anal exposures, if untreated, can result in fistulas or strictures resembling inflammatory bowel disease [13].

Swabs of ulcerations or aspirates of inguinal buboes can be tested for *C. trachomatis*, using culture, direct immunofluorescence, or nucleic acid amplification testing. PCR, which is not widely available for this purpose, can differentiate LGV from non-LGV *C. trachomatis*. Serology can also be sent;

complement fixation titers above 1:64 are suggestive of infection with LGV [4].

Large and/or painful inguinal buboes might require aspiration through intact skin or incision and drainage. Aspiration or drainage may prevent the formation of inguinal/femoral ulcerations. Treatment with doxycycline (100 mg PO every 12 h for 21 days) is preferred. Alternatively, erythromycin base (500 mg PO every 6 h for 21 days) can be used [4]. Based on limited data, azithromycin (1 g orally once weekly for 3 weeks) is likely effective. Doxycycline should be avoided in pregnancy. Pediatric patients over 8 years of age should be treated with doxycycline (200 mg/day in two divided daily doses) for 21 days. As an alternative, erythromycin 50 mg/kg/day divided into four doses per day (up to 500 mg per dose) for 21 days can be used [13].

Individuals who had sexual contact within 60 days of a patient's onset of symptoms should be tested for chlamydia and presumptively treated with azithromycin (1 g PO in a single dose) or doxycycline (100 mg orally twice a day for 7 days) [4].

Candida

Vulvovaginal candidiasis, most commonly caused by *Candida albicans*, is associated with pruritis, dysuria, dyspareunia, and/or thick, white vaginal discharge. Patients may also present with swelling, erythema, vulvar fissures, and excoriations (Fig. 7.3) [5, 27]. Red plaques may be present within skin folds, with satellite lesions; pustules may form erosions [2].

Risk factors for vulvovaginal candidiasis include systemic antibiotic or steroid use, immunosuppression, and diabetes mellitus. A microscope slide of the patient's vaginal discharge prepared with saline and 10 % potassium hydroxide may reveal candida buds and hyphae and provide a rapid diagnosis; a vaginal culture can also be collected. Initial treatment of uncomplicated vulvovaginal candida entails fluconazole 150 mg PO in a single dose; patients may require another dose in 72 h if they continue to have symptoms.

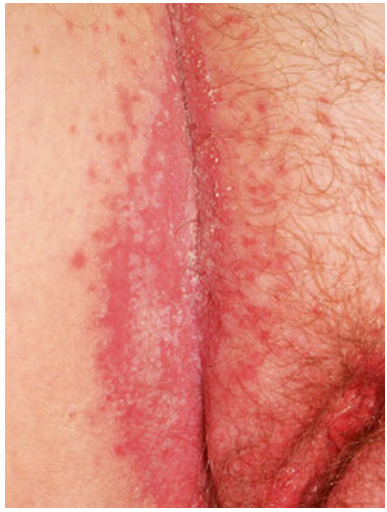

Fig. 7.3 Candida infection of the vulva (Reprinted from Pipkin [3], with permission from Elsevier)

Topical and intravaginal azoles are also acceptable and are available over the counter. Concomitant dermatitis may require short-term medium potency topical steroids. Combined topical antifungal and steroid preparations are available, such as a cream combining clotrimazole and betamethasone dipropionate (Lotrisone®, Merck, Whitehouse Station, NJ).

Folliculitis/Impetigo

Folliculitis can occur on the vulva or mons, particularly with hair removal techniques. These may be infected, developing into a superficial infection called impetigo, often caused by *Staphylococcus aureus* or *Streptococcus pyogenes* [3]. Yellow crusts and/or bullae may be present. Treatment is topical mupirocin 2 % ointment or an oral antibiotic with adequate skin flora coverage, such as cephalexin [3].

Group A β-Hemolytic Streptococcus

Group A β-hemolytic streptococcus vaginitis is more common in children than adults; it is associated with a red vaginal and/or perineal rash with or without fissures or purulent discharge. Vaginal culture is needed for diagnosis, and treatment is with penicillin VK (500 mg PO four times daily for 10–14 days) or clindamycin cream (2 % per vagina for 7–10 days) [5].

Scabies

Scabies are mites transmitted by skin-to-skin contact. Lesions are most often present on the hands, particularly between the fingers, and at the elbow but can also be present on the vulva or buttocks [28]. Classic scabies lesions are raised tracts in which the parasites burrow [29]. Patients can also present with blisters, nodules, or excoriations, rarely with lymphadenopathy. Diagnosis is most commonly made by biopsy or scraping, to assess for eggs. Treatment is permethrin cream (5 %) applied once from the neck down and removed 8 h later. Close contacts are generally counseled for treatment, and bedding and clothing should be washed in hot water [29].

Noninfectious

Allergic or Irritant Contact Dermatitis

Contact dermatitis can result in severe vulvar pruritis; fissures and lichenification can result from scratching. More severe reactions may present with vesicles and bullae, which may result in erosions (Fig. 7.4) [3]. Irritant contact dermatitis develops with exposure, while allergic contact dermatitis can occur at an interval after exposure [30]. Common irritants are related to hygiene, including sweat, urine, soaps and detergents, excessive bathing including douches, hair removal

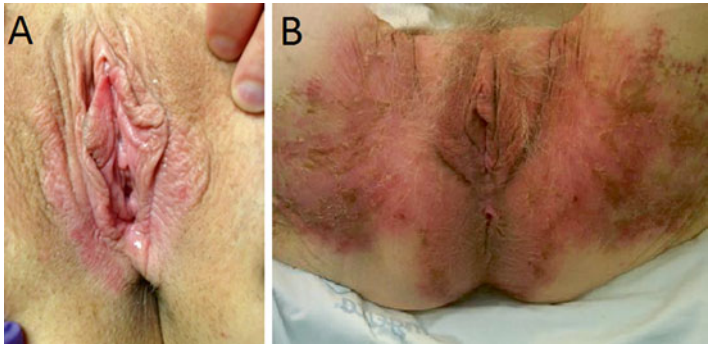

Fig. 7.4 Irritant dermatitis. (**a**) Irritant dermatitis from Dial soap. (**b**) Irritant dermatitis from urinary incontinence with candidal superinfection (Photos courtesy of Natasha R. Johnson, MD)

(shaving, waxing, chemicals), baby wipes, and sanitary pads. Condoms (latex), spermicide, and sperm are also contact irritants [30].

A full history may reveal a possible contact irritant. On physical exam, the dermatitis will be in the distribution of the exposure, though contact dermatitis may coincide with other dermatoses or infections. Excoriations may be seen. Patients should also be assessed for concomitant bacterial or fungal infections. Genital culture for yeast should be performed. Biopsy should be considered for ulcerative lesions to rule out dermatoses. The goal of therapy is to identify the contact irritant and eliminate it. If the contact irritant is difficult to identify, the patient can be referred for patch testing.

Patients should be educated to wear loose-fitting, cotton clothing, avoid use of soap on the vulva, and eliminate douching. Assess hygienic practices and inquire about incontinence of urine and feces, which are irritants. Petrolatum can be applied as a barrier over particularly irritated or ulcerated skin [32]. Patients with erosions or ulcerations should perform sitz baths (sitting in warm water for 5–10 min), twice per day until lesions improve. Patients can use antihistamines to alleviate itching, including loratadine during the day and/or hydroxyzine, a sedating antihistamine, in the evening.

Depending on the severity of the lesions, patients may require topical corticosteroids [32]. For mild irritation, hydrocortisone 2.5 % ointment applied twice each day may be sufficient. Moderate irritation can be treated with triamcinolone acetonide 0.1 % ointment, while high-potency topical steroids may be required for severe dermatitis (clobetasol propionate ointment 0.05 %). Topical steroids should be used twice daily until lesions have resolved. Patients may require oral steroids for severe dermatitis.

Atrophic Vaginitis

In postmenopausal women, the vaginal mucosa becomes thin and dry and increasingly sensitive to irritants, trauma, and bacterial overgrowth. In severely atrophic vaginitis, purulent discharge and fissures of the vulva and vestibule may develop. Vaginal estrogen creams, tablets, and an estradiol vaginal ring are equally effective in treating vulvovaginal atrophy [34].

Lichen Planus

Lichen planus is an uncommon inflammatory condition of the skin, nails, and genitals [35]. The classic mucosal involvement is Wickham striae—reticulate white striations—most often found on the buccal mucosa [35]. Erosive lesions can also appear in the mouth [36]. Up to one-quarter of patients with lichen planus have genital involvement. Lichen planus is characterized by pruritic purple papules of the vulva; erosive lichen planus is associated with deep painful, friable erosions leading to burning, dyspareunia, and vaginal discharge (Fig. 7.5) [37]. Cases involving the vulva and vagina are most commonly erosive, leading to painful desquamation and ulceration, frequently resulting in scarring and destruction of architecture [38].

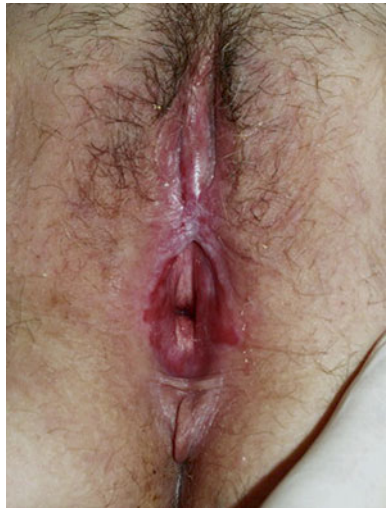

FIG. 7.5 Vulvovaginal lichen planus (Reprinted from Pipkin [3], with permission from Elsevier)

Nonerosive lichen planus is likely to be controlled with high-potency topical corticosteroids and emollients, such as Vaseline® (Unilever, London, England). Treatment can be started with betamethasone valerate 0.1 % ointment twice per day for 6 weeks and then as needed [39]. Erosive vulvar lichen planus is more resistant to treatment; clobetasol propionate 0.05 % ointment can be used daily for 3 months, after which steroid strength and frequency are decreased. Topical steroids are first-line treatment, after which topical tacrolimus, systemic steroids, tacrolimus, cyclosporine, or other immunosuppressants may be considered [38, 39].

When vaginal disease is present, steroid suppositories, such as hydrocortisone acetate 25 mg suppositories twice per day for 2 months, can be used [40]. As with vulvar disease, steroid frequency can be tapered to maintenance usage (once or twice weekly) once improvement has be achieved. Patients with vaginal erosions are at risk for synechiae and vaginal narrowing; use of vaginal dilators with steroid therapy can be considered to maintain vaginal patency [40].

Women with erosive vulvovaginal lichen planus are at increased risk of vulvar squamous cell carcinoma (estimated at 2.4 %) and warrant regular clinical exams [41].

Lichen Sclerosis

Lichen sclerosis is a chronic inflammatory destructive condition of the perineal and perianal skin. The incidence of lichen sclerosis is bimodal, occurring most commonly in prepubertal girls or postmenopausal women [42]. The most common presenting complaint in vulvar pruritis; patients may also report dyspareunia or dysuria. The vagina is spared—unlike in lichen planus—though women may have narrowing of the vaginal introitus. Exam will often reveal vulvar skin that is thin and whitened (white crinkling or "cigarette paper"), often in a "figure of eight" shape around the vaginal introitus and anus, with loss of architecture of the labia minora and phimosis of the clitoral hood to the glans (Fig. 7.6) [38, 43].

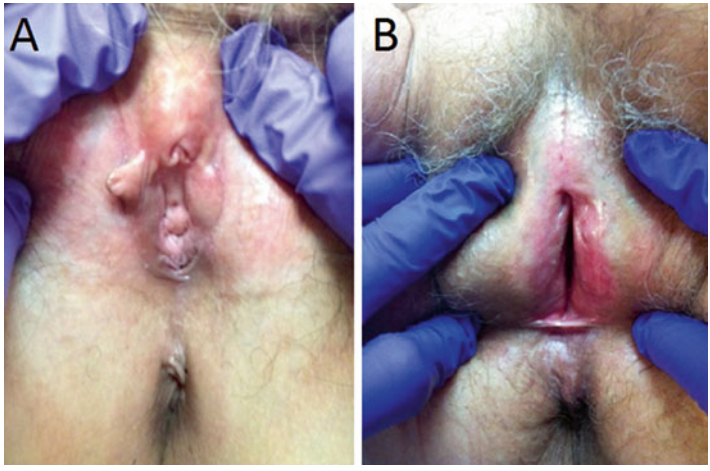

FIG. 7.6 Lichen sclerosis. (**a**) Mild to moderate lichen sclerosis, associated with loss of architecture of the labia minora. (**b**) Severe lichen sclerosis, with loss of labia minora and phimosis of the clitoral hood (Photos courtesy of Natasha R. Johnson, MD)

Patients may also have vulvar fissures or erosions, particularly due to scratching.

Biopsy is recommended in postpubertal women to rule out other causes of pruritis and erosions. Otherwise, diagnosis is clinical. Lichen sclerosis is most often managed with high-potency topical corticosteroids, such as clobetasol propionate 0.05 % ointment one to two times per day for 4–6 weeks, after which frequency can be weaned to maintenance (once or twice weekly) [39, 42]. Patients may require increased frequency of clobetasol for flares.

Approximately 2–5 % of women with lichen sclerosis develop vulvar squamous cell carcinoma and warrant regular clinical exams [39].

Desquamative Inflammatory Vaginitis (DIV)

DIV is a painful vaginitis not associated with infection. Patients report burning, discharge, and dyspareunia. On physical examination, primarily the vagina is involved, though the vestibule can be affected, with erythema and fissures [5]. Microscopic examination of the vaginal discharge reveals parabasal cells (round cells not usually present on microscopy) and increased white blood cells (>10 per high-power field). Treatment is with hydrocortisone acetate suppositories (3–5 g at night for 14 days then tapered) or intravaginal clindamycin (2 % cream vaginally for 2–4 weeks).

Postviral and Lipschutz Ulcers

Genital ulcerations have been noted after infections with cytomegalovirus, influenza A, and Epstein-Barr Virus [44, 45]. These ulcerations are likely often classified as idiopathic, when patients are not tested for viral triggers. EBV is most commonly associated with genital ulcerations, likely through direct cytotoxicity in vulvar epithelium as well as triggering an immunologic response that manifests as vulvar ulcers [44]. CMV and EBV-related ulcers often have more

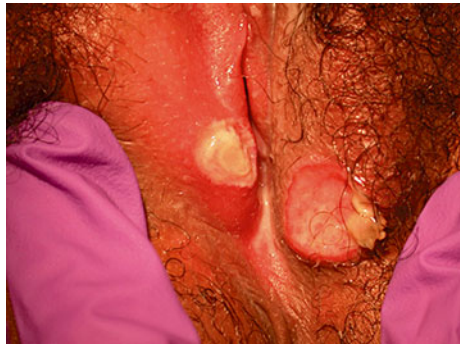

Fig. 7.7 Lipschutz ulcers (Reprinted from Huppert [46], with permission of John Wiley & Sons, Inc.)

marked prodromal symptoms than Lipschutz (idiopathic) ulcers.

Lipschutz ulcers, also called ulcus vulvae acutum or reactive nonsexually related acute genital ulcers, are vulvar ulcerations without other identifiable etiology, often occurring in young, virginal patients (Fig. 7.7) [47]. The lesions are well demarcated, with raised edges, often with a gray or white base. These lesions can sometimes be red-black and develop eschars [44]. Lesions vary in size. They are usually, though not exclusively, located on the interior labia minora, often on opposing surfaces (called "kissing ulcers") [44]. These are often preceded by prodromal symptoms of fever and malaise.

In general, biopsies are not helpful. A targeted infectious workup can be conducted; serologic testing for EBV is indicated if postviral genital ulcers are suspected. CMV is less strongly associated with genital ulcers. EBV can be tested by viral culture or PCR from the ulcer base or by EBV serology [48]. Testing for influenza can also be performed, depending on a clinician's index of suspicion. Depending on patient age and sexual activity (or concern for sexual abuse in a pediatric patient), testing an ulcer for HSV is reasonable.

Consider a Foley catheter for urinary diversion, and 2 % lidocaine jelly for topical analgesia. The utility of steroids is

unclear, though some expert opinions recommend a trial of topical corticosteroids, such as clobetasol 0.05 % ointment 0.05 % every 12 h for 7–10 days [46, 49]. Oral corticosteroids are reserved for refractory cases. Up to 30 % of women may have superinfection of their vulvar ulcers; assess for purulence or overlying erythema and treat with a broad-spectrum antibiotic such as a cephalosporin or sulfonamide to address skin flora [46].

Hidradenitis Suppurativa

Hidradenitis suppurativa is a chronic inflammatory skin disorder of the skin in the axillary, inguinal, and genital regions, characterized by recurrent tender nodules and abscesses that progress to draining sinuses, resulting in raised, firm, and discolored scars [50, 51]. Lesions may also involve the breasts and buttocks. Highest incidence appears to be in women in their 20s, though it has been described in prepubertal and postmenopausal women; in women, lesions often occur premenstrually [31, 51]. Risk factors include family history, smoking, and obesity [50, 51].

Diagnosis is usually made based on a history of recurrent abscesses in the axilla and/or groin with scarring [51]. Biopsies and bacterial cultures are reserved for severe and refractory cases. Localized lesions can be treated with topical clindamycin (10 mg per milliliter (mL) two times per day for 3 months) or triamcinolone (2–5 mg injected into lesions) [51]. For more advanced disease, systemic tetracycline (500 mg PO every 12 h for 3 months), erythromycin (500 mg PO every 12 h), doxycycline (100 mg PO every 12 h), or minocycline (100 mg PO every 12 h), clindamycin, and rifampin (300 mg PO every 12 h, each, for 10 weeks) have also been used [33, 52, 53]. Small series of patients treated with medications with antiandrogenic properties have reported success, including management with combined oral contraceptive pills containing ethinyl estradiol 50 micrograms (μg)/norgestrel 500 μg [54]. In severe cases, isotretinoin or systemic immunosuppression or surgical excision of sinus tracts may

be required; laser therapy has also been used. All patients should be counseled for weight loss and smoking cessation.

Inflammatory Bowel Disease

Inflammatory bowel disease (IBD)—Crohn's disease in particular—is associated with vulvar manifestations. Crohn's disease is a chronic inflammatory disease that primarily affects the gastrointestinal tract, and many involve any section along its length [47]. The perianal region may be directly affected, with sinus or fistulous tracts or ulcerations; mucocutaneous lesions (often resembling aphthous ulcers) occur in up to 75 % of patients [55]. Crohn's disease is also associated with vertical linear fissures (called "knife-cut" ulcers) which may precede diagnosis of the IBD (Fig. 7.8) [5, 56]. The etiology of these ulcerations is unclear; proposed mechanisms include deposits of antigens or immune complexes in the skin or immune system cross-reactivity.

If the lesions are determined to be part of a syndrome of IBD, treatment should be targeted at the underlying illness, usually through immunosuppressive medications. Consultation with gastrointestinal specialists (or the patient's own specialist) is advisable. Topical steroids and

Fig. 7.8 Fissure of the gluteal cleft associated with Crohn's disease (Reprinted from Leu et al. [49], Figure 7.2, with kind permission from Springer Science and Business Media)

tacrolimus have been noted to be helpful in treating perineal disease. Oral metronidazole has also been shown to be helpful as well [5].

Pyoderma Gangrenosum

A destructive sterile inflammatory skin condition, pyoderma gangrenosum (PG) can present with bullae, erosions, and ulcerations with discharge (Fig. 7.9) [57, 58]. Lesions can appear anywhere on the body; vulvar PG is more common in pediatric patients. The ulcers are rapidly progressive and raised with necrotic bases, with up to 2 cm of surrounding erythema; the lesions are often instigated by trauma [47]. Ulcers may coalesce into a larger erosion. These lesions have been described in all age groups.

PG is associated with underlying illness in 50 % of patients, including inflammatory bowel disease, primary biliary cirrhosis, hematologic or other malignancies, Wegener's granulomatosis, and systemic lupus erythematosus [59].

Biopsy is usually required for diagnosis. Proposed diagnostic criteria include rapid progression of a painful ulcer

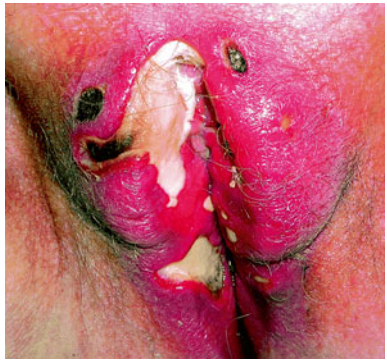

Fig. 7.9 Vulvar pyoderma gangrenosum (Reprinted from Sau and Hill [57], with permission of John Wiley & Sons, Inc., and the Royal College of Obstetricians and Gynaecologists)

with an irregular, violaceous border, for which other causes of ulceration have been ruled out. In addition, two minor criteria are required: (1) pathergy, meaning the lesion was instigated by trauma, or cribriform scarring, (2) a systemic disease associated with PG, (3) specific histopathologic changes, and (4) rapid response to steroid treatment [60]. Patients should have a full workup for underlying conditions, including but not limited to a complete blood count, complete metabolic panel, hepatitis testing, serologic assessment for autoimmune disease and hypercoagulability, and serum and urine immunoelectrophoresis; consider assessment by gastroenterology for IBD if no other source is identified [47, 59].

Once other causes of ulceration have been ruled out, limited disease can be managed with topical steroids, such as clobetasol 0.05 % ointment or cream [61]. Full lesion healing can take 1–2 months. Application of topical tacrolimus—a calcineurin inhibitor that is immunosuppressive—as a 0.1 % ointment applied twice daily over several months, has also been described for limited, local disease [62]. Severe disease may necessitate systemic immunosuppression; consider dermatology consultation in these cases; more aggressive treatment modalities include systemic corticosteroids, dapsone, tumor necrosis factor (TNF) inhibitors, and cyclosporine [47]. Small case series also suggest that gentle sharp debridement and wound reconstruction may encourage healing [57, 63].

Blistering Diseases

A variety of rare blistering diseases can involve the vulvovaginal region, including but not limited to Stevens-Johnson syndrome, toxic epidermal necrolysis, mucous membrane pemphigoid, and pemphigus vulgaris [64]. These patients require multidisciplinary management, and gynecology is usually involved after the diagnosis is made, often by biopsy, for assessment of the vulvovaginal region. These patients often receive systemic immunomodulatory therapies, which will not be covered here, and the role of the gynecologist is to recommend any therapies specific to the vulvovaginal region.

Stevens-Johnson syndrome (SJS) and **toxic epidermal necrolysis (TEN)** are severe necrotizing mucocutaneous reactions, most often occurring in response to medications, though infections such as *Mycoplasma pneumoniae* can be triggers [65]. SJS and TEN are on a continuum with one another, in which TEN affects more body surface area (>30 %).

Erythema multiforme is an immune-mediated condition involving target-like skin lesions, with or without central disruption (vesicles, blistering, or ulceration), with mucosal involvement typically only with the more severe versions [66]. Onset is usually between ages 20 and 40 years. The majority of cases are triggered by infections, namely, HSV; other identified causes include but are not limited to medications, autoimmune disease, and malignancy [67]. Patients with vulvovaginal involvement of erythema multiforme may have pigment changes, but scarring and stenosis may be less common than in SJS and TEN [68].

Mucous membrane pemphigoid (also called cicatricial pemphigoid) is an autoimmune condition characterized by subepithelial blisters leading to painful erosive lesions in mucosal membranes; age of onset is commonly after age 60 years, though it may also appear in children [69]. Lesions occur most often in the mouth, though the nose, upper respiratory tract and esophagus, vagina, and anus can also be involved.

Bullous pemphigoid is the most common autoimmune subepidermal blistering disease; most cases are sporadic, but some have been associated with UV light, radiation, and medications such as furosemide and various antibiotics [3, 70]. Tense bullous lesions form, often preceded by eczematous or urticarial skin. Mean age of onset is over 60 years. Vulvar involvement is more common in children and can occur in isolation [71].

Linear IgA disease is a rare autoimmune disease in which IgA is deposited along the cutaneous basement membrane; it affects adults over 60 years and children under 5 years [3, 72]. Genital involvement is more common in children. Oral

ulcers are often also present. Lesions may present as pruritic vesicles and bullae in groups or as an urticarial plaque with vesicles at the periphery [3].

Pemphigus vulgaris is an autoimmune intraepidermal blistering disease that usually presents after age 60 years, though it may appear in children [72]. Lesions are classically large flaccid blisters of the mucosal surfaces and skin, usually first appearing in the oral cavity and quickly progressing to painful ulcerations. The Nikolsky's sign should be present, in which epidermal detachment occurs when pressure is applied. Vulvovaginal lesions are reported in up to one-half of cases, most commonly involving the distal one-third of the vagina; cervical involvement has also been reported, which may be mistaken for cervical dysplasia on Papanicolaou smears [73–75]. Genital lesions are thought to occur after involvement of other sites, and patients diagnosed with this condition should have genital exams as genital lesions may be unrecognized [76].

Paraneoplastic pemphigus is an autoimmune syndrome similar to these other blistering diseases, occurring in response to a systemic neoplasm, such as lymphoma or leukemia [3].

Vulvovaginal lesions in patients with these blistering diseases are likely manifestations of the disease, though superinfection in the setting of immunosuppression or malignancy should be considered. Any blistering diseases involving the vaginal mucosa may result in vaginal stenosis or agglutination, putting patients at risk for developing chronic pain, sexual dysfunction, dyspareunia, and obstruction of urinary or menstrual flow [77]. Patients with a history of vulvovaginal SJS and TEN are also at risk for developing vulvovaginal adenosis—the presence of ectopic endometrial or cervical glandular epithelium—which has been associated with cellular atypia, squamous cell carcinoma, and adenocarcinoma [78, 79].

A useful set of management guidelines have been outlined for vulvovaginal SJS and TEN, but are applicable to these other conditions. Prevention of adhesion formation and agglutination after desquamating lesions broadly involves

intravaginal glucocorticoids and menstrual suppression [80]. Specifically for SJS and TEN, a regimen of betamethasone valerate 0.1 % cream applied every 12 h to the vulva externally and betamethasone valerate 0.1 % ointment every 12 h to the internal vaginal mucosa via dilators is advisable, to maintain vaginal patency, if appropriate given the patient's age and history of sexual activity [77]. If the patient is not on systemic antifungal therapy, topical antifungal creams can be considered because intravaginal steroids can predispose to fungal infections.

Less Common Causes of Vulvar Lesions

Vulvar malignancy: The most common vulvar malignancy is squamous cell carcinoma, which is most often related to human papilloma virus infections in women younger than 55 years. Rates of vulvar cancer are also increased in women with immunosuppression and lichen sclerosis [81]. Vulvar malignancies usually present with pruritis, though ulcerations and pain can also occur. Any suspicious lesions should be biopsied; vulvar colposcopy can also be considered to guide biopsies. While squamous cell cancers are the most common, vulvar intraepithelial neoplasia (a premalignant condition), basal cell carcinoma, sarcoma, melanoma, and extramammary Paget's disease should also be considered [81]. These diagnoses can be clarified by biopsy. Patients with vulvar dysplasia or malignancy should be referred to a gynecologic oncologist.

Behçet's syndrome: Behçet's syndrome is a vasculitis causing relapsing oral and genital ulcers and uveitis; other symptoms include skin lesions such as erythema nodosum and thrombophlebitis [82]. The prevalence of Behçet's syndrome ranges from 1:10,000 in Japan to 1:500,000 in North America and Europe. It rarely presents before puberty or after age 50. Genital lesions are commonly round and well demarcated. Approximately 60 % of genital ulcers from Behçet's syndrome result in scarring, which may be visible on physical exam [83].

Periodic fever, aphthous stomatitis, pharyngitis, and cervical adenitis (PFAPA) syndrome: PFAPA appears in children, before age 5, and consists of recurrent fevers lasting less than a week, aphthous ulcers, pharyngitis, and cervical adenitis without concomitant signs of infection [84]. This is a diagnosis of exclusion [84].

Tuberculosis is a very rare cause of vulvar lesions or ulcers, but which may be suspected based on a patient's travel and exposures. Cutaneous tuberculosis ulcers may begin as papules and develop into painful ulcers, with a pseudomembrane [47, 85]. Biopsy or PCR can be performed for diagnosis.

For the diagnosis and management of vulvar psoriasis, please see Chap. 11, Vulvovaginitis and Vaginal Bleeding in Pediatric and Adolescent Patients.

Perineal Masses

Bartholin's gland cysts are collections in the Bartholin's glands or ducts, which exit the vestibule at 4:00 and 8:00. These glands are most likely to become obstructed in reproductive age women, and patients present with enlarged, fluctuant masses under the gland exits (Fig. 7.10). Asymptomatic gland cysts do not require intervention. Painful and erythematous gland enlargements may be superinfected, which are usually polymicrobial infections; gland abscesses should be drained.

Patients with symptomatic Bartholin's gland cysts or suspected abscesses can be managed with incision and drainage *inside* the vaginal introitus—not through the labia—preferably with insertion of a Word catheter (a small latex catheter with a terminal balloon holding a few milliliters.) The catheters allow continued drainage and should be in place for 4–6 weeks, with the protruding end tucked into the vagina [86]. Patients do not need antibiotics unless the physical exam suggests overlying cellulitis or systemic infection (i.e., leukocytosis and/or fever). Patients with recurrent symptomatic gland cyst or abscess may undergo marsupialization, in which

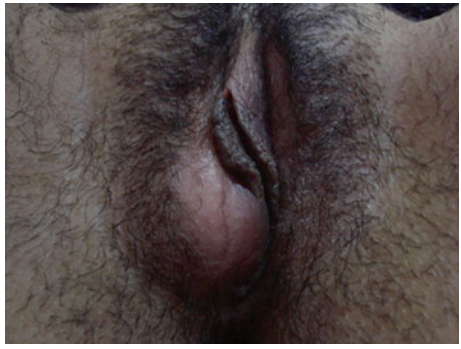

Fig. 7.10 Right Bartholin's gland cyst (Reprinted from Maldonado [86], with permission of Elsevier)

the cyst wall is incised and sutured to the overlying mucosa. In postmenopausal women, Bartholin's gland cysts should be biopsied due to risk of adenocarcinoma.

Skene's glands, also called paraurethral glands, emerge at either side of the urethral meatus and may also become enlarged. A **urethral diverticulum**, which communicates with the urethral lumen, presents in a similar way, though these may be located more proximally than Skene's gland cysts. Ultrasonography, urethrocystoscopy, or MRI can be used to differentiate the two lesions. Skene's gland abscesses can be incised and drained, while urethral diverticula should not be incised and can initially be managed with sitz baths and antibiotics [52]. Persistent, symptomatic urethral diverticula may need to be excised by specialists in female pelvic floor surgery.

Embryologic remnants in the vagina—**müllerian and Gartner (mesonephric) ducts**—can also become cystically enlarged; clinically, the differentiation between the two is unimportant. Symptomatic, enlarged ducts should be imaged with MRI to exclude urethral diverticula and can then be excised [87]. Gartner duct cysts may also be associated with congenital urinary and renal system anomalies.

A **canal of Nuck cyst** is a hernia through the inguinal canal into the labia majora; one-third of these contain bowel [86].

For large symptomatic labial enlargements suspected to be canal of Nuck cyst, an MRI should be obtained to clarify the anatomy; these cysts should be excised by general surgeons. Epidermoid inclusion cysts, which are usually less than 5 mm, occurring on the labia majora at any age, do not usually require intervention [86]. Lipomas, neurofibromas, vulvovaginal endometriosis, and vulvar leiomyomas are less common possibilities as well.

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