9.6 Genital ulceration 1610

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ESSENTIALS Genital ulceration can be caused by many common and rare sexu- ally transmitted infections, dermatological conditions, and trauma. Key to making a specific diagnosis that will direct treatment is a standard sexual and travel history followed by appropriate diag- nostic tests. Genital ulcer disease is a major risk factor for the acqui- sition and transmission of human immunodeficiency virus. Introduction Genital ulceration can represent one of the more complex pres- entations in genito-urinary medicine. The differential diagnosis is wide and encompasses a range of common and rare sexually transmitted infections (STIs), dermatological conditions, and trauma. The list of possible aetiologies can be bewildering (Table 9.6.1), but a standard sexual and travel history will narrow the range of possibilities. Trauma, when acute, is usually easy to recognize, ranging from superficial abrasions to cleanly incised areas. The presence of extragenital lesions, the involvement of other mucous membranes, and a relapsing course might direct attention to dermatological causes. However, breaks in the skin can act as a portal for infection and careful review and further testing might be required if the individual is also at risk of STIs. Various classifications and algorithms are proposed to help iden- tify pathogens and guide immediate management but care must be taken when working through these. Infectious agents can often be present together and the variety of presentations makes classical disease the exception. In recent years, the importance of genital ulcer disease as a major risk factor for the acquisition and transmission of HIV has been recognized. Many of the rarer pathogens such as the L serovars of Chlamydia trachomatis (the cause of lymphogranuloma venereum) and Treponema pallidum (the causative agent of syphilis) circulate at much higher levels in those communities with HIV and their presence should always trigger concerns around risk-taking behaviours. Both initial ex- clusion and carefully timed follow-up are required to exclude HIV in those at risk. Herpes simplex virus infection Herpes simplex infection causes acute and chronic recurrent mucocutaneous ulceration. Both herpes simplex virus type 1 (HSV1) and herpes simplex virus type 2 (HSV2) can cause genital ulceration. Genital acquisition requires direct inoculation into the local epithelium. The usual route for HSV1 is orogenital contact and for HSV2 is genital-to-genital contact. Only one-third of ini- tial acquisitions are symptomatic and these tend to be worse in those that have not been previously infected with the other virus type. It also tends to be worse in females, patients with diabetes, and those with immunocompromized states. Incubation tends to be one to three weeks. Classical presentations with bilateral mul- tiple superficial lesions, lymphadenopathy, and systemic features of myalgia and headache make up the minority of cases. Left un- treated, the episode peaks in severity at 11 days and typically lasts three weeks. Particularly severe episodes with urinary retention and a variety of local neurological complications including auto- nomic dysfunction, radiculitis, and meningeal inflammation can occur.

Disseminated infection is rare. Oral antiviral therapy with aciclovir or valaciclovir is recommended immediately on suspicion of infection. The diagnosis can have significant ad- verse psychosexual impact and it is advisable to confirm all clin- ical diagnoses with a sensitive laboratory method (polymerase chain reaction (PCR) being 30–50% more sensitive then culture). Laboratory viral typing of HSV1 or HSV2 will provide useful in- formation for both prognostication and counselling. Most pa- tients with symptomatic acquisition of HSV will have recurrences with HSV1 recurring less than once a year and HSV2 recurring 4–6 times on average. Up to 10% of patients will have more than 10 episodes per year. Many patients with genital HSV will only present at the time of a recurrence (having had an unnoticed acquisition episode) and care must be taken in declaring any first episode as being re- cently acquired. Recurrences are typically unilateral, sort lived (five days) and milder than first episodes. Many recurrent epi- sodes will be mild and unnoticed by the patient; asymptomatic, subclinical infectious viral shedding can occur and accounts for most transmissions of the virus. Troublesome recurrent disease 9.6 Genital ulceration Patrick French and Raj Patel

9.6 Genital ulceration 1611 (both physical and psychological) responds well to continuous suppressive antiviral medication. This can also be used to modify transmission risk. Syphilis Syphilis is caused by the spirochaete bacterium Treponema pal- lidum subsp pallidum and the early stages of infection are char- acterized by genital ulceration. Ulceration occurs at the site of inoculation with the usual transmission route being orogenital or genital–genital contact. The period between the acquisition of infection and the development of ulceration is between 9 and 90 days, however most individuals develop ulcers two to three weeks after exposure. The initial lesion is a papule or papules, which then ulcerate. Typically the ulcers are indurated and painless and are often single, however they can be multiple and painful. The ulcers are associated with local lymphadenopathy. This stage of syphilis is called primary syphilis with the ulcera- tive lesion called a syphilitic chancre. This ulcer heals spontan- eously after three to four weeks and might then be followed, three to six weeks later, by the more generalized symptoms and signs of secondary syphilis. Secondary syphilis is characterized by a rash and constitutional symptoms such as fever and malaise, but might also be accompanied by mucosal ulceration affecting both the mouth and anogenital area. These ulcers also heal spon- taneously without treatment, but in some cases can be followed by relapsing episodes of secondary syphilis over the following months. The diagnosis of syphilis-associated genital ulceration is made by undertaking a careful clinical assessment and by performing appropriate diagnostic tests. In specialist services, tests to dir- ectly identify T. pallidum in material from the ulcer might be available but, in most settings, clinical assessment followed by serological tests for syphilis remain the cornerstones of diag- nosis. Dark ground microscopy is a point-of-care test in which a sample of fluid from the base of an ulcer is placed on a saline drop and examined using a phase contrast microscope. T. pall- idum is a motile organism and can be identified by its charac- teristic morphology and movement. However, this technique is highly observer-dependant and is only used in specialist centres. Increasingly, Treponema pallidum PCR tests are being em- ployed to diagnose early syphilis when it presents as genital ul- ceration and is highly sensitive and specific. This PCR test can be combined in multiplex tests with herpes simplex type 1 and 2 and, if appropriate, Haemophilus ducreyi PCR tests allowing a single swab to be taken to diagnose all four major causes of STI- associated ulceration. These tests are not available in most set- tings and the mainstay of diagnosis is clinical serological testing for syphilis. The screening test of choice for syphilis in the United Kingdom and the first serological test to become positive after acquiring syphilis is the Treponema pallidum EIA IgM/IgG test, which becomes positive two to three weeks after acquiring syph- ilis. In early primary syphilis, this test

can be negative. If syphilis is suspected, a syphilis PCR test is not available and empirical treatment for syphilis is not being given, an initial negative sero- logical test for syphilis should be repeated two to three weeks later to exclude infection. The diagnosis of secondary syphilis is much more straightforward, as serological tests are invariably strongly positive (see Chapter 8.6.37). Chancroid This bacterial STI caused by Haemophilus ducreyi is predom- inantly seen in the tropical countries of Africa, Asia, South America, and the Caribbean. Outbreaks in closed, relatively iso- lated, European and North American communities have also been reported. Lesions develop within 3–10 days. Typically these are single or multiple nonindurated ('soft sore') painful ano- genital ulcers with a purulent base with contact bleeding. There is usually painful (mostly unilateral) inguinal lymphadenopathy. Complications include tissue destruction (phagedenic ulcer- ation), inguinal abscess formation (bubo) and chronic suppura- tive sinuses. Diagnosis with traditional methods of microscopy or culture have poor sensitivity, while PCR is the most sensitive and becoming increasingly available. Azithromycin 1 g single dose or ceftriaxone 250 mg IM single dose, or ciprofloxacin 500 mg orally twice daily for three days are the mainstays of treatment. Lymphogranuloma venereum Lymphogranuloma venereum (LGV) is caused by an inva- sive L serovar of Chlamydia trachomatis. It is characterized by Table 9.6.1 Causes of genital ulceration Trauma Sexually transmitted agents • Chancroid—Haemophilus ducreyi • Donavanosis—Klebsiella granulomatis • Lymphogranuloma venereum—Chlamydia trachomatis (LGV strains –L serovars) • Syphilis—Treponema pallidum subp pallidum • Herpes simplex Non-STI infective agents • Candida • Herpes zoster • Epstein–Barr virus Dermatological conditions • Erythema multiforme • Behçet's disease • Aphthosis • Crohn's disease • Drug reactions, e.g. Stevens-Johnson syndrome, fixed drug eruptions • Phemphigus • Phemphigoid • Balanitis xerotica obliterans/Lichen sclerosus • Skin malignancies

 lymphangitis and inguinal lymphadenopathy together with systemic symptoms. LGV more recently has emerged as an im- portant cause of proctitis among men who have sex with men. Most individuals with LGV present with either the inguinal syn- drome (inguinal lymphadenopathy) or proctitis; however, some present with a primary ulcerative lesion at the site of inoculation. In studies looking at the frequency of the primary lesion, as few as 3% or as many as 53% of individuals with LGV recall having a genital ulcer in the early stages of infection. The ulcer usually starts as a single painless papule 3–12 days after sexual exposure, which then ulcerates. It can occasionally be painful and multiple. It heals spontaneously. LGV is diagnosed clinically and by testing for C. trachoma- tis. In some settings, such as in the United Kingdom, the DNA from C. trachomatis identified from ulcer or genital specimens can be amplified and sequenced to determine whether it is an LGV-associated L serovar. Serological tests for LGV are avail- able, but are of limited use (see Chapter 8.6.45) in routine clinical practice. Granuloma inguinale (Donovanosis) Granuloma inguinale (Donovanosis) is a bacterial infection caused by Klebsiella granulomatis. It is rare outside resource poor coun- tries. It is characterized by genital ulceration and granulomata which usually appear in the genital area 10–14 days after initial in- oculation. The lesions are initially small and painless and are often raised and nodular, but may present as depressed necrotic ulcers. These nodules can coalesce to form fleshy moist genital plaques. Diagnosis is often made clinically after careful history taking (including travel history) and can be confirmed by tissue biopsy, which reveals Donovan bodies with Wright-Giemsa staining (see Chapter 8.6.10). FURTHER READING BASHH. UK Guidelines. http://www.bashh.org/guidelines/

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