Genital Dermatology

10

Alok Vij, Sarah C. Vij, and Kenneth J. Tomecki

10.1 Inflammatory

10.1.1 Fixed Drug Eruption

Fixed drug eruption is a common hypersensitivity reaction to medications, most commonly analgesics, including ibuprofen, aspirin, and paracetamol. The reaction occurs more commonly in men [1] and can occur as a "sexually transmitted" eruption if the patient's partner has ingested the suspected allergen [2]. Repeated ingestion of the allergen induces recurrence of the eruption in the same distribution.

The eruption is usually a well-defined, oval or circular, violaceous, or red-brown plaque (Fig. 10.1). Upon ingestion of the offending agent, the eruption recurs in the same location, commonly the genitalia, lips, trunk, and hands [3]. Occasionally, a vesicle or bulla may arise within the plaque, but ulceration and necrosis are uncommon [4].

The eruption may be painful or itchy and may be accompanied by systemic symptoms, including fever, chills, malaise, nausea, and vomiting. Treatment relies on identifying and discontinuing the causative agent. Antihistamines and topical corticosteroids help to reduce pruritus and hasten the resolution of the eruption, which typically fade within 2–3 weeks followed post-inflammatory hyperpigmentation [3].

A. Vij, M.D. (✉) • K.J. Tomecki, M.D.
Department of Dermatology, Cleveland Clinic Foundation, Dermatology and Plastic Surgery Institute, 9500 Euclid Avenue, Desk A61, Cleveland, OH 44195, USA
e-mail: vija@ccf.org

S.C. Vij, M.D.
Department of Urology, Cleveland Clinic, Cleveland, OH USA

J.M. Potts (ed.), *Men's Health*, DOI 10.1007/978-1-4939-3237-5_10

Fig. 10.1 Ulcerative fixed drug eruption. Upon ingestion of ibuprofen, this patient developed a painful red plaque of the glans that ulcerated with further doses of ibuprofen. A second course of ibuprofen months later caused a recurrence of the ulcer

10.1.2 Allergic/Irritant Contact Dermatitis

Contact dermatitis can be allergic or irritant in nature. Causes of allergic contact dermatitis of the male genitalia include sensitivity to condoms, diaphragms, spermicides, lubricants, and topical medications. Sensitivity to other allergens, including industrial or plant allergens, may induce genital dermatitis by "hand transfer." Affected patients usually have sharply demarcated erythematous and edematous plaques on the glans, penile shaft, scrotum, and occasionally the thighs or mons pubis (Fig. 10.2). The eruption of allergic contact dermatitis is pruritic more commonly than painful; irritant dermatitis is more commonly painful than itchy.

The most common contact allergens in condoms are tetramethylthiuram, mercaptobenzothiazole, and dithiocarbamates [5]. Patients allergic to tetramethylthiuram and mercaptobenzothiazole should use Trojan brand condoms or nonrubber condoms made from sheep or lamb intestines. Patients using the latter should be counseled on the reduced efficacy of preventing the spread of sexually transmitted infections compared to latex condoms [6]. Other condom-related contact sensitizers include lubricants or anesthetics such as benzocaine [7].

The five most common causes of non-condom-related allergic contact dermatitis of the genitalia are balsam of Peru, fragrance, tolu balsam, phenylmercuric acetate, and neomycin [8]. The preservatives methylisothiazolinone and methylchloroisothiazolinone are increasing in frequency and severity as cause of genital contact

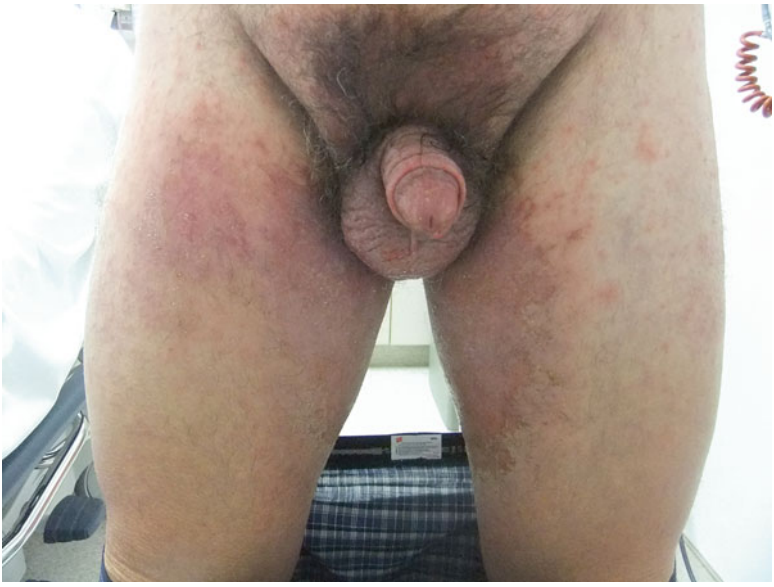

Fig. 10.2 Impetiginized allergic contact dermatitis. After using triple antibiotic ointment to prevent an infection, this patient developed a symmetric, pruritic eruption. Note the honey-colored crusting, suspicious for secondary impetiginization, superimposed upon the erythematous plaques

dermatitis [9]. Less common causes include topical medications such as corticosteroids, antibiotics other than neomycin, or diphenhydramine [10].

Propylene glycol is the relevant allergen in K-Y Jelly, while parabens are the allergens in other lubricants [11, 12]. Alternative products include the aqueous lubricant Surgilube, which does not contain propylene glycol, or SKYN brand condoms, containing non-sensitizing silicone-based lubricant [13].

One allergen frequently transferred from the hands to the genitalia is urushiol, the allergen responsible for Rhus dermatitis, commonly known as "poison ivy" [13]. Urushiol, present in poison ivy, oak, and sumac, can be transferred over 2–3 weeks after initial exposure by oil remaining under the fingernails, clothing, or from pets that had contact with the plants.

Affected patients should wash thoroughly with soap and water within 60 min of plant contact, and clothing should be promptly laundered. Topical steroids can alleviate limited outbreaks, but systemic steroids are needed for generalized or severe outbreaks. Prednisone 1 mg/kg body weight tapered slowly over 2–3 weeks usually suffices; secondary infection is common and may be detected by foul odor, pain, or increasing discharge.

Irritant contact dermatitis is a common cause of chronic or recurrent balanitis that may occur after physical or chemical damage to the skin. Detergents and soaps more commonly than antiseptics or disinfectants cause skin irritation after their use to prevent sexually transmitted infections. Atopic history or frequent washing of the genitalia can induce or aggravate balanitis [14, 15].

10.1.3 Plasma Cell Balanitis (Zoon's Balanitis)

Plasma cell balanitis (Zoon's balanitis) is an inflammatory disease of the penis named after its histologic findings. The balanitis is a persistent, shiny, red plaque on the glans, corona, or mucosal surface of the prepuce in older uncircumsized men. Hemosiderin deposition may yield specks of "cayenne pepper" within the larger plaque (Fig. 10.3) [16]. Usually asymptomatic, the plaques may occasionally be itchy, tender, or painful. Predisposing factors include infection with human papillomavirus (HPV) or *Mycobacterium smegmatis*, heat, occlusion, friction, and poor hygiene [17, 18].

Biopsy is important to differentiate the benign inflammation of Zoon's balanitis from the premalignant or malignant conditions of erythroplasia of Queyrat and squamous cell carcinoma [19]. Histopathology of Zoon's balanitis displays an abundant infiltrate of plasma cells without squamous atypia [20]. In rare cases of long-standing balanitis with coinfection with HPV, squamous cell carcinoma may arise [21].

Definitive treatment is circumcision. Alternative treatments include topical corticosteroids, topical calcineurin inhibitors, cryotherapy, and ablative laser therapy [17, 18, 22].

10.1.4 Vitiligo

Vitiligo is an inflammatory skin disease marked by autoimmune destruction of melanocytes. The resultant depigmented patches can be accentuated by Wood's light examination or by sun exposure, increasing the contrast between normal and

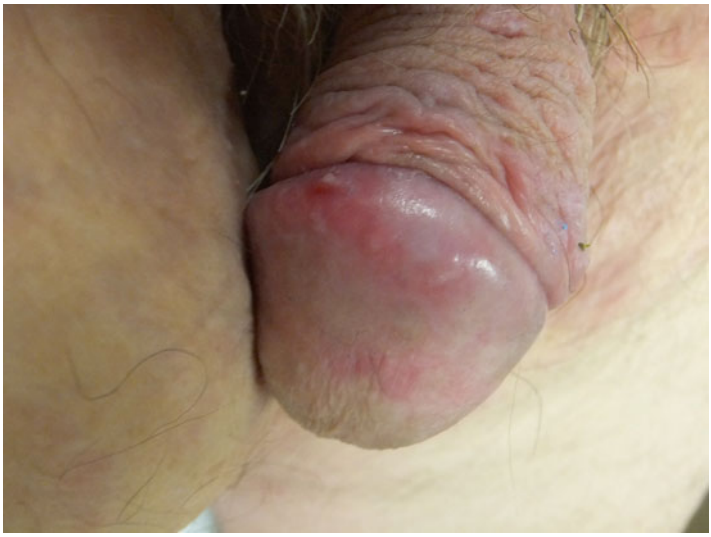

Fig. 10.3 Zoon's balanitis with focal squamous cell carcinoma in situ. The erythematous, shiny plaque persisted despite topical steroids and topical calcineurin inhibitors. A keratotic papule developed at the margin of the plaque; biopsy revealed squamous cell carcinoma in situ

Fig. 10.4 Vitiligo. This patient had generalized depigmented patches of the face, trunk, extremities, and genitals. He responded to UV-B phototherapy

affected skin. Vitiligo has several classic patterns: genital (Fig. 10.4), perioral, segmental, linear, and a "confetti-like" distribution of the extremities [23]. Vitiligo must be differentiated from chemical leukoderma, which may be induced by occupational exposures [24] or immunomodulatory therapy [25].

Treatment of vitiligo is based on the distribution. Several effective brands of camouflage cosmetics can be used in the absence of other therapies. Focal disease can be treated with a combination of topical calcineurin inhibitors and vitamin D analogues. Topical steroids can be used when cost is a limiting factor, but corticosteroid use has been associated with iatrogenic hypopigmentation [26].

Sun protection is advised due to the increased risk of ultraviolet (UV) damage to the skin due to loss of the pigment network [27]. However, UV-B phototherapy or 308 nm xenon chloride (excimer) laser therapy can be used for large areas or as adjunctive therapy for resistant disease [28]. Repigmentation typically begins around the hair follicles, creating a salt-and-pepper-like distribution at the periphery of the affected areas.

10.2 Papulosquamous Diseases

10.2.1 Lichen Simplex Chronicus

Lichen simplex chronicus (LSC) is an overarching term that encompasses pruritus scroti and chronic dermatitis. It is a benign but uncomfortable condition which may arise from pruritus of neuropathic origin [29]. Known as "the itch that rashes," LSC often results from chronic itching or rubbing that leads to lichenification of the skin and an ever-worsening itch-scratch cycle [30]. Primary LSC starts on normal skin, while secondary LSC begins on skin affected by another primary process, such as atopic dermatitis.

Examination reveals lichenified plaques of the scrotum with broken hairs and dyspigmentation; history should focus on hygienic practices and the current use of topical medications, which may be inciting or aggravating factors. Acute pruritus scroti is more commonly caused by infections than neurodermatitis. Allergic/irritant contact dermatitis should be considered in the differential diagnosis [31], and gentle care with hypoallergenic products should be recommended.

Treatment is difficult, including topical steroids or topical calcinuerin inhibitors [32], systemic antihistamines, and inhibition of manipulation with occlusion. Refractory or recalcitrant disease may require antidepressant medications [31] or injection of botulinum toxin [33].

10.2.2 Psoriasis

Psoriasis is a chronic, multisystem inflammatory disorder most commonly affecting the skin and joints, with a known risk of associated obesity and cardiovascular disease [34]. Psoriasis of the genitalia, or inverse psoriasis, affects approximately 50 % of men with psoriasis in the course of their disease [35].

Psoriasis is the most common inflammatory disease of the penis [36], typically exhibiting pink, shiny patches or plaques (Fig. 10.5). Patients are usually asymptomatic, except for occasional sensitivity during intercourse. Genital psoriasis has a negative impact on quality of life due to this sexual dysfunction [37, 93].

Treatment consists of mild topical corticosteroids often with adjuvant vitamin D analogues, topical calcineurin inhibitors, or coal tar preparations for recalcitrant disease [35]. Widespread or severe disease may benefit from systemic therapy, including methotrexate, retinoids, or biologic agents.

10.2.3 Lichen Planus/Lichen Nitidus

Lichen planus (LP) is an inflammatory disease of squamous epithelia characterized by pruritic, purplish, polygonal papules with an overlying fine, white, reticulated striations. LP affects less than 1 % of the population, but 25 % of affected patients have isolated genital disease, and 20–25 % have both oral and genital disease [38].

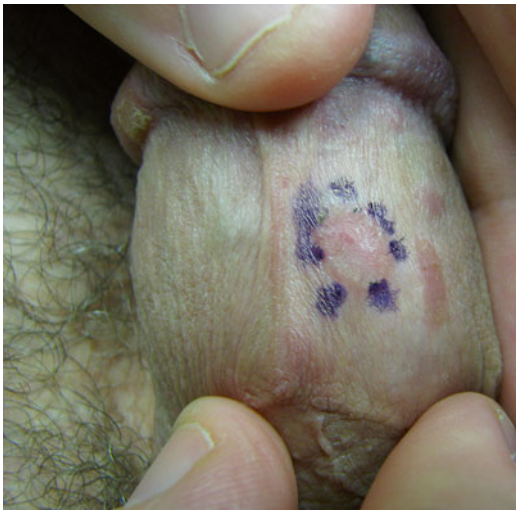

Fig. 10.5 Penile psoriasis. Inverse psoriasis typically lacks induration and scale classic for psoriatic plaques elsewhere on the body

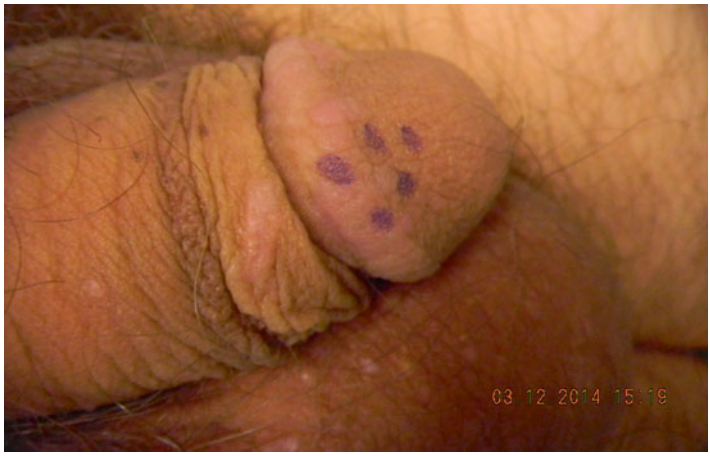

Fig. 10.6 Lichen planus. Wickham's striae are lacy, reticulated thin white plaques that overlie purplish papules and plaques of lichen planus and can be helpful to clinically distinguish lichenoid eruptions from other papulosquamous diseases

Genital LP has a varied morphology, from polygonal papules to annular plaques (Fig. 10.6). Erosive disease is common, with a rare risk of transformation into SCC [39]. Symptoms include pruritus, burning, and sensitivity during intercourse. Exacerbation of pain in concert with enlarging papules or ulceration may indicate the development of malignancy.

Aggressive treatment is indicated given the negative effect on quality of life, including skin disease-related stress and depression [40]. Treatment starts with topical steroids [41]. Topical pimecrolimus is an appropriate steroid-sparing agent; tacrolimus should be avoided as it can induce a burning sensation [42].

Lichen nitidus is an uncommon inflammatory skin disease that exhibits small, monomorphous skin-colored to white papules, which are usually asymptomatic but may be pruritic and may be a significant cosmetic concern to patients or their partners.

Treatment consists of topical steroids for the pruritus and appearance [43]. Cryotherapy, retinoids, or UV-B phototherapy have also been beneficial [44].

10.2.4 Balanitis Xerotica Obliterans

Balanitis xerotica obliterans (BXO) is the rarer, male variant of lichen sclerosus et atrophicus. Characterized by white, atrophic plaques on the glans penis, prepuce, and coronal sulcus, progressive BXO may lead to phimosis and urethral stenosis. Affected patients have a 5 % risk of developing SCC within long-standing plaques [45].

BXO starts insidiously with a burning or stinging pain, often with urethral discharge. Later, phimosis, dysuria, or voiding difficulties may occur [46]. Treatment involves potent topical steroids. Ablative laser therapy may help resistant disease of the glans [47]. Phimosis requires circumcision. Urinary symptoms merit urologic evaluation, especially for urethral stenosis. Interventions may include meatotomy, meatoplasty, or urethroplasty [46, 48].

10.2.5 Miscellaneous Inflammatory Diseases

Patients with a widespread or generalized skin disease may have genital involvement. For example, tense bullae or erosions may be present in bullous pemphigoid (Fig. 10.7), pemphigus vulgaris (Fig. 10.8), or cicatricial pemphigoid.

Additionally, systemic diseases may affect the genitalia. Crohn's disease may present with episodic swelling and induration of the penis or scrotum, particularly in pediatric patients (Fig. 10.9). Sarcoidosis or IgG4-related disease may present with similar periodic infiltration of the genitalia. Histopathology would help to differentiate these conditions.

10.3 Infectious

10.3.1 Human Papillomavirus

Human papillomavirus (HPV) is the cause of genital warts, a condition that affects 1 % of the US population. The prevalence of genital warts is highest among adults ages 18–28 years, with HPV serotypes 6 and 11 accounting for 90 % of warts [49]. Warts are typically asymptomatic, but can be pruritic, painful, and occasionally may cause bleeding and discharge if the urethra is affected.

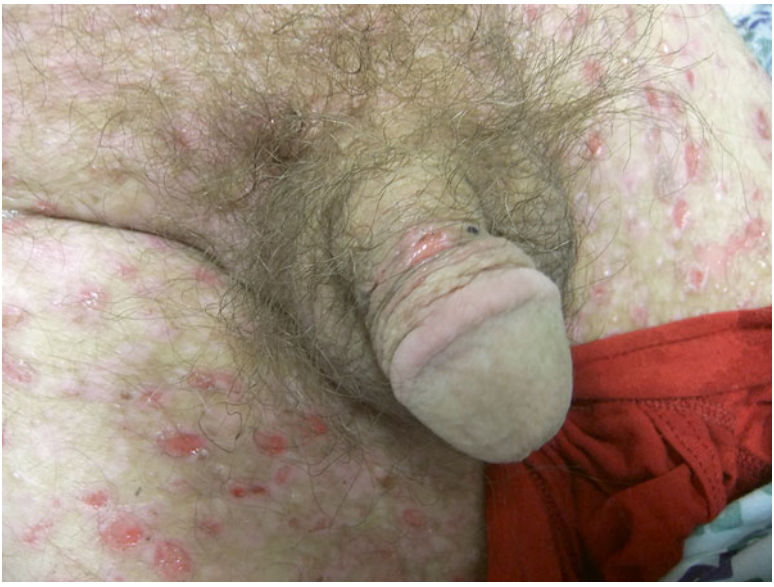

Fig. 10.7 Bullous pemphigoid. Tense bullae and urticarial plaques erupted diffusely over this patient's body. Due to friction, many of the bullae were eroded. Image courtesy of Dr. Sean Condon

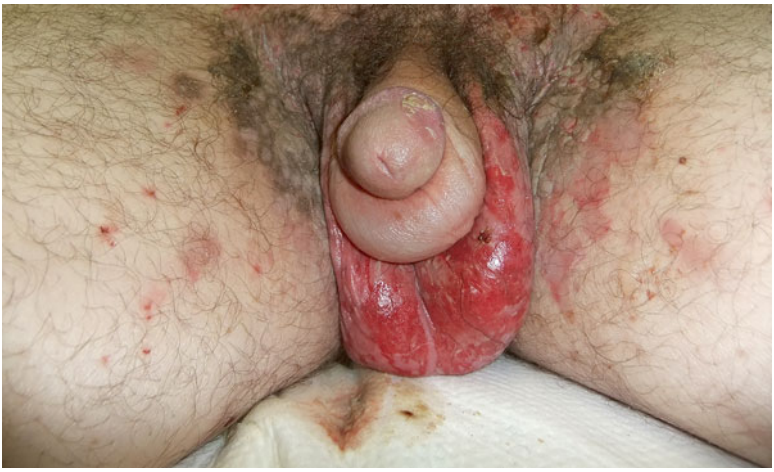

Fig. 10.8 Pemphigus vulgaris. Erosions with crusting affect the skin and mucosae of this patient. Scrotal involvement is common in pemphigus vulgaris

HPV warts are skin-colored to dark brown verrucous papules that may be sessile or pedunculated, distributed on the glans penis, scrotum, perineum, and perianal skin (Fig. 10.10). Clinical morphology usually establishes the diagnosis; biopsy may be diagnostic and therapeutic in certain circumstances.

Fig. 10.9 Metastatic Crohn's disease. This patient had several episodes of painful swelling of his penis and scrotum. Biopsy revealed granulomatous infiltration suggestive of Crohn's disease. Esophagogastroduodenoscopy and colonoscopy revealed stricture formation and granulomatous inflammation in several locations throughout the GI tract

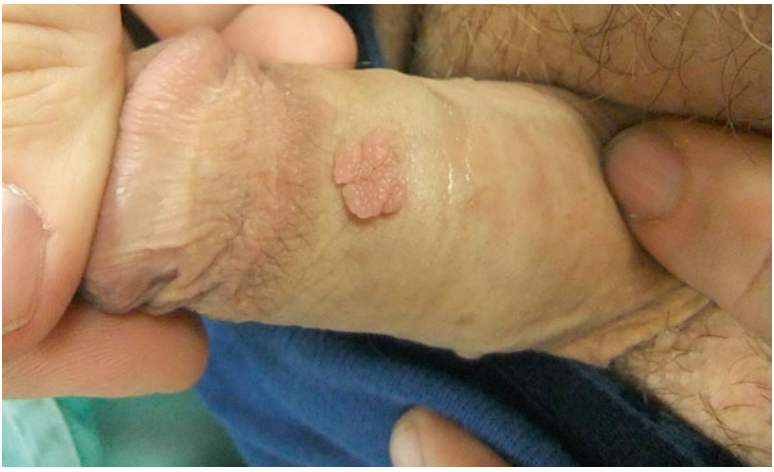

Fig. 10.10 Genital warts. This pedunculated verrucous papule was removed by shave biopsy, a potentially diagnostic and therapeutic procedure

Treatment options are chemical or physical destruction, immunologic therapy, and surgical therapy. Chemical options include the application of podophyllotoxin and 80 % trichloroacetic acid; physical treatments include cryotherapy and curettage. Topical 5-fluorouracil and imiquimod can be used for field therapy. Surgical

options include shave excision and CO_2 laser ablation, with caution related to the aerosolization of viral particles.

Two vaccines, one bivalent (Cervarix, GlaxoSmithKline) and the other quadrivalent (Gardasil, Merck & Co., Inc), offer protection against HPV types 6 and 11. The quadrivalent vaccine also protects against serotypes 16 and 18, which account for approximately 70 % of cervical cancers.

10.3.2 Herpes Simplex Virus

Herpes simplex virus (HSV) can produce oral and genital ulcerative disease. Historically, HSV1 caused oral disease, while HSV2 caused genital disease. Today, both types produce disease in both locations, and the incidence of genital HSV1 disease is increasing [50]. HSV infection presents as shallow, well-circumscribed, painful ulcers often with adenopathy (Fig. 10.11), but 85–90 % of infected individuals are asymptomatic [51].

Morphology suggests the diagnosis, but viral culture or direct fluorescent antibody screen can be used to confirm the diagnosis. Treatment for the initial episode is with either acyclovir 200 mg five times per day, valacyclovir 1 g twice daily, or famciclovir 250 mg three times daily, each for 10 days. For recurrent episodes, acyclovir 400 mg three times per day for 5 days, valacyclovir 500 mg twice daily for 3–5 days, or a single dose of famciclovir 1000 mg is recommended. Chronic suppression for patients with more than six outbreaks per year consists of acyclovir 400 mg twice daily, valacyclovir 500 mg once to twice daily, or famciclovir 250 mg daily. These three antivirals have shown to be equivalent in their efficacy in presenting recurrences [52]; medication cost and expected compliance with the prescribed regimen may dictate the choice of therapy.

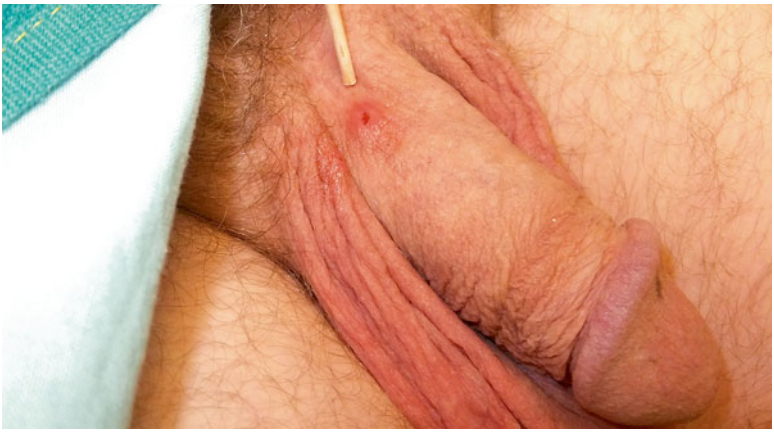

Fig. 10.11 Herpes simplex virus. This patient developed a painful ulcer on the shaft of the penis. Viral culture confirmed HSV-1 infection

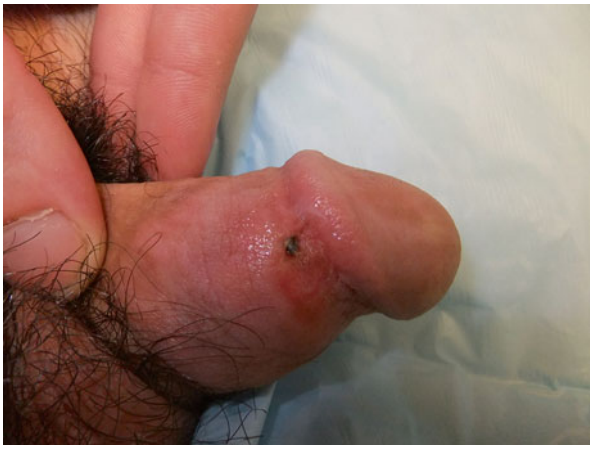

Fig. 10.12 Syphilitic chancre. The chancre is a painless, indurated ulcer that often presents with regional lymphadenopathy

10.3.3 Syphilis

Syphilis is a venereal disease caused by the spirochete *Treponema pallidum*. The rate of reported primary and secondary syphilis in the United States is 5.3 cases per 100,000, double the rate in 2001 [53]. When untreated, syphilis proceeds through three stages: primary, secondary, and tertiary.

The primary infection occurs 10–90 days after exposure, initially presenting with a well-circumscribed, painless, indurated ulcer with regional lymphadenopathy (Fig. 10.12). Most commonly, the ulcer occurs on the prepuce or glans penis, but extragenital presentation may occur depending on the site of inoculation. The secondary stage occurs 3–10 weeks later and manifests classically as copper-colored macules and papules on the trunk, palms, and soles, along with oral ulcers, "moth-eaten" alopecia, condyloma lata, and flu-like symptoms.

The genital ulcer should suggest primary syphilis, which can be confirmed by dark-field examination and rapid plasma regain (RPR) or Venereal Disease Research Laboratory (VDRL) assay. A second confirmatory test for treponema-specific antibodies such as the fluorescent treponemal antibody absorption (FTA-ABS) assay or biopsy can help to confirm the diagnosis. Anti-treponemal immunohistochemical stains are available to aid in the histopathologic diagnosis.

A single dose of 2.4 million units benzathine penicillin is the treatment of choice for primary syphilis. In the setting of penicillin allergy, a single dose of azithromycin 2 g by mouth or doxycycline 200 mg daily for 14 days should be administered.

10.3.4 Candida

Candida albicans is a common cause of balanitis. Approximately 15–20 % of males are asymptomatic carriers, although sexual transmission of *Candida* is not implicated in the pathogenesis of *Candida* vulvovaginitis [54, 55]. True *Candida* balanitis accounts for approximately one-third of all cases of balanitis [56, 57].

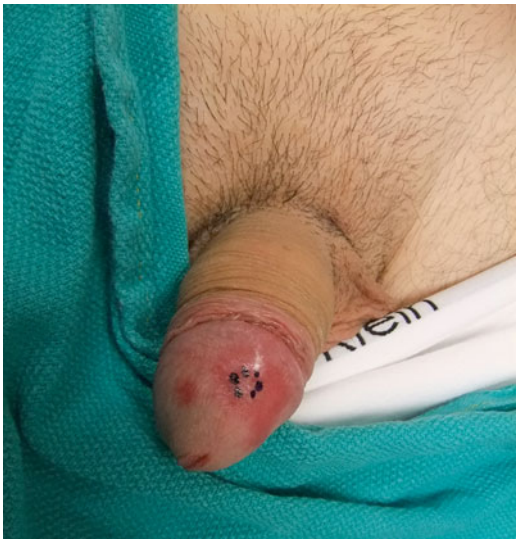

Fig. 10.13 Candidal balanitis. Punch biopsy of this red plaque with a satellite papule revealed Candidal forms in the stratum corneum, confirming the diagnosis of Candidal balanitis. Oral fluconazole was an effective treatment

Risk factors include uncircumcised state, phimosis, incontinence, poor hygiene, advanced age, malnutrition, antecedent use of antibiotics, diabetes, obesity, and intercourse with women with *Candida* vulvovaginitis [58]. Patients complain of pain, pruritus, or burning.

Physical examination reveals glazed, red plaques of the penis, scrotum, or thighs with "satellite papules or pustules" at the periphery (Fig. 10.13). Erosions, crusting, and maceration may occur secondarily. In severe, untreated disease or in immunocompromised individuals, phimosis or ulceration can occur. Diagnosis can be confirmed by potassium hydroxide (KOH) examination, which will reveal pseudohyphae [57].

Treatment includes optimization of hygiene, using compresses followed by an anti-yeast cream (e.g., clotrimazole) two to three times a day for 2–3 weeks. Oral fluconazole, 150–200 mg, in a single dose is beneficial [59]. Recalcitrant or recurrent disease may warrant culture with sensitivities, as resistant species of *Candida glabrata* are emerging [60].

10.4 Neoplasia

A variety of growths or tumors can affect the genitalia, and certain entities have a predilection for the penis or scrotum. Pearly penile papules and angiokeratomas are two of the most common benign neoplasms with localization to the penis and scrotum, respectively.

Squamous cell carcinoma and melanoma occur infrequently on the genitalia [61]. Extramammary Paget's disease and Buschke-Lowenstein tumor occur almost exclusively on the genitalia [62]. Cutaneous metastases from genitourinary or gastrointestinal cancers commonly affect the genitalia.

10.4.1 Pearly Penile Papules

Pearly penile papules affect young men, with incidence decreasing with age. They are asymptomatic, 1–2 mm, dome-shaped, pink to skin-colored, smooth papules. They usually arise in a single row on the coronal sulcus of the penis, although they can arise in doubles, on the glans or penile shaft. They may vary in shape, size, and color from person to person, but are invariably uniform in an individual [63].

Pearly penile papules are benign and do not represent a venereal infection, although patients often believe they are genital warts. Biopsy can differentiate those entities, but treatment is based on reassurance of the noninfectious, benign nature of the neoplasms [14].

10.4.2 Angiokeratomas

Angiokeratomas are proliferations of capillaries within the papillary dermis and epidermis that appear as dark red to black papules, usually on the scrotum, penile shaft, or medial thigh (Fig. 10.14). Angiokeratomas may occur in adults in association with varicocele, inguinal hernia, or thrombophlebitis. Bleeding may occur spontaneously or after trauma [64].

Treatment is not necessary unless bleeding, discomfort, or cosmetic concern dictate otherwise. Papules can be treated with excision, cryotherapy, electrocautery, or laser techniques [65].

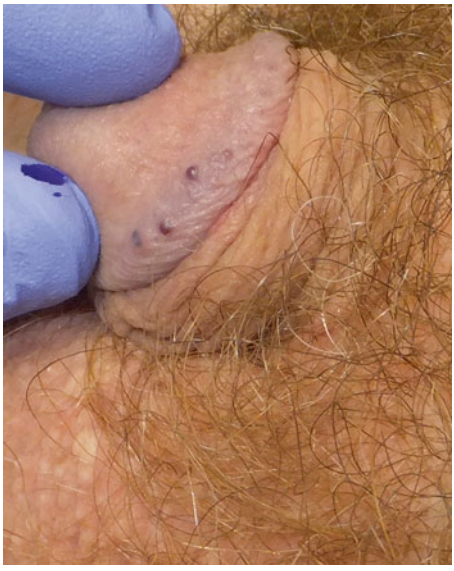

Fig. 10.14 Angiokeratomas. This patient's partner requested dermatologic examination because she was concerned that these papules were warts

10.4.3 Erythroplasia of Queyrat and Squamous Cell Carcinoma

Erythroplasia of Queyrat (EQ), akin to SCC in situ, is a precancerous condition that may evolve into invasive SCC. EQ affects uncircumcised men in the fifth decade or later as solitary or multiple, well-circumscribed, moist, erythematous plaques covering the glans or prepuce [66]. Pain, itching, or bleeding can occur, and retraction of the foreskin may be painful or difficult. High-risk HPV types and chronic irritation from poor hygiene, friction, trauma, and retained smegma may be causative or aggravating factors [66–68].

Penile cancer is rare in circumcised men and in the United States, but represents 10–20 % of all cancers in men in South America, Asia, and Africa [69, 70]. Risk factors for penile cancer include smoking, psoralen treatment, genital warts, phimosis, multiple sexual partners, delayed or absent circumcision, lichen planus, or balanitis xerotica obliterans [71–73]. Increasing pain or ulceration may herald malignant invasion (Fig. 10.15).

Scrotal SCC, usually a slow-growing papule that ulcerates, is rare domestically; however, specific exposures account for the high incidence in certain African tribesmen, chimney sweepers, and industrial oil workers around the globe [74].

Treatment is based on size and location of the cancer. Treatment options include circumcision, cryosurgery, excision, and Mohsmicrographicsurgery [66, 75].

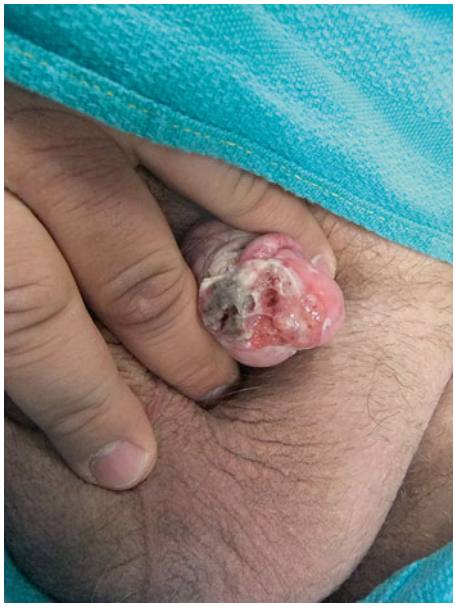

Fig. 10.15 Squamous cell carcinoma of the penis. This patient required penectomy for this long-standing indurated ulcer of the penis

10.4.4 Melanoma

Genital melanoma is rare, most commonly occurring in the sixth or seventh decade. Genital melanoma begins as an enlarging pigmented patch or plaque with variegated color (blue-black, black, brown, or reddish brown) that often ulcerates [76]. It represents less than 10 % of all genitourinary cancers and less than 10 % of all melanomas [77]. Two-thirds of all cases occur on the glans and less frequently on the prepuce, urethral meatus, penile shaft, or coronal sulcus [78–81].

At the time of diagnosis, 60 % of patients have distant metastases [82]. Treatment is wide excision or Mohs micrographic surgery, followed by adjunctive radiotherapy or chemotherapy. Lymph node resection remains controversial [78]. Genitourinary melanoma has the lowest survival rate of all melanomas.

10.4.5 Extramammary Paget's Disease

Extramammary Paget's disease (EMPD) primarily affects women, except in eastern Asians [83]. EMPD begins innocuously as a solitary, well-demarcated, moist eczematous plaque that is often likened to "strawberries and cream" (Fig. 10.16) [84]. The differential diagnosis includes lichen simplex chronicus, psoriasis, SCC, and cutaneous T-cell lymphoma [85].

Primary EMPD is an adnexal adenocarcinoma of the skin without underlying malignancy; secondary EMPD is related to an adjacent internal cancer. About 75 %

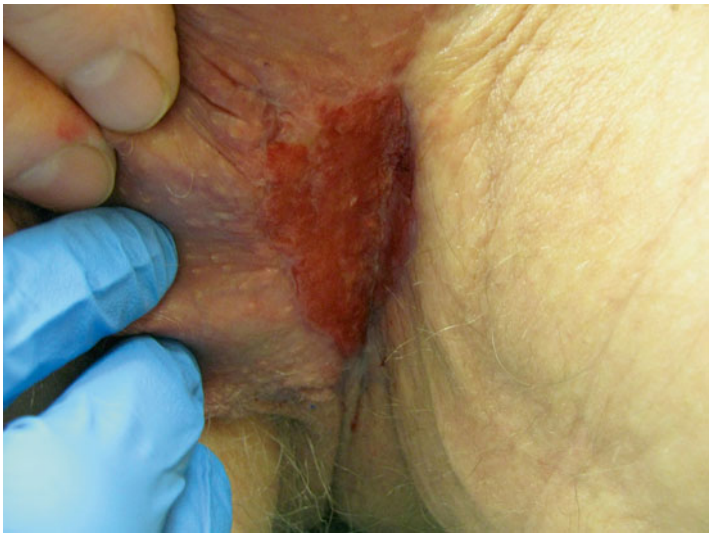

Fig. 10.16 Extramammary Paget's disease. After work-up, this tumor was confined to the skin only. Immunomodulatory therapy with imiquimod initially cleared the plaque, but disease recurrence was noted 1 year later. Mohs micrographic surgery was performed, and the patient has remained disease free after 3 years. Images courtesy of Dr. Jon Meine

of EMPD is primary, and 25 % is secondary [86]. Affected patients deserve screening for distant cutaneous disease as well as underlying visceral malignancy.

Primary EMPD can be treated with topical chemotherapy, e.g., 5-fluorouracil, or topical immunomodulatory therapy, e.g., imiquimod, or Mohs micrographic surgery, although the disease tends to recur [87, 88]. Treatment of secondary EMPD is directed at the underlying malignancy.

10.4.6 Verrucous Carcinoma (Buschke-Lowenstein Tumor)

Buschke-Lowenstein tumor is a rare neoplasm of the anogenital region with a benign histological appearance but locally aggressive behavior. HPV type 6 and 11, traditionally thought to be low risk for malignant potential, are the causative agents [89]. The exact pathogenesis of the locally aggressive tumor is unknown. Radical surgical excision with close follow-up is considered standard of care [62].

10.5 Traumatic

Any cause of trauma to the genitalia can have devastating sequelae. Self-induced lacerations follow accidental or factitious trauma. Penile tourniquet syndrome usually occurs in boys when a hair encircles the penis, causing pain, swelling, and erythema of the glans. If untreated, urethral fistula and amputation of the glans can result [90].

Factitial dermatitis (dermatitis artefacta) is a self-inflicted, often mutilating disease characterized by cuts, excoriations, and sharply demarcated ulcers created by self-induced trauma. Associated guilt, embarrassment, or psychological disturbance are common and may make diagnosis and treatment difficult.

10.5.1 Idiopathic Calcinosis of the Scrotum

Persistent, asymptomatic, pale to yellow-white papules of the scrotum represent idiopathic calcification. Most patients are boys or young men with normal serum calcium, phosphate, and uric acid levels. The papules are usually asymptomatic, but may discharge a chalky white material or may increase in number to cause scrotal deformity. If warranted, excision is usually curative [91, 92].

10.6 Summary

Skin disease of the male genitalia has a wide variety of presentation and etiologies, from self-inflicted trauma to malignancy. Inflammatory skin disease may be a primary process, extension of a generalized disorder, or a manifestation of systemic disease. Diagnosis and treatment may be hindered by patient embarrassment or guilt, but a thorough history and physical examination coupled with interdisciplinary management are sufficient to manage any condition.

References

1. Gendernalik S, Galeckas K. Fixed drug eruptions: a case report and review of the literature. Cutis. 2009;84(4):215–9.
2. Maatouk I, Moutran R, Fahed M, Helou J. A "sexually transmitted" fixed drug reaction. Sex Transm Dis. 2014;41:10.
3. Flowers H, Brodell R, Brents M, Wyatt J. Fixed drug eruptions: presentation, diagnosis, and management. South Med J. 2014;107(11):724–7.
4. Patell R, Dosi R, Shah P, Joshi H. Widespread bullous fixed drug eruption. BMJ Case Rep. 2014. pii: bcr2013200584.
5. Blyumin M, Rouhani P, Avashia N, Jacob S. Acquiring allergen information from condom manufacturers: a questionnaire survey. Dermatitis. 2009;20(3):161–70.
6. Lytle C, Carney P, Vohra S, Cyr W, Bockstahler L. Virus leakage through natural membrane condoms. Sex Transm Dis. 1990;17(2):58–62.
7. Muratore L, Calogiuri G, Foti C, Nettis E, Leo ED, Vacca A. Contact allergy to benzocaine in a condom. Contact Dermatitis. 2008;59(3):173–4.
8. Bhate K, Landeck L, Gonzalez E, Neumann K, Schalock P. Genital contact dermatitis: a retrospective analysis. Dermatitis. 2010;21(6):317–20.
9. Uter W, Geier J, Bauer A, Schnuch A. Risk factors associated with methylisothiazolinone contact sensitization. Contact Dermatitis. 2013;69(4):231–8.
10. Bauer A, Oeheme S, Geier J. Contact sensitization in the anal and genital area. Curr Probl Dermatol. 2011;40:133–41.
11. Fisher A, Brancaccio R. Allergic contact sensitivity to propylene glycol in a lubricant jelly. Arch Dermatol. 1979;115(12):1451.
12. Jones S. Anaphylaxis from rectal lubricant jelly. Am J Med. 1988;85(6):890.
13. Smith G, Sharma V, Knapp J, Shields B. The summer penile syndrome: seasonal acute hypersensitivity reaction caused by chigger bites on the penis. Pediatr Emerg Care. 1988;14(2):116–8.
14. Keller E, Tomecki K. Genitourinary dermatology. In: Potts JM, editor. Essential urology: a guide to clinical practice. Current Clinical Urology. New York, NY: Humana Press; 2012.
15. Birley H, Walker M, Luzzi G, Bell R, Taylor-Robinson D, Byrne M, et al. Clinical features and management of recurrent balanitis; association with atopy and genital washing. Genitourin Med. 1993;69(5):400–3.
16. Kavanagh G, Burton P, Kennedy C. Vulvitis chronica plasmacellularic (Zoon's vulvitis). Br J Dermatol. 1993;129:92–3.
17. Pastar Z, Rados J, Lipozencic J, Skerlev M, Loncaric D. Zoon plasma cell balanitis: an overview and role of histopathology. Acta Dermatovenereol Croat. 2004;12(4):268–73.
18. Retamar R, Kien M, Chouela E. Zoon's balanitis: presentation of 15 patients, five treated with a carbon dioxide laser. Int J Dermatol. 2003;42(4):305–7.
19. Fernandez-Acenero M, Cordova S. Zoon's vulvitis (vulvitis circumscripta plasmacellularis). Arch Gynecol Obstet. 2010;282:351–2.
20. Albers SE, Taylor G, Huyer D, Oliver G, Krafchik B. Vulvitis circumscripta plasmacellularis mimicking child abuse. J Am Acad Dermatol. 2000;42(6):1078–80.
21. Balato N, Scalvenzi M, Bella SL, Costanzo LD. Zoon's balanitis: benign or premalignant lesion? Case Rep Dermatol. 2009;1(1):7–10.
22. Virgili A, Mantovani L, Lauriola M, Marzola A, Corazza M. Tacrolimus 0.1% ointment: is it really effective in plasma cell vulvitis? Report of four cases. Dermatology. 2008;216:243–6.
23. Speeckaert R. Geel Nv. Distribution patterns in generalized vitiligo. J Eur Acad Dermatol Venereol. 2014;28(6):755–62.
24. Broding H, Monse C, Bruning T, Fartasch M. Induction of occupational leucoderma and vitiligo. Can butylated hydroxytoluene induce vitiligo similarly to p-tert-butylphenol? Hautarzt. 2011;62(3):209–14.
25. Zhang R, Zhu W. Genital vitiligo following use of imiquimod 5% cream. Indian J Dermatol. 2011;56(3):335–6.

26. Silverberg N. Update on childhood vitiligo. Curr Opin Pediatr. 2010;22(4):445–52.
27. Tamesis M, Morelli J. Vitiligo treatment in childhood: a state of the art review. Pediatr Dermatol. 2010;27(5):437–45.
28. Mehraban S, Feily A. 308nm excimer laser in dermatology. J Lasers Med Sci. 2014;5(1): 8–12.
29. Cohen A, Andres I, Medvedovsky E, Peleg R, Vardy D. Similarities between neuropathic pruritus sites and lichen simplex chronicus sites. Isr Med Assoc J. 2014;16(2):88–90.
30. Rajalakshmi R, Thappa D, Jaisankar T, Nath A. Lichen simplex chronicus of anogenital region: a clinico-etiological study. Indian J Dermatol Venereol Leprol. 2011;77(1):28–36.
31. Weichert G. An approach to the treatment of anogenital pruritus. Dermatol Ther. 2004;17(1): 129–33.
32. Aschoff R, Wozel G. Topical tacrolimus for the treatment of lichen simplex chronicus. J Dermatolog Treat. 2007;18(2):115–7.
33. Heckmann M, Heyer G, Brunner B, Plewig G. Botulinum toxin type A injection in the treatment of lichen simplex: an open pilot study. J Am Acad Dermatol. 2002;46(4):617–9.
34. Romero-Talamas H, Aminian A, Corcelles R, Fernandez A, Berthauer PSS. Psoriasis improvement after bariatric surgery. Surg Obes Relat Dis. 2014;10(6):1155–9.
35. Meeuwis K, de Hullu J, Massuger L, van de Kerkhof P, van Rossum M. Genital psoriasis: a systematic literature review on this hidden skin disease. Acta Derm Venereol. 2011;91(1): 5–11.
36. Kohn F, Pflieger-Bruss S, Schill W. Penile skin diseases. Andrologia. 1993;31 Suppl 1:3–11.
37. Meeuwis K, de Hullu K, van de Nieuwenhof H, Evers A, Massuger L, van de Kerkhof P, et al. Quality of life and sexual health in patients with genital psoriasis. Br J Dermatol. 2011;164(6): 1247–55.
38. Schmidt H. Frequency, duration, and localization of lichen planus. A study based on 181 patients. Acta Derm Venereol. 1961;41:164–7.
39. Matoso A, Ross H, Chen S, Allbritton J, Epstein J. Squamous neoplasia of the scrotum: a series of 29 cases. Am J Surg Pathol. 2014;38(7):973–81.
40. Lundqvist E, Wahlin Y, Bergdahl M, Bergdahl J. Psychological health in patients with genital and oral erosive lichen planus. J Eur Acad Dermatol Venereol. 2006;20(6):661–6.
41. Mirowski G, Goddard A. Treatment of vulvovaginal lichen planus. Dermatol Clin. 2010;28:717–25.
42. Lonsdale-Eccles A, Velangi S. Topical pimecrolimus in the treatment of genital lichen planus: a prospective case series. Br J Dermatol. 2005;153:390–4.
43. Fox B, Odom R. Papulosquamous diseases: a review. J Am Acad Dermatol. 1985;12(4): 597–624.
44. Kim Y, Shim S. Two cases of generalized lichen nitidus treated successfully with narrow-band UV-B phototherapy. Int J Dermatol. 2006;45:615–7.
45. Neill S, Lewis F, Tatnall F, Cox N. British association of dermatologists' guidelines for the management of lichen sclerosus. Br J Dermatol. 2010;163:672–82.
46. Das S, Tunuguntla H. Balanitis xerotica obliterans – a review. World J Urol. 2000;18(6): 382–7.
47. Aynaud O, Plantier F. Genital lichen sclerosus treated by carbon dioxide laser. Eur J Dermatol. 2010;20(3):387–8.
48. Neuhaus I, Skidmore R. Balanitis xerotica obliterans and its differential diagnosis. J Am Board Fam Pract. 1999;12(6):473–6.
49. Koutsky L. Epidemiology of genital human papillomavirus infection. Am J Med. 1997; 102(5Z):3–8.
50. Roberts C, Pfister J, Spear S. Increasing proportion of herpes simplex virus type 1 as cause of genital herpes infection in college students. Sex Transm Dis. 2003;30(10):797–800.
51. Leone P. Reducing the risk of transmitting genital herpes: advances in understanding and therapy. Curr Med Res Opin. 2005;21(10):1577–82.
52. Lebrun-Vignes B, Bouzamondo A, Dupuy A, Guillaume J, Lechat P, Chosidow O. A meta-analysis to assess the efficacy of oral antiviral treatment to prevent genital herpes outbreaks. J Am Acad Dermatol. 2007;57(2):238–46.

53. Patton M, Su J, Nelson R, Weinstock H. Primary and secondary syphilis--United States, 2005-2013. MMWR Morb Moral Wkly Rep. 2014;63(18):402–6.
54. Rodin P, Kolator B. Carriage of yeasts on the penis. Br Med J. 1976;1(6018):1123–4.
55. Lisboa C, Costa A, Ricardo E, Santos A, Azevedo F, Pina-Vaz C, et al. Genital candidosis in heterosexual couples. J Eur Acad Dermatol Venereol. 2011;2011(25):145–51.
56. Abdullah A, Drake S, Wade A, Walzman M. Balanitis (balanoposthitis) in patients attending a department of genitourinary medicine. Int J STD AIDS. 1992;3(2):128–9.
57. Dockerty W, Sonnex C. Candidal balano-posthitis: a study of diagnostic methods. Genitourin Med. 1995;71(6):407–9.
58. Mayser P. Mycotic infections of the penis. Andrologia. 1993;31 Suppl 2:13–6.
59. Stary A, Soeltz-Szoets J, Ziegler C, Kinghorn G, Roy R. Comparison of the efficacy and safety of oral fluconazole and topical clotrimazole in patients with candida balanitis. Genitourin Med. 1996;72(2):98–102.
60. Farmakiotis D, Tarrand J, Kontoyiannis D. Drug-resistant Candida glabrata infection in cancer patients. Emerg Infect Dis. 2014;20(11):1833–40.
61. Suyama T, Teramoto Y, Yamamoto A, Kuroda I, Yoshikawa S, Kiyohara Y. Case of giant basal cell carcinoma around the base of the penis in an elderly patient. J Dermatol. 2014;41(5): 439–42.
62. Sandhu R, Min Z, Bhanot N. A gigantic anogenital lesion: Buschke-Lowenstein tumor. Case Rep Dermatol Med. 2014;2014:650714.
63. Agarwal S, Bhattacharya S, Singh N. Pearly penile papules: a review. Int J Dermatol. 2004;43(3):199–201.
64. Taniguchi S, Inoue A, Hamada T. Angiokeratoma of Fordyce: a cause of scrotal bleeding. Br J Urol. 1994;73(5):589–90.
65. Odezmir M, Baysal I, Engin B, Odezmir S. Treatment of angiokeratoma of Fordyce with long-pulse neodymium-doped yttrium aluminum garnet laser. Dermatol Surg. 2009;35:92–7.
66. Graham J, Helwig E. Erythroplasia of Queyrat. A clinicopathologic and histochemical study. Cancer. 1973;32(6):1396–414.
67. Cupp M, Malek R, Goellner J, Smith T, Espy M. The detection of human papillomavirus deoxyribonucleic acid in intraepithelial, in situ, verrucous and invasive carcinoma of the penis. J Urol. 1995;154(3):1024–9.
68. Sarkar F, Miles B, Plieth D, Crissman J. Detection of human papillomavirus in squamous neoplasm of the penis. J Urol. 1992;147(2):389–92.
69. Burgers J, Badalament R, Drago J. Penile cancer: clinical presentation, diagnosis, and staging. Urol Clin North Am. 1992;19(2):247–56.
70. Narayana A, Olney L, Loening S, Weimar G. Carcinoma of the penis: analysis of 219 cases. Cancer. 1982;49(10):2185–91.
71. English H, Laws R, Keogh G, Wilde J, Foley J, Elston D. Dermatoses of the glans penis and prepuce. J Am Acad Dermatol. 1997;37(1):1–24.
72. Campus G, Alia F, Bosincu L. Squamous cell carcinoma and lichen sclerosus et atrophicus of the prepuce. Plast Reconstr Surg. 1992;89(5):962–4.
73. Johnson D, Fuerst D, Ayala A. Carcinoma of the penis. Experience with 153 cases. Urology. 1973;1(5):404–8.
74. Lowe F. Squamous cell carcinoma of the scrotum. J Urol. 1983;130(3):423–7.
75. Lai K, Mercurio M. Medical and surgical approaches to vulvar intraepithelial neoplasia. Dermatol Ther. 2010;23:477–84.
76. An J, Li B, Wu L, Lu H, Li N. Primary malignant amelanotic melanoma of the female genital tract: report of two cases and review of literature. Melanoma Res. 2009;19(4):267–70.
77. Piura B. Management of primary melanoma of the female urogenital tract. Lancet Oncol. 2008;9:973–81.
78. Schellhammer P, Jordan G, Robey E, Spaulding J. Premalignant lesions and nonsquamous malignancy of the penis and carcinoma of the scrotum. Urol Clin North Am. 1992;19(1): 131–42.

79. Rashid A, Williams R, Horton L. Malignant melanoma of penis and male urethra: is it a difficult tumor to diagnose? Urology. 1993;41(5):470–1.
80. Zurrida S, Bartoli C, Clemente C, Palo GD. Malignant melanoma of the penis. A report of four cases. Tumori. 1990;76(6):599–602.
81. Manviel J, Fraley E. Malignant melanoma of the penis and male urethra: 4 case reports and literature review. J Urol. 1988;139(4):813–6.
82. Johnson D, Ayala A. Primary melanoma of penis. Urology. 1973;2(2):174–7.
83. Lee S, Choel Y, Jung H, et al. A multicenter study on extramammary Paget's disease in Korea. Int J Dermatol. 2011;50:508–15.
84. Smith D, Handy F, Evans J, Falzon M, Chapple C. Paget's disease of the glans penis: an unusual urological malignancy. Eur Urol. 1994;25(4):316–9.
85. Lam C, Funaro D. Extramammary Paget's disease: summary of current knowledge. Dermatol Clin. 2010;28:807–26.
86. Lowe E, Schmitt A, Bordeaux J. Management of unusual cutaneous malignancies: atypical malignant fibroxanthoma, fibrous histiocytoma, sebaceous carcinoma, extramammary Paget disease. Dematol Clin. 2011;29:201–16.
87. Hegarty P, Suh J, Fisher M. Penoscrotal extramammary Paget's disease: The University of Texas M. D. Anderson Cancer Center Contemporary Experience. J Urol. 2011;186:97–102.
88. Kitagawa K, Bogner P, Zeitouni N. Photodynamic therapy with methyl-aminolevulinate for the treatment of double extramammary Paget's disease. Dermatol Surg. 2011;37:1043–6.
89. Martin J, Molina I, Monteagudo C, Marti N, Lopez V, Jorda E. Buschke-Lowenstein tumor. J Dermatol Case Rep. 2008;2(4):60–2.
90. Sallami S, Rhouma SB, Cherif K, Noura Y. Hair-thread tourniquet syndrome in an adult penis: case report and review of the literature. Urol J. 2013;10(2):915–8.
91. Pak K, Takayama H, Tomoyoshi T. Idiopathic calcinosis of scrotum. Urology. 1983;21(5): 521–3.
92. Yahya H, Rafindadi A. Idiopathic scrotal calcinosis: a report of four cases and review of the literature. Int J Dermatol. 2005;44(3):206–9.
93. Molina-Leyva A, Almodovar-Real A, Ruiz-Carrascosa J, Naranjo-Sintes R, Serrano-Ortega S, Jimenez-Moleon J. Distribution pattern of psoriasis affects sexual function in moderate to severe psoriasis: a prospective case series study. J Sex Med. 2014;11(12):2882–9.