Management of Chronic Recurrent Vulvovaginitis

Abigail Kingston and Emma Torbé

Abstract

Vulvovaginitis is the most common reason why women present to a gynecologist. The term covers inflammation or infection of the vagina and/or vulva. Women with symptoms lasting for more than 6 months experience chronic vulvovaginitis. The clinical presentation of women with vulvovaginitis is similar regardless of the underlying cause and so a careful history is important. Symptoms include itching, discharge, irritation, dysuria, vaginal odor, rash, and burning. Other findings on exam include erythema, edema and excoriation of the vulvar skin, ulceration, or chronic vulvar skin changes. The following investigations can aid diagnosis: vaginal pH, amine whiff test, vulvar biopsy, fungal cultures, and wet smears. Causes fall into four main groups, infections, dermatoses, atrophy, and neoplasia. Infectious causes include bacterial vaginosis, vulvovaginal candidiasis, *Trichomonas vaginalis*, and threadworms (pinworms). Symptoms will improve following effective treatment of the infection. Vaginal atrophy is a result of estrogen deficiency and is improved with estrogen replacement. Dermatoses include lichen sclerosus, lichen planus, contact dermatitis, and lichen simplex. Treatment of dermatoses includes steroids, topical estrogens, tacrolimus ointment, retinoids, and emollients. All women will benefit from good vulvar skin care and emotional support.

Keywords

Vulvovaginitis • Bacterial vaginosis • Vulvovaginal candidiasis • *Trichomonas vaginalis* • Atrophic vaginitis • Contact dermatitis • Lichen simplex • Lichen sclerosus • Lichen planus • Threadworms

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A. Kingston (✉) • E. Torbé
Great Western Hospitals NHS Foundation Trust, Swindon, UK
e-mail: Abigail.Kingston@salisbury.nhs.uk; emmatorbe@doctors.org.uk

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1 Introduction

The term vulvovaginitis is very general and is used by medical professionals to cover inflammation or infection of the vagina and/or the vulva. It is one of the top 25 reasons why women seek medical care and is the most common reason that women present to a gynecologist (Kent 1991). As so many women suffer with this condition, it is not surprising that there is a subgroup with chronic vulvovaginitis (symptoms lasting for more than 6 months). Etiologies fall into four main groups: infections, dermatoses, atrophy, and neoplasia:

- Infections – *Trichomonas vaginalis*, bacterial vaginosis, vulvovaginal candidiasis, and threadworms.
- Atrophy – atrophic vaginitis.
- Dermatoses – lichen sclerosus, lichen planus, contact dermatitis, and lichen simplex.
- Neoplasia – many women with squamous cell carcinoma experience vulva irritation.

2 Pathophysiology

During a woman's reproductive years, the vagina maintains a moist environment that fluctuates during the menstrual cycle. Estrogen causes the vaginal nonkeratinized stratified squamous epithelium to be thick, rugated, and elastic by maintaining the epithelial collagen content. Estrogen also causes the epithelium to be rich in glycogen. The vaginal epithelium and cervical glands secrete an alkaline transudate resulting in a moist environment. The microflora of the vagina forms an environment that is unique and balanced. While it reacts to external stimuli, once the stimuli are removed, the vaginal environment returns to normal. The bacterial flora of a healthy vagina is made up of many microorganisms both aerobic and anaerobic gram-positive bacteria and gram-negative bacteria. *Lactobacillus* and *Corynebacterium* predominate over other bacteria *Streptococcus*, *Bacteroides*, *Staphylococcus*, and *Peptostreptococcus*. *Lactobacillus* and *Corynebacterium* produce lactic and acetic acid from glycogen that lowers the vaginal pH resulting in a normal pH ranging from 3.8 to 4.5. The optimal/normal vaginal discharge is clear, thin, and odorless and is between 1 and 4 ml/day although the presence of thick, white discharge with mild odor occurs in asymptomatic women and is not considered abnormal.

Vaginal pH increases with age, menses, cervical mucus associated with ovulation, contraceptive choice, sexual activity, ejaculation, pregnancy, rupture of membranes in pregnancy, exposure to exogenous hormones, antibiotics, use of hygiene products, douching, foreign bodies in the vagina, high sugar diet, and infections (trichomoniasis, bacterial vaginosis, group A streptococcal infection).

The vulvar skin is sensitive to the vaginal environment as well as hormonal, metabolic, and allergic influences. The most significant change occurs during the menopause where there is a compromise in the vulva's barrier function. This is due to both estrogen deficiency and aging, resulting in a rise in pH and a decrease in the antimicrobial defenses of the skin. There is concomitant loss of lipid production that slows healing in response to injury, and there is an increased risk of infections due to an age-related decline in cell-mediated immunity (Summers and Hunn 2007).

3 Presenting Symptoms

The symptoms of vulvovaginitis are varied and common, and many medical therapies for treatment or symptomatic relief are available to purchase over-the-counter. Although there is an expectation and an acceptance that women are capable of self-medication, the accuracy of women to correctly self-diagnose is poor leading many to inappropriate medications, sometimes exposing themselves to irritant dermatitis and

perpetuating symptoms (Nyirjesy et al. 1997). It is therefore essential that a clinician takes a full history and performs a careful examination, particularly in those with chronic symptoms, to establish a correct diagnosis and direct correct treatment. In the United States, an estimated $250 million is spent annually, on over-the-counter antifungal therapies, the most common medication used for vulvar itching or discharge.

Clinical presentations of women with vulvovaginitis are often similar regardless of the underlying causes so a careful history should be taken. The history includes specific symptoms such as itching, discharge, irritation, dysuria, dyspareunia, vaginal odor, rash, pain, and burning. Documenting the nature of the discharge including color, consistency, and quantity, as well as duration of the symptoms, prior treatments, and responses, along with mitigating or aggravating factors is important. Differentiating particular vulvar complaints from vaginal complaints is helpful.

4 Physical Examination

Inspection of the vulva may reveal areas of erythema, edema, excoriation of vulvar skin, ulceration, skin changes, color changes, rashes, raised lesions, or chronic vulvar skin changes. Vulvar tenderness can be elicited by manual palpation or by using a cotton-tip applicator. The vagina and cervix should be thoroughly inspected using a speculum. Samples can be obtained from the lateral vaginal wall or vaginal fornix for laboratory evaluation. These evaluations include:

- Vaginal pH.
- Fungal cultures.
- Smears for microscopic examination to identify trichomonads, clue cells, as well as presence of white blood cells or bacteria. A ratio of white blood cells to epithelial cells of more than 1:1 indicates an underlying infection, so further sexually transmitted infection screening should be performed in wet-mount/amine (whiff) test. A drop of 10% potassium hydroxide (KOH 10%), which is alkaline, is added to the vaginal secretions. The epithelial cells undergo lysis which increases the ability to identify hyphae or blastospores. Anaerobic bacteria produce amines. Adding 10% KOH results in volatilization of the amines causing a sharp fishy odor.
- Biopsy of abnormal vulvar lesions may identify vulvar cancer, vulvar intraepithelial neoplasia, lichen sclerosus, vulvar dermatoses, and other conditions.
- Herpes culture should be performed in patients with vulvar or vaginal ulcerations.

5 Vulvovaginal Candidiasis

Vulvovaginal candidiasis is caused by an overgrowth of yeasts and is very common as 75% of all women will develop a yeast infection in their lifetime (Faro et al. 1997). There is a peak of yeast infections during pregnancy when the vagina is exposed to high levels of estrogen (Sobel et al. 1998). Rarely vulvar candidiasis is present without concomitant vaginal candidiasis. *Candida albicans* is responsible in up to 90% of cases with non-albican species making up the remainder (Sobel et al. 1998).

Recurrent vulvovaginal candidiasis (RVVC) occurs in less than 5% of women (Nyirjesy 2001). It is defined as four or more symptomatic *proven* episodes in 1 year (Sobel et al. 1998). Predisposing factors to RVVC are antibiotic use, corticosteroid use, diabetes, high sugar intake, high estrogen contraception, and deficiency in the immune system (such as women affected with human immunodeficiency virus) (Goswami et al. 2000; Duerr et al. 2003).

After treatment some women remain colonized with small number of yeast, and particularly if they have risk factors, they may have a new clinical episode of vulvovaginal candidiasis that is not from a reinfection (Vazquez et al. 1994). Extended length of treatment is often recommended due to the fact that while the yeast hyphae can be eliminated by anti-yeast medications, the spores are resistant and will not be affected by the medicine until they hatch. Up to 33% of recurrent infections

are caused by non-albican species such as *Candida glabrata*, *Candida parapsilosis*, *and Saccharomyces cerevisiae* (Nyirjesy et al. 1995).

Diagnosis Women present with symptoms of burning and irritation, vulvar pain, itching, dyspareunia, and vaginal discharge. On examination the white vaginal discharge is curd-like [cottage cheese], thick, and white. The vulva is erythematous and there may be labial swelling.

A wet-mount preparation usually reveals the hyphae and spores of *Candida albicans.* The branching, budding, and hyphal cell walls are easily visualized. However, the spores of *Candida glabrata* are more difficult to identify. They are spherical or ovoid and more variable in size but smaller than red blood cells. They are often clustered in groups and are associated with hyphal filaments. 10% KOH of lyse red and white blood cells reveal spores not otherwise seen on saline preparation.

Gram stain will show *Candida* spores as gram positive and the filaments as uniformly gram positive or with large gram-positive granules. In recurrent VVC cultures should be taken to accurately identify the species of candida. Detection can take up to a month. Sugar fermentation reactions are the most reliable for differentiating between species (Haefner 1999).

Treatment 10–20% of women are colonized with candida (Sobel et al. 1998) and so treatment is only required in the context of symptoms. Women with RVVC should be reviewed to identify an underlying chronic illness affecting the underlying metabolic and immunological state (e.g., diabetes, systemic lupus erythematous, thyroid dysfunction). Controlling underlying medical conditions may minimize exogenous factors. Lowering the estrogen dose in combination oral contraceptive pills may also be beneficial for women with RVVC. Modification of a high sugar intake may also be helpful.

A suppression and maintenance regime may be considered such as fluconazole capsule 150 mg every 72 h for three doses followed by fluconazole capsule 150 mg once a week for up to 6 months (this regime is not advised in pregnancy or breastfeeding). For some recurrent cases, concomitant treatment for BV as discussed below can be considered. Approximately 90% of women will remain disease-free at 6 months and 40% at 1 year (BASHH 2007).

Alternative regimes are:

- Topical imidazole therapy for 10–14 days according to symptomatic response followed by:
  - Clotrimazole pessary 500 mg once a week
  - Fluconazole capsule 50 mg daily
  - Itraconazole capsule 50–100 mg daily
  - Ketoconazole capsule 100 mg daily or 200 mg orally twice daily x 1 day (BASHH 2007)

There may be inherent differences between *C. albicans* and non-*C. albicans* infections; therefore, obtaining a positive fungal culture, which includes identifying the infecting organism, is an essential first step in the management of RVVC. In RVVC secondary to *C. albicans*, resistance to antifungal therapy seems rare in that the vast majority of patients will, at a minimum, do well while on antifungal maintenance regimens. However, for infections caused by non-*C. albicans* species, particularly those due to *C. glabrata*, clinically evident resistance seems more common (Nyirjesy et al. 1995).

High-dose combined oral contraceptive pills are associated with increases in VVC infection. Affected women can consider swapping to a lower-dose combined preparation or an alternative non-estrogen-containing method of contraception. Low-dose combined oral contraceptives are unlikely to contribute to candida infections, so discontinuing their use is not recommended but could be considered in resistant recurrent cases. Copper intrauterine contraceptive devices have been identified as a possible risk factor as it has been shown that yeasts can adhere to the devices for recurrent VVC. Therefore women could consider changing to an alternative method of contraception. Progesterone injectables may reduce women's predisposition to VVC due to the relative hypoestrogenic state and anovulation. Women with male partners should be warned that some vaginal and vulvar treatments for

VVC can damage and weaken latex condoms (FRSRHC 2012).

The majority partners of women with recurrent VVC have negative cultures for candida (O'Connor and Sobel 1986) and so they are unlikely the source of reinfection. Treating the partners of women with RVVC does not reduce the risk of recurrence (Fong 1992) and is not recommended (FSRHC 2012).

Pregnant women are at high risk of VVC especially in the third trimester. The vaginal environment, under the influence of increased reproductive hormone levels, is conducive to yeast growth. Vaginal glycogen content increases, providing an abundant source of carbon for growth, adherence, and germination of candida. There is no evidence that VVC has an adverse effect on the pregnancy. Oral antifungals should be avoided. Women should be treated with topical imidazoles (FSRHC 2012).

6 Bacterial Vaginosis

Bacterial vaginosis (BV) is the commonest cause of abnormal vaginal discharge in women of reproductive age.

BV is characterized by an overgrowth of anaerobic bacteria in concentrations up to thousands of times greater than normal. Conventional culture techniques have identified *Gardnerella vaginalis*, *Prevotella* spp., *Mycoplasma hominis*, and *Mobiluncus* spp. as those most commonly found. *Gardnerella vaginalis* presence alone is not diagnostic as it can be a vaginal commensal in up to 50% of asymptomatic women (BASHH 2012). However its role in forming a biofilm with other bacteria may be key to the etiology of BV (Swidsinski et al. 2005). Molecular techniques have identified the presence of other species of bacteria including *Atopobium vaginalis*, *Clostridiales* spp., *Leptotrichia* spp., and *Sneathia* spp. (Fredricks et al. 2005).

BV is sexually associated as opposed to sexually transmitted. It is linked with sexual behaviors including sexual, oral, and digital intercourse (Fethers et al. 2008). BV has not been found in women who are truly sexually inexperienced (Fethers et al. 2005). Other risk factors include vaginal douching, recent change of sex partner, multiple sexual partners, smoking, presence of a sexually transmitted disease, and copper intrauterine contraceptive device (Klatt et al. 2010). There is debate about whether BV is merely an imbalance in vaginal ecology or is initiated as a sexually transmitted infection.

The presence of BV increases the risk of preterm birth with low birth weight by 40%.

Diagnosis At least three of the four following criteria must be present to diagnose BV by Amsel's criteria (Amsel et al. 1983): (1) thin, white, homogeneous discharge which looks like skimmed milk that can be adherent to vaginal walls, (2) clue cells on wet-mount microscopic examination, (3) pH of vaginal fluid >4.5, and (4) positive whiff test – release of a fishy odor on adding alkali (10% KOH).

If a gram-stained vaginal smear is taken, BASHH recommends evaluating this by the Hay/Ison criteria (Ison and Hay 2002). Hay/Ison criteria are defined as follows: grade 1 (normal), *Lactobacillus* morphotypes predominating; grade 2 (intermediate), mixed flora with some *Lactobacilli* present, but *Gardnerella* or *Mobiluncus* morphotypes also present; and grade 3 (BV), predominantly *Gardnerella* and/or *Mobiluncus* morphotypes, few or absent *Lactobacilli*. BV is caused by a complex change in normal vaginal bacterial flora making a vaginal culture generally useless. Additionally, *G. vaginalis* can be cultured in up to 50–60% of asymptomatic, healthy women. The Affirm VP III [DNA probe] test can detect high concentration of *G. vaginalis*.

Treatment Most patients will initially respond to pharmacological treatments to BV; however, recurrence is common with more than half recurring by 12 months (Bradshaw et al. 2006). Metronidazole or clindamycin administered either vaginally or orally results in 70–80% of cure rates. Tinidazole 2 g once daily for 2 days or 1 g once daily for 5 days has been approved for treatment of bacterial vaginosis.

Options for treatment of recurrent bacterial vaginosis include suppressive treatment with metronidazole vaginal gel used twice a week, oral antibiotics followed by boric acid, and then suppressive therapy with metronidazole vaginal gel. Probiotic therapy may be considered but optimal treatment has not been established. Acidifying gels [boric acid] may be effective (FSRHC 2012).

Women using a copper intrauterine contraceptive device may consider changing to an alternative method of contraception (FSRHC 2012). Oral combined contraceptive pills and condoms are associated with a reduced risk of BV (Calzolari et al. 2000).

Symptomatic pregnant women should be treated in the usual way. Women with BV with additional risk factors for preterm birth may benefit from treatment before 20 weeks of gestation, and therefore screening such high-risk groups should be considered. Treatment of choice is metronidazole and is not associated with teratogenicity (BASHH 2012). Metronidazole alters the taste of breast milk, so breastfeeding mothers should receive clindamycin or be advised to stop feeding during the course and for 24 h after.

7 Trichomonas vaginalis

*Trichomonas vaginalis (*TV*)* is a flagellated anaerobic protozoan. It is rarer than RVVC and BV. It is sexually transmitted. TV can increase the risk of getting or spreading other sexually transmitted infections including HIV.

Diagnosis Up to 50% of women are asymptomatic with a third becoming symptomatic within 6 months. Those with symptoms complain of vaginal and vulvar discomfort, characteristically soreness or burning, dyspareunia, and vaginal discharge. Occasionally women complain of abdominal pain or vulvar ulceration. The typical vaginal discharge described as copious yellow green, frothy, and foul smelling only occurs in up to 30% of women. The discharge can range from thin and scanty to profuse and thick (Fouts and Kraus 1980; Wolner-Hanssen et al. 1989). On examination the cervix may have the classic "strawberry appearance" as a result of punctate cervical microhemorrhages. This can be seen by the naked eye but more commonly picked up at colposcopy (Fouts and Kraus 1980; Wolner-Hanssen et al. 1989). Vaginal pH is generally greater than 5.0 and often greater than 6.0. *T. vaginalis* may be seen on Papanicolaou smears.

Trichomonads are seen by a light field microscopy of the vaginal discharge on wet mount. Vaginal discharge is collected and mixed with saline on a glass slide and a coverslip is placed on top. The wet preparation slide should be scanned at both low and high magnification to confirm trichomonads [oval- or pear-shaped protozoans with a flagella] and to visualize the flagella. The slide must be read in under 10 min as trichomonads quickly lose their motility and after this time are more difficult to identify (Kingston et al. 2003). Microscopy can be performed in a clinic setting, near to the patient.

Point of care tests are available [such as rapid antigen tests and nucleic acid amplification] that do not require instrumentation. They can provide a result in 30 min and so can be performed in the clinic setting near the patient. It is a suitable alternative to culture or molecular testing. These tests have a high sensitivity (greater than microscopy) and specificity (Nye et al. 2009).

In difficult-to-diagnose cases, culture of TV or nucleic acid amplification tests could be considered as they have a higher sensitivity compared to microscopy (BASHH 2014a). Several culture mediums are available. Specimens should be incubated anaerobically and growth detected in 48 h.

Treatment *T. vaginalis* is a multifocal infection of the vaginal epithelium. Vaginal epithelium, Skene's glands, Bartholin's glands, and urethra can all be affected, so systemic treatment is essential for complete cure. Nitroimidazole drugs [metronidazole] given in either single dose or over a prolonged period result in cure in over 90% of cases (Forna and Gulmezoglu 2003).Therefore recommended regimes are metronidazole 2 gm orally in a single dose or metronidazole 400–500 mg twice daily for 5–7 days (BASHH 2014a).

Recurrent TV can be due to drug resistance but is more commonly due to reinfection due to failure to treat all sexual partners or a new sexual contact. Therefore it is important to reconfirm the diagnosis in patients who return with recurrence of symptoms. Partner notification is required and all parties treated. To best achieve this, sexual health services should be involved. Patients should be advised to avoid sexual contact for at least a week and until their partners have completed treatment and follow-up (BASHH 2014a).

A strain of *T. vaginalis* exists that is resistant to metronidazole and other nitroimidazoles (Kirkcaldy et al. 2012). Therefore for patients that continue to have *T. vaginalis* despite therapy and in which reinfection is excluded, increased doses and longer duration of therapy may be required. Regimes include metronidazole or tinidazole 2 gm a day for 5–7 days (BASHH 2014a). In women who continue to not respond, resistance testing should be considered. Treatment protocols guided by resistance testing results have improved outcome (Bosserman et al. 2011). For women with recurrent symptoms, a test of cure is then recommended (BASHH 2014a). Concomitant treatment with an antifungal can also be considered.

8 Threadworms

Threadworms are nematode infection with *Enterobius vermicularis*. It is also known as pinworm or enterobiasis. It is the most common helminthic infection in the United Kingdom. It is most commonly seen in children aged 5–9 years but can affect any age, frequently affecting family groups or institutions. Overcrowding and poor hygiene contribute to spread and reinfection.

The female threadworm is 1 cm long and a little under 1 mm in diameter. It is white and pointed at each end. The male threadworm is much smaller at only 4 mm long and is rarely seen. Female threadworms may be seen at night emerging from the anus to lay eggs. The female lays on average over 10,000 eggs outside around the anus, vagina, and urethra. The eggs are so small that they are invisible to the naked eye and are accompanied by an irritant mucus, which causes intense itching and scratching. Scratching transmits the eggs from the perineal and perianal skin to the hands. Then these are transmitted to the mouth, swallowed or inhaled, and then ingested. The larvae hatch in the small intestine and migrate to the colon where they reach maturity over 2 weeks. Adult worms live for up to 6 weeks. Its only host is humans (Ibarra 2001). Transmission may occur through handling of contaminated food, clothing, and bed linen.

Diagnosis Women present with itching of the vulva or anus, especially at night. Itching can cause loss of sleep. Threadworms do not move much and so can be easily missed, but if seen are diagnostic. Eggs can be detected by the adhesive tape test. Transparent wide hypoallergenic adhesive tape is applied to the perianal skin first thing in the morning, before wiping or bathing. It does not have to be left on overnight. The tape is then examined microscopically and the eggs are seen adherent to the tape.

Treatment Mebendazole kills the threadworms. It is administered as a single oral dose and is best repeated after 2–3 weeks in case reinfection has occurred. Other possible drugs include albendazole and pyrantel pamoate. Asymptomatic infections can occur and so all the family and close contacts should be treated at the same time, regardless of symptoms.

Drug treatment is not mandatory. The life cycle of threadworms is 6 weeks and the eggs remain viable for further 2 weeks. Excellent hygiene over this time frame will lead to the threadworms dying out without a chance to reinfect. Hygiene measures include:

- Wash each morning especially around the anus and vulva.
- Keep fingernails short and clean.
- Wash hands and scrub under the nails first thing in the morning. Wash hands and nails prior to preparing food and eating.
- Wash hands and nails after using the toilet.
- Put toothbrushes in a closed cupboard and rinse them well before use.

- Do not share towels or beds. If possible wash towels and bed linen daily.
- Change and wash underwear and nightwear daily.
- Do not shake the laundry as this can spread eggs.

9 Atrophic Vaginitis

The storage of glycogen by the vaginal epithelium is under the influence of estrogen. If women have extremely low endogenous estrogen production such as after the menopause or bilateral salpingo-oophorectomy, gonadotrophin-releasing hormone analogues, radiation, chemotherapy, or immunological disorders, the glycogen content of the vaginal epithelium drops resulting in atrophy (Summers et al. 2007). It has been estimated that up to 50% of women experience symptoms due to atrophic vaginitis at 5 years postmenopause (Sturdee and Panay 2010). Postpartum women can also experience atrophy because of the decline in estrogen levels in conjunction with the loss of placental estrogen, and prolactin produced during lactation has an antagonistic effect on estrogen.

Reduced endogenous estrogen reduces the glycogen content of the epithelium which in turn reduces lactic acid production and increases the vaginal pH leading to a reduction in *Lactobacillus*.

Diagnosis History and physical examination will usually lead to a diagnosis. Symptoms of atrophic vaginitis include soreness, postcoital burning, dyspareunia, dryness, vulvar or vaginal itching, leucorrhea, pressure, yellow discharge, and occasional spotting. There are often associated urinary tract symptoms such as dysuria, frequency, leaking, and infection. The vaginal epithelium appears thin, smooth, pale, or shiny. The vaginal rugae will have disappeared. Petechiae and increased friability or erythema may be present. There can be fusion of the labia minora, introital stenosis, thin vulvar skin with patch erythema, or discolored lesions. Vaginal pH will be raised and a wet mount shows white blood cells and a lack of *Lactobacillus* (Kingston 2009).

Treatment Many women with vaginal atrophy are not symptomatic, so treatment may not be desired. For symptomatic women treatment with vaginal estrogens is effective. Tablets and creams can be used nightly for 2 weeks and then twice weekly. Twice weekly maintenance dose can be continued long term and should be continued for as long as women have distressing symptoms. Systemic absorption is minimal and so progesterone is not required (BMS 2016). Some women may prefer to switch to oral or patch therapy after a few weeks or months of vaginal treatment. Those patients presenting with symptoms while already taking systemic HRT may show improvement in symptoms with the addition of vaginal estrogens (NICE 2015).

10 Lichen Sclerosus

Lichen sclerosus (LS) is an inflammatory dermatosis of unknown etiology. It is a chronic relapsing disease. There is evidence to suggest that autoimmune factors may be involved in its pathogenesis, and recent evidence has shown autoantibodies to extracellular matrix protein 1 (Oyama et al. 2003). There is an increased frequency of other autoimmune disorders in females with lichen sclerosus (Meyrick-Thomas et al. 1988).

Diagnosis Presentation is usually with intense vulvar itching, but soreness, burning, or pain may be the primary symptom, particularly where there has been chronic itch. Pruritus is often worse at night and many women have disturbed sleep. If the introitus has narrowed, they may disclose dyspareunia and sexual dysfunction.

On examination LS classically appears as porcelain-white plaques on the vulva, perineum, and perianal skin. The texture of "parchment" or "cigarette paper" skin is characteristic and helps to distinguish lichen sclerosus from lichen planus or vitiligo. Changes may be localized or distributed in a "figure of eight" around the perianal skin figure involving the labia minora and majora, vestibule, clitoral hood, and perineum. Other features include fissuring, hyperkeratosis, and erosions. Loss of

architecture may be manifest as loss of the labia minora and/or midline fusion. The clitoral hood may be sealed over the clitoris so that it is buried or a clitoral pseudocyst may have formed. The vagina and cervix are spared, but extragenital lesions can be seen in around 10% of cases.

Clinical diagnosis of lichen sclerosus is accepted as standard practice (Neill et al. 2002). Outpatient biopsy under local anesthesia is well tolerated. Histology shows epidermis thinning with subepidermal hyalinization and deeper inflammatory infiltrate. It is useful in diagnostically difficult cases, those which fail to respond to treatment and suspicious areas. The lifetime risk of squamous cell carcinoma (SCC) in cases of lichen sclerosus is less than 5% (BASSH 2014b), and this may be higher than actual risk owing to the probable high prevalence of undiagnosed lichen sclerosus (Neill et al. 2002).

Investigation for autoimmune disease should be considered, especially thyroid dysfunction (i.e., T4 and TSH) as it is often asymptomatic.

Treatment Women are treated symptomatically. Recommended treatment is by ultrapotent topical steroids, e.g., clobetasol propionate. Various regimens are used with no evidence of an optimal regime (BASSH 2014b). One of the most common regimes is to use the treatment daily for one month, then alternate days for one month, then twice weekly for one month, followed by reviewing the patient after the three months. It can then be used as needed depending on symptoms (Neill et al. 2002). Ointment bases are much better to use on the anogenital skin because of the reduced need for preservatives in an ointment base and hence less risk of a secondary contact allergy (BASHH 2014b). Topical steroids are safe to use while pregnant or breastfeeding.

Women with active LS should have dermatology involvement. Those with complex conditions, such as developed vulvar intraepithelial neoplasia (VIN) or SCC on a background of LS, should be seen and followed up by an experienced clinician or in a specialized vulvar clinic. Surgery should only be used for the treatment of coexistenting abnormality requiring excision or to release labial fusion. Disease tends to recur around the scars (BASSH 2014b).

Patients should be informed about the condition and given written information. Women must be made aware of the small risk of neoplastic change. They should be advised to contact the doctor if they notice a change in appearance or texture (e.g., lump or hardening of the skin), or if there is a major change in symptoms. Good vulvar hygiene may be helpful.

11 Lichen Planus

Lichen planus (LP) is a rare inflammatory disorder of unknown but probably autoimmune pathogenesis. It has manifestations on the skin, genital and oral mucous membranes, and more rarely the lacrimal duct, esophagus, and external auditory meatus. It affects 1% of women.

Diagnosis Vulvar LP presents with intense itching, pain, soreness, dyspareunia, and bleeding. A purulent discharge caused by desquamative vaginitis is common when the vagina is involved (Goldstein and Metz 2005). The heterogenous appearance can be divided into three groups:

- Erosive LP is the most common subtype to cause vulvar symptoms. The mucosal surfaces are eroded looking “red raw”; the edges of the erosions are mauve. The lesions consist of friable telangiectasia with patchy erythema which are responsible for the common symptoms of postcoital bleeding, dyspareunia, and a variable discharge which is often serosanguinous. As erosions heal synechiae and scarring can develop, therefore vaginal lesions should be recognized and treated early to prevent scarring, stenosis, and introital narrowing (Genadry and Provost 2006). This type is also seen in the oral mucosa although synechiae are uncommon. Wickham striae are pathognomonic. On the vulva they appear as white reticular or linear papules and they may also be found on the buccal mucosa of the mouth. Vulvovaginal gingival syndrome

describes erosive disease occurring in the vulva, vagina, and mouth.
- Classical LP usually presents well-circumscribed papules which are flat topped. They are found on the trunk, extremities, and the keratinized anogenital and vulvar skin, with or without striae. Hyperpigmentation frequently follows their resolution, particularly those with dark skin. This type of lichen planus may be asymptomatic.
- Hypertrophic LP is relatively rare and can be difficult to diagnose. Lesions particularly affect the perineum and perianal area, presenting as thickened warty plaques which may become ulcerated, infected, and painful. Because of these features, they can mimic malignancy. They do not appear to be accompanied by vaginal lesions.

Skin changes elsewhere can be helpful to aid diagnosis. Histology of a vulvar biopsy is diagnostic and shows irregular saw-toothed acanthosis, increased granular layer and basal cell liquefaction, and band-like dermal infiltrate which is mainly lymphocytic (BASSH 2014b). Biopsy is essential if diagnosis is uncertain or to exclude coexisiting VIN or SCC. The incidence of developing SCC is as high as 3% (Cooper and Wojnarwowska 2006).

Treatment Good vulvar care (see below) should be recommended. Potent or ultrapotent topical steroids constitute first-line treatment and suppositories can be used for vaginal disease. Daily application for up to 3 months has been advocated, reducing as required (Goldstein). However there is not an evidence-based optimal regime. Maintenance can be achieved by either regular application of a weaker steroid or less frequent use of a potent steroid. Delivery of corticosteroids to the vagina is a challenge. A proprietary preparation containing hydrocortisone (Colifoam) is introduced with an applicator. Prednisolone suppositories may be used in more severe cases (BASHH 2014b). There is not any good evidence in systemic therapy. Vaginal dilators should be used early in vaginal involvement to prevent adhesions and topical local anesthetic gel can help with discomfort.

Surgery is reserved for reversal of severe scarring, particularly for younger or sexually active women.

Lichen planus is very often misdiagnosed as lichen sclerosus because of similarities in the presentation and the presence of white plaques. However, vaginal involvement precludes lichen sclerosus as a diagnosis. Lichen sclerosus and lichen planus are thought to be present simultaneously in some cases, perhaps those with more resistant disease (Neill et al. 2002). Referral to a multidisciplinary vulvar clinic should be considered for any woman with erosive disease, coexisting diseases, intractable symptoms, or scarring complications.

12 Contact Dermatitis

Contact dermatitis resulting in inflammation commonly affects the vulva. Inflammation of the skin occurs after exposure to an allergen (allergic dermatitis) or an irritant (irritant dermatitis).

Allergic reactions are a cell-mediated (type 4) immunological response that occurs on reexposure after an initial sensitizing episode. The history of symptoms may reveal the diagnosis. Patch testing can be performed to determine the allergen among women in ongoing symptoms. Common allergens include nail polish, latex preservatives, and lanolin. There are also reports of semen rarely causing symptoms, and absence of symptoms following intercourse with a condom may lead to a suspicion of this.

Irritant-induced contact dermatitis can be acute or chronic. It may occur from acute exposure to a potent irritant or after repeated exposure to a weak irritant. The postmenopausal vulva is particularly susceptible to irritation. Possible irritants include detergent, panty liners, moisturizers, chronic wetness from vaginal discharge or urine, topical medications, antifungals, latex, lubricants, spermicides, cosmetics, fragrances, cleansing

products, and washing powders/liquids (Kingston 2009).

Diagnosis A history of itching is the most common symptom. However, a potent stimulant causing acute reactions involving the mucosa can cause symptoms of burning, rawness, and pain. Persistent scratching in chronic conditions will also frequently lead to pain. On examination the skin can be red and edematous with exudation and weeping and rarely erosions and ulcers. If the exposure to the stimulant is prolonged, then lichenification, scaling, and thickening of the skin can occur with fissuring (see lichen simplex chronicus below).

Treatment Where possible, identify and remove the exacerbating agent. Provide women with written information on good vulvar care (see below). Aqueous cream has been shown to be a potential irritant when it is used as an emollient, so it's recommended as a soap substitute only. Consider topical corticosteroids in ointments for 7–10 days to treat the inflammation. In severe, rare cases, oral steroids could be considered. Any superimposed infection may need treating with antibiotics or antifungals as appropriate (Kingston 2009).

13 Lichen Simplex Chronicus

Lichen simplex chronicus results from an itch-scratch-itch cycle caused by another pathology making LSC a secondary condition. The original itch can be triggered by (BASHH 2014b):

- Underlying dermatosis – atopic dermatitis and superficial fungal (tinea and candidiasis) infections
- Environmental factors causing irritant or allergic dermatitis
- Systemic illness – obstructive biliary disease (primary biliary cirrhosis and primary sclerosing cholangitis), renal failure, hyper- or hypothyroidism, Hodgkin's lymphoma, and polycythemia rubra vera
- Psychiatric disorders – obsessive-compulsive disorder, anxiety, depression, and dissociative experiences

Diagnosis Patients characteristically describe an intractable itching and scratching, especially during sleep at night. Examination reveals lichenification, i.e., thickened, slightly scaly, pale, or earthy-colored skin, exaggerated skin markings, and excoriations, maybe more obvious on the side opposite the dominant hand. There may be erosions and fissuring. Pubic hair can be lost as a result of scratching (BASHH 2014b).

Treatment To treat the women, the cycle must be broken by treating the underlying cause, if known, and good vulvar care. Use a soap substitute for all washing and an emollient regularly. Emphasizing daily drying techniques may be helpful. If needed sedating antihistamines at night can be used to reduce the itch and aid sleep (such as hydroxyzine). Potent or ultrapotent topical corticosteroids can reduce the inflammation and treat the lichenification. These should be in ointment form as creams can cause irritation. Secondary infection is possible and so must be screened for and treated, if present, with an antifungal or antibiotic as necessary. Cognitive behavioral therapy may be helpful if there are coexisting mental health issues (BASHH 2014a).

14 Vulvar Care

Women who are prone to symptoms may benefit from the following advice on personal hygiene:

- Keep genital area dry (moisture encourages growth of candida and may cause chronic skin irritation).
- Wear 100% cotton underwear, avoid synthetic materials, and wear loose fitting clothing.
- Use cool baths to help to soothe the skin, but do not wash excessively.

Table 1 Summary of vulvovaginitis

	Vulvovaginal candidiasis	Bacterial vaginosis	*Trichomonas vaginalis*	Threadworms	Atrophic vaginitis	Contact dermatitis	Lichen sclerosus	Lichen planus	Lichen simplex chronicus
Discharge	Thick white curd-like	Thin adherent to walls, white/gray	Scanty to profuse, frothy yellow	Normal	Normal	Normal	Normal	Normal	Normal
Odor	Nil	Fishy offensive	Fishy	Nil	Nil	Nil	Nil	Nil	Nil
Itch	Present	Nil	Present	Present worse at night	Nil	Present	Present predominately at night	Present predominately at night	Present (characteristic)
Other symptoms	Dyspareunia, burning	Nil	Vaginal and vulvar discomfort, burning, dyspareunia	Vaginal and vulvar discomfort, burning, dyspareunia	Burning, dyspareunia, postmenopausal bleeding	Irritation, burning,	Vaginal and vulvar discomfort, burning, dyspareunia	Vaginal and vulvar discomfort, burning, dyspareunia	Nighttime scratching
Visible Signs	Erythema, edema, satellite lesions	Nil	Vulvitis, vaginitis, cervicitis, "strawberry cervix"	Worms may be seen Eggs can be collected on adhesive tape	Thin epithelium, loss of rugae, pale, petechiae	Erythema of vulva	White plaques and "parchment" skin. In a figure-of-eight distribution		Vulvar lichenification, excoriation
Vaginal pH	<4.5	>4.5	>4.5 typically 5–6	<4.5	>4.5	<4.5	<4.5	<4.5	<4.5
Cause	Yeasts	Reduced lactobacilli with overgrowth of anaerobic species	Trichomonads	Nematode infection	Reduced estrogen	Environmental factors	Underlying condition itch-scratch-itch cycle	Unknown inflammatory condition	Unknown likely autoimmune
Treatment	Antifungals, vulvar care	Metronidazole	Systemic antibiotics	Mebendazole	Vaginal estrogens, vulvar care	Remove factor, steroids, vulvar care	Treat underlying condition, steroids, vulvar care	Ultrapotent topical steroids	Ultrapotent topical steroids

- Do not wear panty liners or change them often.
- Do not use soaps, perfumes, or detergents on the vulva, including feminine hygiene or wet wipes. Instead use a soap substitute. Shampoo hair over a basin separate from bath and shower.
- Avoid frequent or prolonged use of hot tubs.

With regard to treatment, when topical steroids are used in addition to an emollient, then the emollient should be used first and the steroid 15 min later. This moisturizes the skin and prevents spread of the steroid onto normal skin. Women should be reassured that atrophy is rare with short-term use of potent corticosteroids.

15 Emotional Care

Patients should be given a detailed explanation of their condition with details of any long-term implications for their own health and that of their partners. They should be offered written information and directed to further resources, such as online websites and/or patient support. Recurrent vulvovaginitis can affect patients psychologically. They are often frustrated and feel despair. The symptoms may have an effect on their sexual relationships and personal relationships.

16 Summary

Vulvovaginitis is the most common reason why women present to a gynecologist. The term covers inflammation or infection of the vagina and/or vulva. Women with symptoms lasting for more than 6 months experience chronic vulvovaginitis. Clinical presentation of women with vulvovaginitis is similar regardless of the underlying cause and so a careful history should be taken. Symptoms include itching, discharge, irritation, dysuria, vaginal odor, rash, and burning. Signs include erythema, edema and excoriation of vulvar skin, ulceration, or chronic vulvar skin changes. The following investigations can aid diagnosis, vaginal pH, amine whiff test, vulvar biopsy, fungal cultures, and wet smears. The commonest causes, their presentation, and the treatment are summarized in Table 1.

17 Conclusion

Obtaining an accurate history from women, with vaginal vulvar symptoms, is critical in diagnosing vulvovaginitis. The results of physical examination and investigation will in turn lead to the underlying cause and therefore determine correct treatment. All women will benefit from good vulvar skin care [clean and dry] and emotional support.

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