

Skin as a Window to Sexually Transmitted Disease

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Sexually transmitted diseases (SD) usually present with lesions affecting the external genitalia. The common presentations of sexually transmitted infections (STI) are genital ulcer disease, urethral discharge, genital growth or a swelling in the groin or the scrotum. Women can present with vaginal discharge and lower abdominal or lumbar pain. However, the extragenital skin may also be affected directly or indirectly. Therefore, a detailed cutaneous examination may give valuable clues to the diagnosis, as well as of high-risk behaviour. The cutaneous manifestations of STIs may be classified as : (i) Extragenital lesions of STIs. (ii) Surrogate markers of sexually acquired infection. (iii) Surrogate markers of high-risk behaviour and human immunodeficiency virus (HIV) infection.

Extragenital lesions of STIs :

Nearly all the bacterial and viral pathogens that cause STIs can have extragenital manifestations. The important presentations of each will be dealt with briefly -

Syphilis^{1,2} : The natural history of syphilis can be divided into primary, secondary and tertiary stages. Each stage can involve the extragenital skin and a high index of suspicion is required to arrive at the diagnosis.

Primary syphilis¹ : After an incubation period of 9-90 days, the primary lesion or the chancre develops at the site of inoculation of *Treponema pallidum*. The extragenital sites of occurrence of chancre are the lips, buccal cavity, tongue, tonsil, pharynx and rarely, the finger. The typical chancre is a single, painless, well-defined, indurated ulcer with a smooth, flat, dull-red surface which may be covered with a thin yellow or brown crust. On pressure, serous fluid may ooze. Within a few days, there is discrete and rubbery regional lymph node enlargement. The primary lesion heals within 3-8 weeks leaving a thin, atrophic scar, even without treatment.

Secondary syphilis³ : Skin lesion (syphilides) are seen in over 80% of patients with secondary syphilis. Mucocutaneous lesions are very infectious. The skin eruptions are polymorphic and several types may appear simultaneously during the course. Although secondary syphilides are considered to be asymptomatic, over 40% of patients complain of pruritus². There are various clinical subtypes-

Macular syphilide (roseola) : Rose-pink, faint, about 1cm evanescent macules are the earliest manifestations. However, because of their faint colour, indistinct margins and the dark skin color of our race, they are easily overlooked. The lesions last for a few days only.

Papular and papulosquamous syphilide : These are commonest lesions appearing as firm, symmetrical, dull-red lesions, variable

in size, present especially over the flexor aspects of the body. Occasionally, a larger papule may be surrounded by smaller lesions. This is called the 'corymbose' syphilide. As the lesion ages, scaling may appear. The sites affected may be -

Face : The chin, nasolabial folds and the margins of the scalp may be affected. The lesions on the forehead parallel to the hairline are called the 'corona veneris'.

Scalp : Scalp involvement presents as alopecia. This may be the classic 'moth-eaten' type, or the nonspecific reactional 'telogen effluvium'.

Palms and soles : These sites show the classic 'copper macules'. The papular lesions on these sites do not project above the surface of the skin, probably because of its thickness. They appear as firm, dull-red lesions with a collarette of scale.

Condylomata lata - Hypertrophied, moist, flat-topped plaques with the surface often eroded are the classical lesions seen in moist and angulated areas like the groin, vulva and perianal region. However, the axillae, nasolabial folds, and the angles of the mouth may be similarly affected (split papules). The exudate in such lesions is teeming with spirochaetes.

Pustular syphilide⁴ : Rarely, necrosis of the upper dermis and epidermis, as a result of the endarteritis cause pustulation. This variant is seen in immunocompromised patients and heals with scarring.

Malignant or ulcerative syphilide⁵ : This severe form of syphilide presents with well-defined ulcerations. The patient presents with fever, malaise, joint pains and a papulo-pustular eruption that soon becomes necrotic, resulting in the sharply marginated ulcers with a thick, rupioid crust. This is also called 'lues maligna'.

Mucosal lesions : These are found in about 30% of patients. The characteristic lesion is the 'mucous patch'. These are round or oval, grey areas surrounded by a narrow zone of erythema. Shedding of the grey, necrotic membrane reveals superficial ulceration. If many such lesions coalesce, a 'snail-track ulcer' may result.

Pigmentary changes : the syphilides heal without any residual inflammatory signs. However, areas of mild pigmentation may persist for months. In dark-haired women, depigmentation of the neck that lasts for life can be observed. This is called 'leukoderma colli'.

Tertiary Syphilis^{1,2} : This stage is characterized by 'gumma' formation. Gummata are superficial or deep destructive granulomatous lesions which can involve the skin, the subcutaneous tissue and bone. They tend to occur on the face, neck and distal extremities at the sites of trauma. They begin as asymptomatic cutaneous or subcutaneous nodules that ulcerate and coalesce to form large irregular plaques with 'punched-out' edges and arcuate, irregular borders. The floor of the ulcer shows the typical 'wash-leather' appearance. Healing occurs with atrophy leaving behind hyperpigmented scars.

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Diagnosis : This is made by demonstration of the treponemes by dark-field microscopy, and more commonly by serologic tests, namely the VDRL and the TPHA.

Treatment : Penicillin is the treatment of choice. The dosage differs according to the stage of the disease.

Chancroid^{6,7} : This is caused by *Haemophilus ducreyi*. The incubation period varies from 24 hours to 5 days. The lesion begins as a papule, which pustulates and ulcerates to form a painful, tender, shallow ulcer with ragged, undermined edges. It bleeds readily on touch. Auto-inoculation is common and multiple lesions may be present in a small area. Painful and tender local lymph nodes that may form a fluctuant 'bubo' are characteristic. Extragenital disease is rare, as is dissemination. Diagnosis is confirmed by microscopy, which shows Gram-negative bacilli with a typical 'rail-road' or 'school of fish' arrangement. Azithromycin, ceftriaxone and ciprofloxacin are the drugs of choice.

Donovanosis^{6,8} : This is caused by *Calymatobacterium granulomatis*. The incubation period is unknown. It may vary from 3 days to 6 months. Extragenital disease has been described, usually secondary to chronic genital disease. The earliest lesion is variable. It may begin as a papule, a pruritic nodule or most commonly, an ulcer. The ulcer is painless, of variable size, soft, velvety, with bright red granulation tissue and a serpiginous edge. It bleeds readily when abraded. The disease is slowly progressive and the mean duration is 18 months. Fibrosis is seen in chronic cases, especially in females. The diagnosis is confirmed by demonstration of the bipolar staining "closed safety pin" organism in tissue smears by Giemsa, Leishman or Wright's stain. Doxycycline, azithromycin, fluoroquinolones, erythromycin, cotrimoxazole or ceftriaxone are recommended for treatment.

Lymphogranuloma venereum (LGV)⁹ : This is caused by *Chlamydia trachomatis*, serovars L1, L2 and L3. The incubation period is 3-12 days and the primary lesion is an evanescent herpetiform papule that is self-limiting. This is followed by the 'inguinal syndrome' and the 'genito-ano-rectal syndrome'. Regional lymphadenopathy, painful, tender and matted due to periadenitis leading to the formation of fluctuant 'pseudobuboes', is seen after 1-6 weeks. Untreated the disease lasts for 6-8 weeks and may resolve completely. Approximately 70% affected individuals show lymphatic obstruction and intermittent recurrences. Extragenital disease is rare, although oral lesions are known to occur. There are a number of uncommon associated skin lesions. Erythema nodosum (EN) is seen in 10% of women and 2% of men affected by the disease. Erythema multiforme (EM) has also been described. Rarely, a photosensitive eruption has also been described in affected individuals. Diagnosis is confirmed by demonstration of the organism in culture on cycloheximide-treated McCoy cell lines, and by serologic tests such as the complement fixation test (LGVCF) and the micro-immunofluorescence test (MIF). Doxycycline, erythromycin and aminoglycosides form the mainstay of treatment.

Gonorrhoea^{10,11} : This is caused by the Gram-negative intracellular diplococcus (GNID), *Neisseria gonorrhoeae*. The incubation period varies from 24-72 hours, and the commonest presentation is a painful urethral or genital discharge. Dissemination is an uncommon complication occurring in less than 1% cases, especially in women with asymptomatic anogenital infection. The clinical manifestations are fever, rash and arthralgia/arthritis. Skin involvement is seen in 90% patients. The lesions begin as erythematous macules and may take two forms¹¹-hemorrhagic

infarcts, or vesiculopapular lesions on an erythematous base. The lesions appear in crops, and are asymmetrically distributed mainly on the extremities and around the affected joints. The organism may be detected by Gram stain or direct immunofluorescence from the skin lesion. Blood culture is usually negative, but synovial fluid gives positive results in 50%. EM and EN have also been described in these patients. Treatment consists of intravenous cephalosporins, quinolones or spectinomycin in the initial stage, after which oral agents may be continued.

Herpes simplex virus (HSV) infection¹²: This is caused by the herpes simplex viruses, 1 and 2 (HSV-1 & -2). The incubation period varies from 3-9 days. HSV-1 usually causes orolabial disease, while HSV-2 can be sexually transmitted and manifests as a genital ulcer disease (GUD). However, orogenital intercourse may cause primary HSV-2 pharyngitis. The virus has been isolated from the pharynx in 11% individuals with primary HSV-2 genital herpes. Primary herpetic pharyngitis presents as a painful sore throat with mild erythema of the pharynx, small vesicles or erosions with red areolae, or diffuse ulceration covered with a white exudate. Dissemination is rare and seen in immunocompromised hosts. The skin lesions are either well-defined vesicles or pustules. Diagnosis is confirmed by Giemsa or Wright-stained Tzanck smear which shows multinucleate giant cells, and by culture on human fibroblast cell lines. Nowadays, ELISA and PCR have simplified the diagnosis. Treatment consists of acyclovir, valaciclovir and famciclovir.

Human papilloma virus (HPV) infection¹³: This manifests as warts, which are cutaneous neoplasms caused by the human papilloma virus. The incubation period may last from weeks to years. There are certain HPV types that are sexually transmitted and cause genital warts; the common being types 6, 11, 16, 18 and 33. Extragenital lesions due to these types are rare. However, orogenital intercourse may result in oral papillomas, which usually affect the soft palate and the frenulum, dorsum or the lateral borders of the tongue, and rarely the lips. The lesion is usually solitary, pink, sessile or pedunculated verrucous swelling. Biopsy is diagnostic and treatment consists of surgery, electrical or chemical.

Scabies¹⁴ : *Sarcoptes scabiei* var *hominis* is the arthropod responsible for this extremely pruritic, primarily cutaneous eruption. The mite can be transmitted from an infested person to another by prolonged physical and sexual contact, or by sharing clothes or bed linen. The incubation period i.e. the time to onset of symptoms is about 4-5 weeks. The patient presents with an intensely itchy eruption affecting classically the web spaces, the wrist, the axillary folds, the waist and the genitalia. Sexually acquired scabies presents with excoriated papules or nodules on the shaft of the penis and the scrotum in males, and on the labia in females. The classical lesion of scabies is the 'burrow', from which the mite can be isolated by scraping the overlying skin. In immunocompromised patients, a severe variant of scabies with a mite load of millions is seen. It is called 'crusted' or 'Norwegian' scabies. Treatment consists of topical miticides such as 25% benzyl benzoate, 1% gamma benzene hexachloride, 0.5% malathion, 6-10% sulfur or 5% permethrin cream. A contact period of 12-36 hours is required depending on the agent used and a repeat application is required after 2 weeks, except if permethrin has been used. Oral ivermectin, as a single dose of 200 microgram/kg, is also recommended.

Phthiriasis^{15,16} : This condition is caused by infestation by the crab louse, *Phthirus pubis*. The arthropod inhabits the pubic, perianal or the axillary region by attachment to the hair by its

claws. Rarely, the eyelashes, the eyebrows and the scalp margins may also be affected. Intense pruritus is the only symptom. Blue spots, called 'maculae coeruleae' produced as a result of an enzyme secreted by the louse during its bite, are the characteristic features. Topical malathion (0.5%), 1% lindane are 1% permethrin creme rinse are the treatments of choice.

Surrogate markers of sexually acquired infection :

Apart from the viruses mentioned above, Hepatitis B Virus (HBV) and Hepatitis C virus (HCV) can also be acquired as a result of sexual transmission. These viruses do not affect the skin directly. There are a number of conditions associated with both infections that have well-defined cutaneous manifestations. Thus, these conditions can be regarded as surrogate markers of STDs.

Hepatitis B virus : HBV infection in humans may be associated with the following conditions :

i. *Polyarteritis nodosa (PAN)*¹⁷ - This is a systemic vasculitis characterized by necrotizing inflammatory lesions and involvement of the skin, the kidneys, the joints and musculoskeletal system, the gut and peripheral nerves. HBV-associated PAN shows features of immune-complex induced disease. Cutaneous involvement is observed in 25-60% of cases. The spectrum of lesions includes palpable purpura, infarctions, ulcerations, livedo reticularis, and ischemic changes of the distal digits. Subcutaneous nodules are infrequent. Limited cutaneous forms of PAN, sometimes associated with myalgias, arthralgias, and peripheral neuropathy, may occur. Only 1% or less of the total population of patients who are HBV positive develop PAN. Clinical symptoms of non-HBV-related and HBV-related PAN are the same except for orchitis, which appears to be more frequent in groups with HBsAg⁺. Diagnosis is confirmed by histopathology which reveals necrotizing arteritis involving arterioles, venules, capillaries, small- and medium-sized arteries; and by evidence of p-ANCA in serum. Presence of hepatitis B surface antigen is noted in 7-36%. Corticosteroids and cytotoxic drugs form the mainstay of treatment. In addition, ribavirin and interferon alpha-2b is advocated to enhance clearance of virions.

ii. *Porphyria cutanea tarda (PCT)*¹⁸: Porphyria cutanea tarda is an autosomal dominant disorder caused by the deficiency of the hepatic enzyme Uroporphyrinogen decarboxylase (UROD) and characterized by onset of light-sensitive dermatitis in later adult life, associated with the excretion of large amounts of uroporphyrin in urine. It was so named by Waldenstrom (1937). On areas of skin exposed to sunlight, especially the face, ears and backs of the hands, chronic ulcerating lesions commence as blisters, and the skin may also be mechanically fragile (Grossman et al., 1979). HBV/HCV and HIV infection may precipitate this condition as a consequence of the hepatic inflammation, which further reduces the enzyme levels. Hyperpigmentation and hypertrichosis also occur. Diagnosis is confirmed by examination of urine for uroporphyrins, and skin biopsy which reveals a subepidermal blister with minimal inflammation. Treatment of the viral infection, photoprotection, chloroquine and phlebotomy form the mainstays of treatment.

iii. *Gianotti-Crosti syndrome (Papular Acrodermatitis of Childhood)*¹⁹: Gianotti-crosti syndrome is a benign, self-limited childhood exanthem that occurs in a characteristic distribution. It is rarely associated with systemic findings. The original cases,

described in Italy by Gianotti in 1955, were associated with hepatitis B virus infection. Adult cases are rare. A prodromal upper respiratory infection is reported in 31% of patients. Pruritus accompanies the eruption in 23% of patients. Patients may also have lymphadenopathy and mild constitutional symptoms, such as low grade fever and malaise. The cutaneous eruption is characterized by pale, pink-to-flesh-colored papules localized symmetrically over the extremities, the buttocks, and the face. Over days to weeks, the papules may acquire a smooth-topped, polished, or lichenoid appearance. The eruption lasts longer than 6 weeks in more than 50% of patients, and complete resolution typically takes more than 2 months. In cases caused by hepatitis B virus, anicteric hepatitis is evident by elevations in the levels of hepatic transaminases and antiviral antibodies. Diagnosis and reassurance are usually sufficient. Soothing, anti-itch topical preparations with pramoxine or oral antihistamines may be useful for relief of pruritus.

iv. *Miscellaneous findings* : These include palmar erythema, spider nevi, thin (paper-money) skin and 'caput medusae' on the abdomen.

Hepatitis C virus (HCV)²⁰: Cutaneous symptoms or findings relevant to HCV infection manifest in 20-40% of patients presenting to dermatologists and in a significant percentage (15-20%) of general patients. HCV is suggested and must appear in the differential diagnosis of these patients to avoid missing this important but occult factor in clinical disease in the appropriate setting. Primary causation results from direct infection of HCV in the skin, lymphocytes, dendritic antigen-presenting cells, and blood vessels. An example of this type of disorder is the recent finding of epidermal cells with HCV-RNA particles. Secondary causation occurs when HCV infection manifests in the skin due to epiphenomena resulting from the disruption of immune responses. Leukocytoclastic vasculitis due to cryoglobulinemia is a good example. Tertiary causation of dermatologic manifestations results when the disruption of another organ infected or affected by HCV causes skin manifestations that are nonspecific and typical of skin responses to that organ. Thus the various cutaneous manifestations of HCV infection may be classified as :

Primary manifestation:

These include : a) *Lichen planus* : Intracellular HCV infection of epithelial cells is proven for LP²¹. Lesions are similar to those seen in uninfected individuals. Often, the popular lesions of LP suddenly appear on the volar acral surfaces of the wrists and arms and are pruritic. Oral symptoms are less common. Hair loss in lichen planopilaris, exquisite pruritus of markedly hypertrophic plaques on the lower legs in hypertrophic LP, and painful genital erosions and be presenting findings.

b) *Acral necrolytic erythema*²²: The symptomatology of acral necrolytic erythema includes pruritus associated with recurrent, erythematous, papular eruptions with blisters and erosions on the dorsal aspects of the feet and ankles. Pain is common with variable-sized erosions. Chronic lesions are hyperkeratotic plaques with erosions and peripheral erythema preferring the acral parts of the legs. These lesions provide unusually specific markers for HCV infection.

c) *Leukocytoclastic reactions* (some): This tends to appear as an eruption of palpable purpura on the lower extremities. It may represent an HCV immune complex disease.

Secondary manifestations :

a) *Cryoglobulinemia* : Leukocytoclastic vasculitis occurs with type II mixed cryoglobulinemia in the skin and mucous membranes. These disorders display palpable purpura of the legs (which is worse distally and inferiorly), livedo reticularis, ulcerations, urticaria, symmetric polyarthritis, myalgias, cutis marmorata, and fatigue.

b) *Sialadenitis*: Dry mouth without dry eyes is the most prominent symptom of sialadenitis associated with HCV infection. Sialadenitis is an inflammatory disorder of the salivary, parotid, sublingual, and minor glands. Findings include xerostomia resulting from a chronic lymphocytic infiltrate and destruction of the salivary glands. Sjogren disease and its markers ssRo and ssLa are not found.

c) *Antiphospholipid syndrome* is a serious multisystemic illness resulting from pathologic production of the antiphospholipids anticardiolipin and lupus anticoagulant. Severe coagulopathies in the eye, the brain, the kidney, and large vessels in symptomatology refrable to vascular destruction or bleeding in these organs.

Tertiary manifestations :

Symptoms are those of disease in the specific organs, as follows-

a) *Liver failure in CHC infection* results from cirrhosis, autoimmune hepatitis, cholangitis, and HCC. Symptoms of liver failure are identical to symptoms caused by other conditions, such as ascites, jaundice and liver failure.

b) *Thyroid failure* : Thyroid destruction leading to failure occurs; symptoms of hypothyroidism are noted.

c) *Miscellaneous conditions* : These include Behcet syndrome, canities, prurigo nodularis, polyarteritis nodosa, pruritus, erythema nodosum (EN), erythema multiforme (EM), porphyria cutanea tarda (PCT), erythema dyschromicum perstans, disseminated superficial porokeratosis, generalized granuloma annulare and progressive pigmented purpura (Gougerot-Blum disease).

Surrogate markers of high-risk behaviour and human immunodeficiency virus (HIV) infection :

Certain skin conditions are consistently associated with HIV infection and thereby, high-risk behaviour. These include -

1. *Molluscum contagiosum (MC)*²³: In HIV infection, MC may be widespread and atypical. The lesions may be observed on extragenital sites, such as the face, the neck, and the scalp; or they may show altered morphology and size. Such unusual forms include solitary endophytic, aggregated, inflamed, and giant MCs. MCs mimicking sebaceous nevus of Jadassohn, ecthyma, and giant condylomata acuminata have been reported. Imiquimoid is curative. Topical aciclovir, ablation and curettage may be useful.

2. *Candidiasis*²⁴: Recurrent and persistent mucocutaneous candidiasis is common in HIV-infected patients. Clinically, it manifests as whitish, curd-like exudates on the dorsal or buccal mucosa than can be easily scraped away with a cotton swab, leaving behind a reddish friable surface that may be associated with a burning sensation - the so-called pseudomembranous candidiasis or thrush. Sometimes, only a beefy red, eroded surface can be seen (erosive candidiasis). Chronic atrophic candidiasis, presenting as angular cheilitis and candidial leukoplakia are also noted. The symptoms include burning pain, altered taste sensation and dysphagia, which is more prominent with oesophageal candidiasis.

3. *Oral hairy leukoplakia (OHL)*²⁵: Epstein-Barr (EBV) has been implicated in the pathogenesis of oral hairy leukoplakia (OHL). OHL is characterized by white, filiform, corrugated and feathery plaques on the sides of the tongue and sometimes on the oropharyngeal mucosa, which may be mistaken for candidiasis. It has no malignant potential, but it may be the initial sign of progressive immunosuppression. Treatment is usually not necessary. If symptomatic, the patient may be prescribed systemic aciclovir (3200 mg/d), topical application of 25% podophyllum resin, ganciclovir, or foscarnet.

4. *Varicella zoster virus (VZV) infections*²⁶: Primary varicella infection with visceral dissemination may be seen in HIV-infected adults, but is rare. It may progress to chronic skin involvement. Disseminated and severe varicella infections are observed in advanced AIDS. Atypical manifestations, including hyperkeratotic papules, folliculitis, verrucous lesions, chronic ulcerations, disseminated ecthymatous lesions, and chronic VZV infection mimicking basal cell carcinoma, have also been described. Herpes zoster ophthalmicus may involve the conjunctiva, cornea, anterior chamber, or the retina. Blindness is a complication of zoster retinitis. Recurrent, multidermatomal or disseminated herpes zoster is an AIDS-defining illness. Aciclovir is the treatment of choice for HSV/VZV diseases in these patients. This class of antivirals is activated by viral thymidine kinase. In some disseminated cases, the virus may develop resistance to aciclovir as a result of defective enzyme activity and prolonged/suboptimal dosage. In this scenario, use of other drugs, including cidofovir, foscarnet, and vidarabine, may be necessary.

5. *Kaposi's sarcoma (KS)*²⁷: KS was the first neoplasm reported in HIV disease. It is usually seen in gay and bisexual men, and in women in Africa. The worldwide incidence of KS in patients with AIDS may approach 34%. KS occurs at all stages of HIV disease, and its severity is not strictly correlated with the degree of immunosuppression. KS is believed to be a proliferation of endothelial cells induced by human herpes virus-type 8 (HHV-8), acquired through sexual transmission.

6. *Cytomegalovirus (CMV) infection*²⁴: CMV is also associated with nonspecific cutaneous lesions: generalized bullous toxic epidermal necrolysis-like eruption, purpuric or petechial rash as a result of thrombocytopenia, hyperpigmented indurated cutaneous plaques and bluish-red cutaneous nodules in pediatric patients (blueberry muffin lesions), which indicate extramedullary erythropoiesis.

7. *Seborrheic dermatitis (SD)*²⁸: It may be the initial cutaneous manifestation of HIV disease. The prevalence ranges from 7% to 80%. Its presence correlates inversely with decreasing CD4+T cell counts and thus, the incidence and severity in HIV-infected persons is closely related to the stage of HIV infection. Most cases have an ordinary clinical presentation. However, atypical features such as thick greasy scales on the face and the scalp, and involvement of axillae, groins and perianal areas have been described. It may progress to erythroderma. Histologically, distinct features such as parakeratosis, keratinocyte necrosis, lymphoid clusters at the dermoepidermal junction and a perivascular plasma cell infiltrate have been reported. SD occurs with increased frequency in patients with AIDS-associated dementia. A neurohormonal regulatory dysfunction leads to increased sebum production and consequent overgrowth of the yeast, *Malassezia furfur*.

Treatment is difficult. Application of antifungal/

corticosteroid creams separately or in combination, is the treatment of choice. Treatment with coal tar, sulfur, and salicylic acid shampoos and topical tacrolimus may be effective.

9. Pruritic papular eruption (PPE)²⁹: Various descriptions have been proposed for this entity. The etiology is obscure and no definite cause has been detected. PPE may present with different types of rashes. These include -

- *Transient*, maculopapular eruptions occur most frequently on the face and trunk. They usually heal within 4 to 6 weeks. Histologically, a lymphoplasmacytic angitis is repeatedly observed in many cases.

- A more *chronic eruption* has also been described in individuals with AIDS and ARC. It consists of multiple discrete, 2 to 5mm skin-colored papules distributed over the head, neck and upper trunk. Histology is nonspecific. No correlation has been found between disease severity and stage of HIV infection.

- A chronic, *follicular eruption* on the limbs and trunk, which is characterized histologically by a perifollicular neutrophilic infiltrate.

No pathogen has been detected in any of these conditions. The treatment is also empirical. Topical corticosteroids, phototherapy with PUVA and UVB, dapsone, topical 4% cromolyn sodium and pentoxifylline⁷⁸ have been reported to be effective.

Miscellaneous conditions²²: These include psoriasis, Reiter's disease, aphthosis, pigmentary changes, drug eruptions and certain bacterial and subcutaneous/deep fungal infections such as cryptococcosis and histoplasmosis.

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