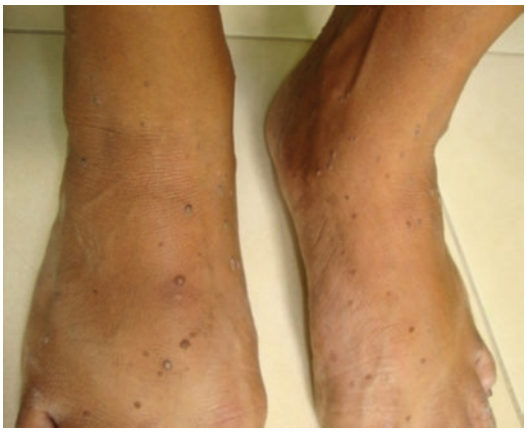
Viral Infections

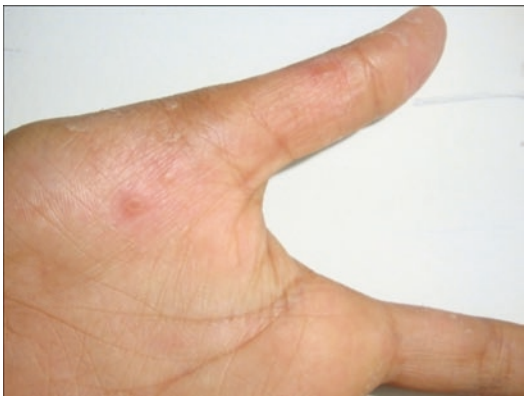
16

Premini Rajendiran



(Photographed by Dr. Ranthilaka R. Ranawaka)

1. A 19-year-old girl is worried about this asymptomatic papular eruption. What is the diagnosis?



(Photographed by Dr. Ranthilaka R. Ranawaka)

2. An 8-year-old child came with asymptomatic blisters on palms and soles for 3 days.
   a. What is the probable diagnosis?
   b. Where else would you examine to come to the definite diagnosis?

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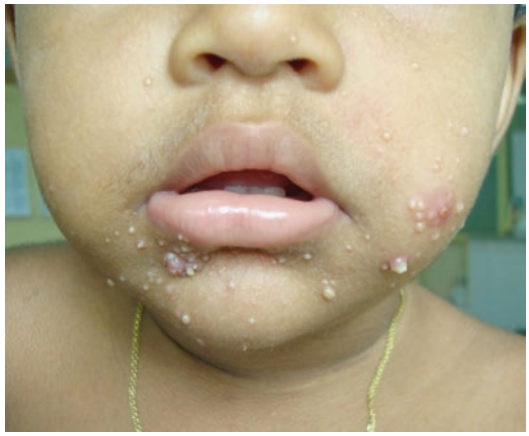
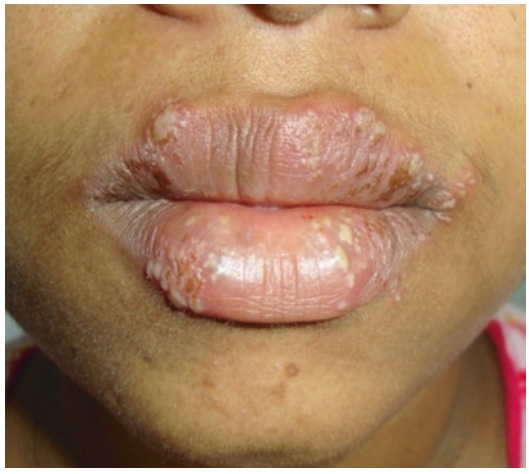


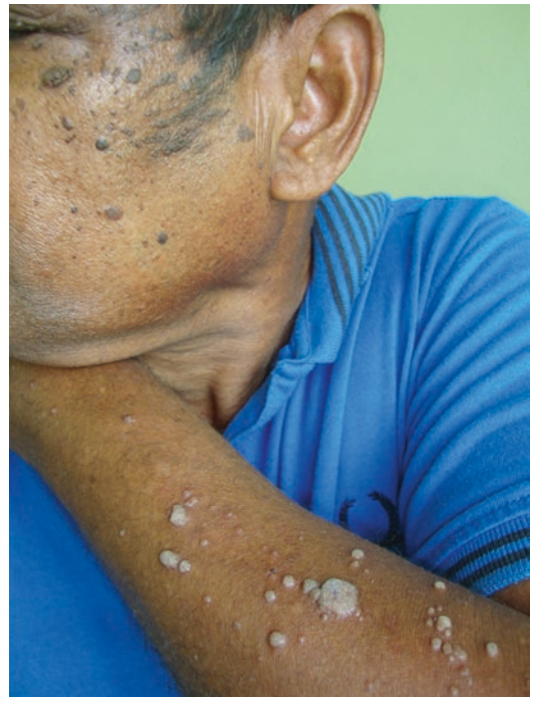
(Photographed by Dr. Ranthilaka R. Ranawaka)

3. A 1-year-old child's mother is worried about this skin eruption. What is the diagnosis?



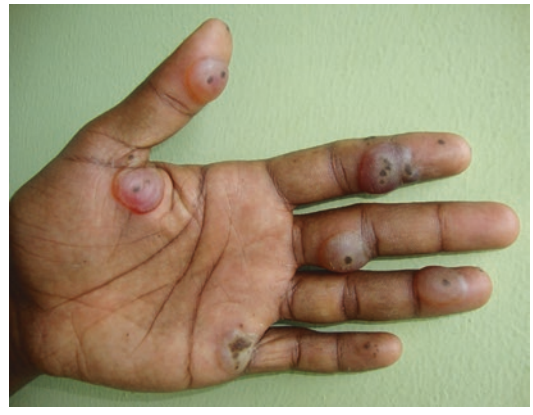
(Photographed by Dr. Ranthilaka R. Ranawaka)

4. A 24-year-old woman came with this eruption around the mouth.
   a. What is the diagnosis?
   b. What bed side test can you do to confirm the diagnosis?



(Photographed by Dr. Ranthilaka R. Ranawaka)

5. This man has asymptomatic warty growths on the (a) face and (a) the left forearm.
   What are the diagnoses in (a) and (b)?



(Photographed by Dr. Ranthilaka R. Ranawaka)

6. A 19-year-old boy's viral warts were treated with liquid nitrogen cryotherapy. The next day he came with this. What is the diagnosis? How do you manage this?

Answers

(1) Plane warts

(2) Hand, foot and mouth disease

Blisters or tiny erosions on the mouth, palms and sole. Also elbows and knees.

(3) Molluscum contagiosum

(4) Herpes gingivalis

Tzanck smear for giant cells (scrapings from the base of the blister)

(5) (a) Seborrheic keratosis (b) Viral warts

(6)He has developed blisters as a complication to cryotherapy.

Treat symptomatically, add oral antibiotics if signs of secondary bacterial infections. This will resolve in few days.

16.1 Pox Viruses

16.1.1 Molluscum Contagiosum

Causative Organism Poxvirus family—*Molluscipox.*

Clinical Presentation Common in children between 2 and 5 years age. Shiny, pearly white, hemispherical, umbilicated papule which show a central pore. After trauma, or spontaneously after several months, inflammatory changes result in suppuration, crusting and eventual destruction of the lesion. Widespread, giant or refractory molluscum are seen in HIV disease and with iatrogenic immunesuppression.

Differential Diagnosis Solitary molluscum resembles a pyogenic granuloma, keratoacanthoma and squamous cell carcinoma.

Management Self-limiting within 6–9 months; stimulation of the immune response may occur after destructive or inflammatory therapies.

Physical modalities: Cryotherapy, curettage, electrodesiccation, manual extraction

Topical therapy: Cantharidin, cidofovir, imiquimod, podophyllotoxin and trichloroacetic acid

Laser therapy: Co_2 laser, pulsed dye laser (Figs. 16.1, 16.2) (Sterling 2016).

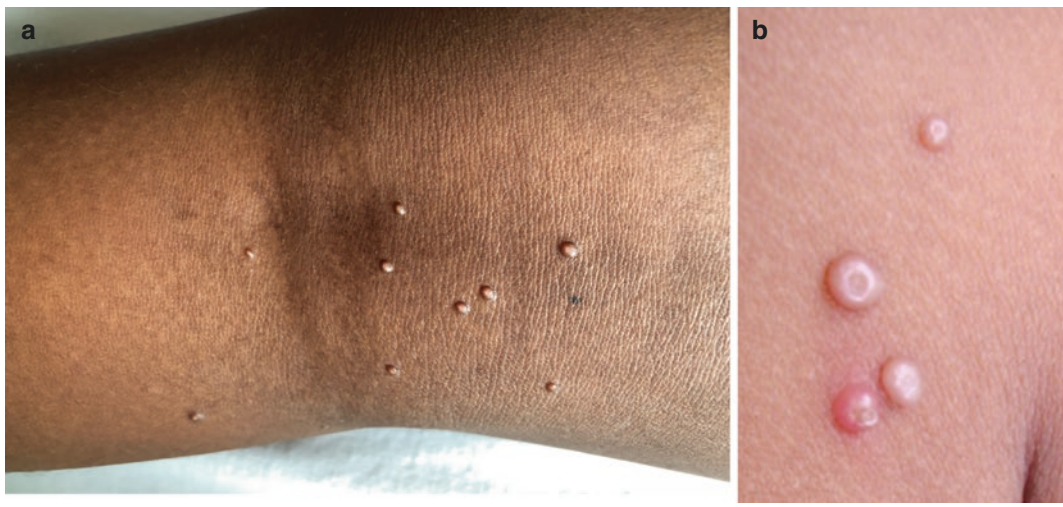


Fig. 16.1 Molluscum contagiosum. (**a**) Multiple pearly umbilicated papules in the popliteal fossa (**b**) closer view (photographed by Dr. Premini Rajendiran)

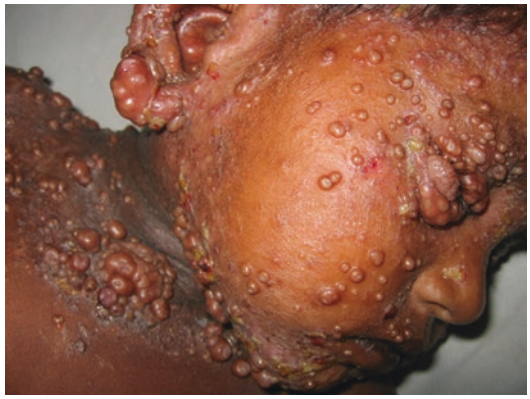


Fig. 16.2 Molluscum contagiosum. Extensive lesions in an immunocompromised child (photographed by Dr. Premini Rajendiran)

16.2 Herpes Virus Infections

Eight members of the group can infect humans. Herpes simplex virus (HSV) and varicella-zoster virus (VZV) predominantly cause cutaneous disease.

A notable feature of infection by members of the herpes virus group is the absence of virus elimination following clinical recovery. Virus persists throughout the person's life as a latent infection in the cells for which the strain is specific. Under certain conditions, especially immune suppression, the virus may become reactivated and produce an acute infective episode with cellular damage.

16.2.1 Herpes Simplex Virus

16.2.1.1 Primary Herpetic Gingivostomatitis/Herpes Labialis/Cold Sore

Causative Organisms Herpes simplex virus type I, less frequently HSV 2.

Clinical Presentation Itching or burning precedes by an hour or two the development of small closely grouped vesicles on an inflamed base. In genitals they break easily and form ulcers.

Differential Diagnosis Streptococcal infections, diphtheria, candidiasis, aphthosis, Coxsackie infections including herpangina, Behcet's syndrome and the Stevens-Johnson syndrome.

Management Acyclovir, famciclovir and valacyclovir reduce the duration of viral shedding, pain and time to healing in the treatment of primary and recurrent orolabial and genital herpes simplex infection (Figs. 16.3–16.6) (Mendoza et al 2012).

16.2.1.2 Primary Herpes Genitalis

Causative Organisms Commonly HSV 2, also HSV 1.

Clinical Presentation Penile ulceration is most frequent on the glans, prepuce and shaft of the penis. They are sore and painful, and there may be associated oedema. In male homosexuals, this is common in the perianal area and may extend into the rectum.

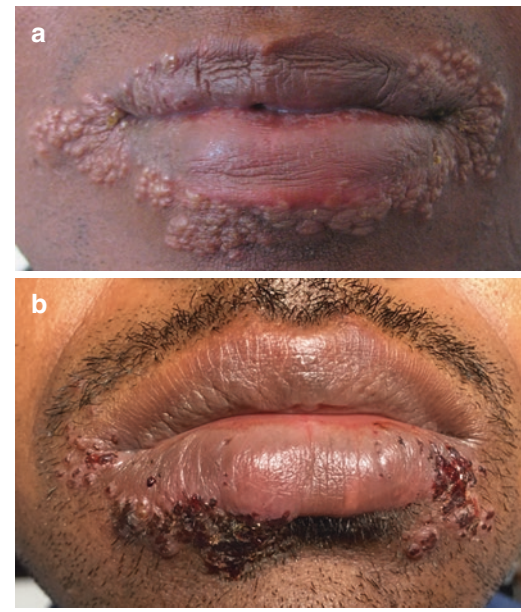


Fig. 16.3 Labial herpes simplex virus infection. Grouped vesicular lesions (**a**), crusted lesions (**b**) (photographed by Dr. Premini Rajendiran)

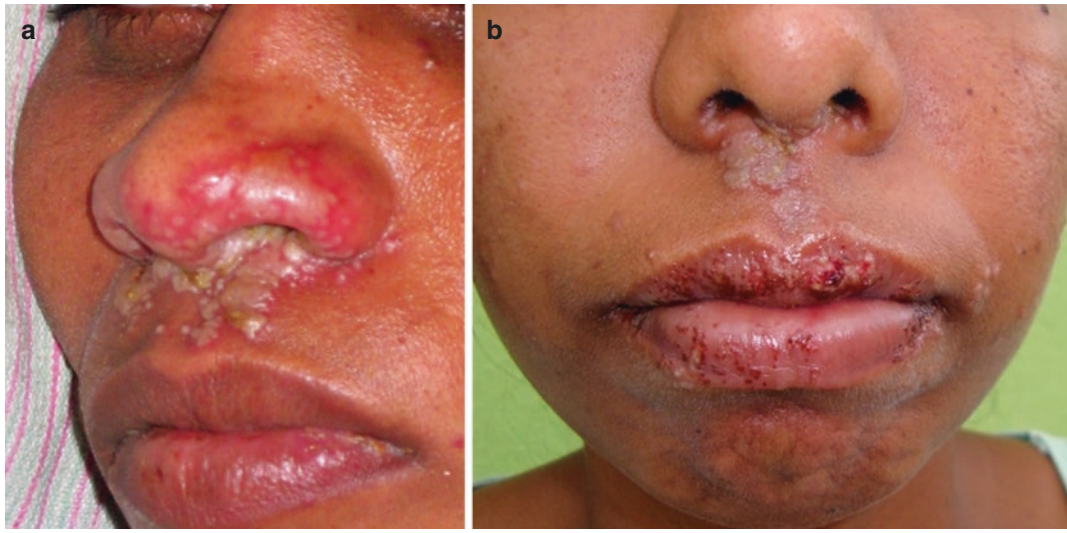


Fig. 16.4 Herpes simplex infection. Grouped pustular lesions on the anterior nares and on the philtrum (photographed by Dr. Premini Rajendiran and Dr. Ranthilaka R. Ranawaka)

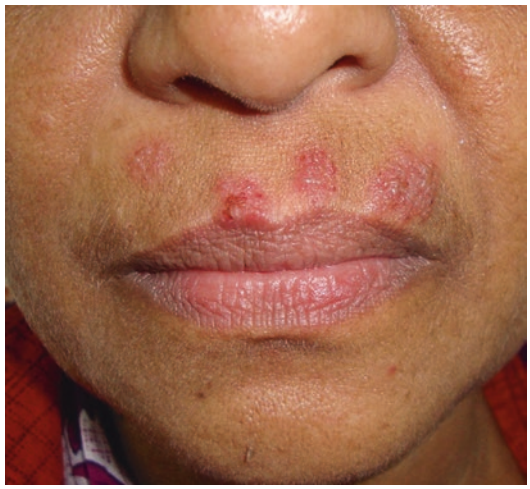


Fig. 16.5 Labial herpes simplex virus infection. Vesicular eruption may be very subtle (photographed by Dr. Ranthilaka R. Ranawaka)

In the women, similar lesions occur on the external genitalia and mucosae of the vulva, vagina and cervix. Pain and dysuria are common. Infection of the cervix may progress to a severe ulcerative cervicitis. Recurrences are fairly common, occurring two to six times per year.

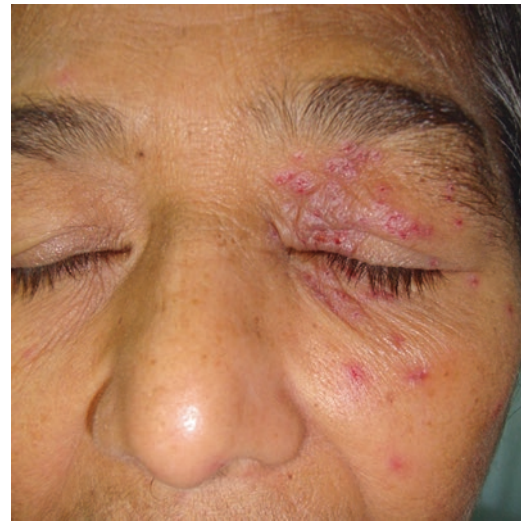


Fig. 16.6 Herpes infection around the left eye. Note the localized punched out grouped ulcers (photographed by Dr. Ranthilaka R. Ranawaka)

Management

Preventive strategies to avoid spread include the use of barrier contraception and local microbicides.

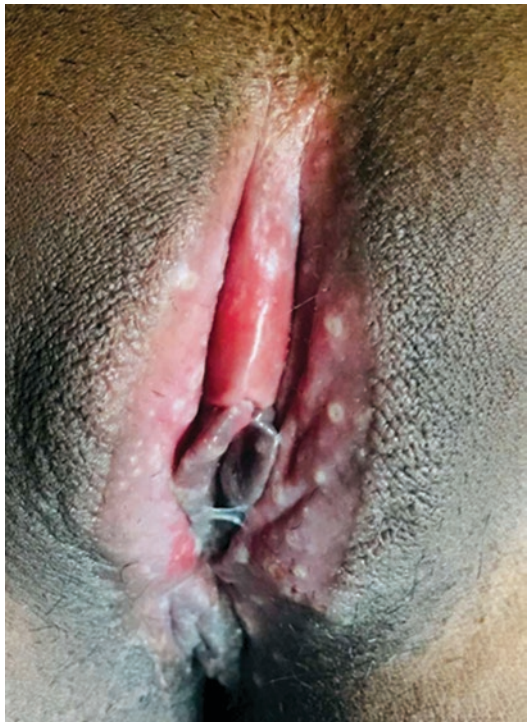


Fig. 16.7 Primary genital herpes. Multiple vesiculopustules on the labia majora (photographed by Dr. Premini Rajendiran)

Aciclovir, valaciclovir and famciclovir are used.

In children with genital herpes, sexual abuse must be considered (Fig. 16.7).

16.2.1.3 Neonatal Herpes

Herpes simplex infection of a baby within 28 days of birth, usually acquired vertically from the mother.

Primary genital herpes infection or active recurrent infection in the mother at the time of delivery makes the risk of transmission to the baby during vaginal delivery very high.

The effects on the baby range in severity from disseminated disease, affecting multiple organs, or limited to the skin, eyes and mouth.

16.2.2 Varicella-Zoster Virus Infections

Varicella-zoster virus is the cause of both varicella (chickenpox) and zoster (shingles). The primary infection of varicella includes viraemia and a widespread eruption, after which the virus persists in nerve ganglion cells, usually sensory. Zoster is the result of reactivation of this residual latent virus. The virus is transmitted by droplet infection from the nasopharynx. Patients are infectious to others from about 2 days before to 5 days after the onset of the rash.

16.2.2.1 Varicella/Chickenpox

Causative Organism Primary infection with varicella-zoster virus (VZV).

Clinical Features The incubation period is usually 14–17 days. After a day or two of fever and malaise, often slight or absent in children, a scarlatiniform or morbilliform erythema is followed by the development of papules which very rapidly become tense, clear vesicles. Within a few hours, the contents become turbid, and the pustules are surrounded by red areolae.

They are most numerous on the trunk, then on the face and scalp and on the limbs. Their distribution is centripetal, and on the limbs, the eruption is more profuse on the thighs and upper arms than on the lower legs and forearms. The total number of lesions is very variable. Vesicles are found in mouth and other mucous membranes. In the absence of secondary infection, the lesions heal without scarring.

Varicella confers lasting immunity and second attacks are uncommon.

Clinical variant include haemorrhagic varicella where there is extensive eruption of haemorrhagic vesicles with high fever and severe constitutional symptoms.

Infection during pregnancy has extra risks for both mother and foetus. Maternal infection may

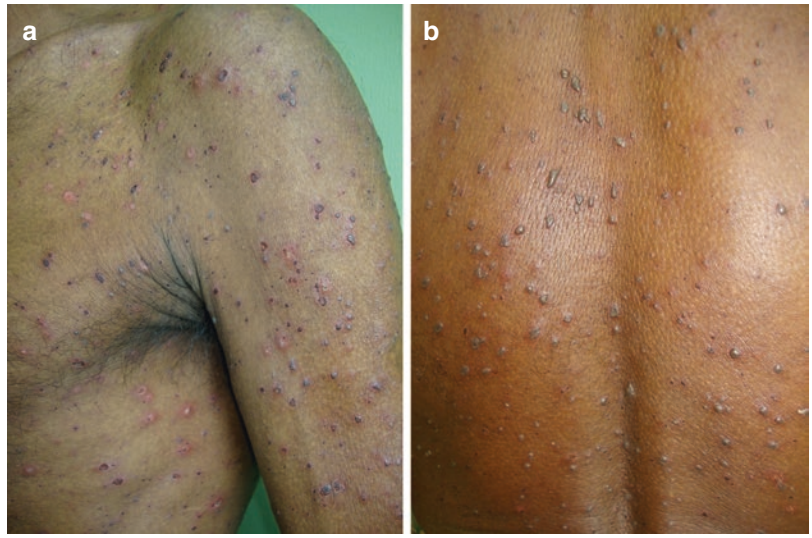


Fig. 16.8 Chickenpox in a 64-year-old man. The vesicles distribution is centripetal (i.e. most numerous on the trunk). A characteristic feature is the presence of lesions at different stages in each site. Fever and malaise precede vesicular eruption by day or two (photographed by Dr. Ranthilaka R. Ranawaka)

lead to pneumonitis. Infection during second trimester carries 2% risk of developing congenital varicella syndrome.

Complications Encephalitis, varicella pneumonia, hepatitis, secondary bacterial infection, rhabdomyolysis, Stevens-Johnson syndrome and chronic varicella

Investigations In most cases the diagnosis is clinical. In atypical cases, PCR of vesicle fluid is used for confirmation.

Management

Pre-exposure prophylaxis

A live attenuated vaccine is effective in preventing varicella in healthy children. The vaccine is given in 2 doses, 3 months apart, resulting in approximately 90% seroconversion with 75% of responding recipients maintaining detectable antibody for up to 10 years.

Post-exposure prophylaxis

Specific zoster immune globulin administered within 10 days of contact reduces the severity of varicella but does not always prevent it.

Antiviral prophylaxis. In the immunocompetent person, aciclovir given from about 9 days after exposure for 1 week appears to be effective in aborting or reducing the severity of chickenpox.

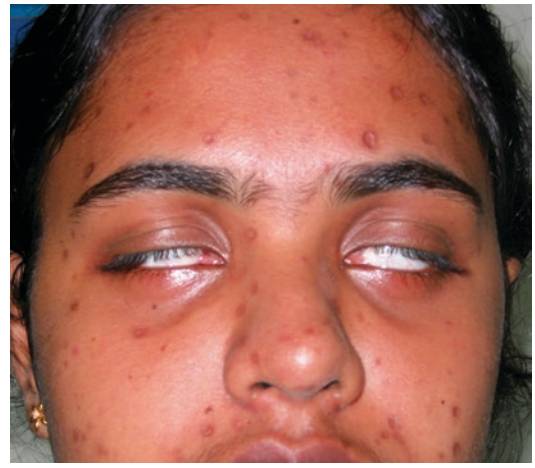


Fig. 16.9 Varicella-zoster scars. Note the bilateral lower motor type of seventh nerve palsy following varicella-zoster infection (photographed by Dr. Premini Rajendiran)

Treatment: Acyclovir (Figs. 16.8, 16.9).

16.2.2.2 Zoster/Shingles/Herpes Zoster

Causative Organism Reactivation of latent VZV

Zoster is a segmental eruption due to reactivation of latent VZV from dorsal root ganglia. Zoster patients are infectious, both from virus in the lesions and, in some instances, the nasopharynx. Zoster is not common in childhood and

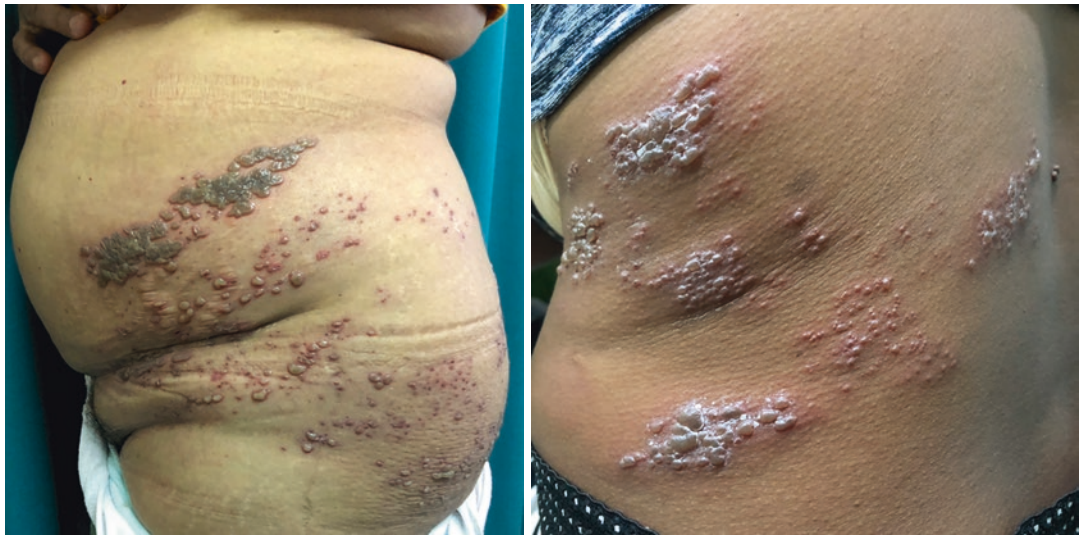


Fig. 16.10 Herpes zoster of thoracic dermatomes (photographed by Dr. Premini Rajendiran)

young adult life. Over the age of 50 and with immunosuppression the incidence rises.

Clinical Features Herpes zoster begins with a prodrome of itching, tingling, hyperesthesia, intense pain and tenderness followed by painful eruption of grouped vesicles in a dermatomal distribution. The eruption can involve more than one dermatome and occasionally cross the midline.

Complications of zoster include postherpetic neuralgia, secondary bacterial infection, scarring, meningoencephalitis, pneumonitis and hepatitis. The most common complication is postherpetic neuralgia characterized by dysesthetic pain that lasts after skin lesions have healed.

Differential Diagnosis Zosteriform HSV infection, cellulitis, bullous impetigo, localized contact dermatitis and phytophotodermatitis.

Treatment
Acyclovir, famciclovir and valacyclovir are all FDA-approved for the treatment of herpes zoster. Beginning antiviral treatment within 72 hours of the skin eruption is optimal, but initiation up to 7 days appears to be beneficial.

Multiple treatments are available for postherpetic neuralgia. Low-dose tricyclic antidepressants, gabapentin alone and in combination can be used (Figs. 16.10, 16.11, 16.12, 16.13, 16.14, 16.15, 16.16, 16.17).

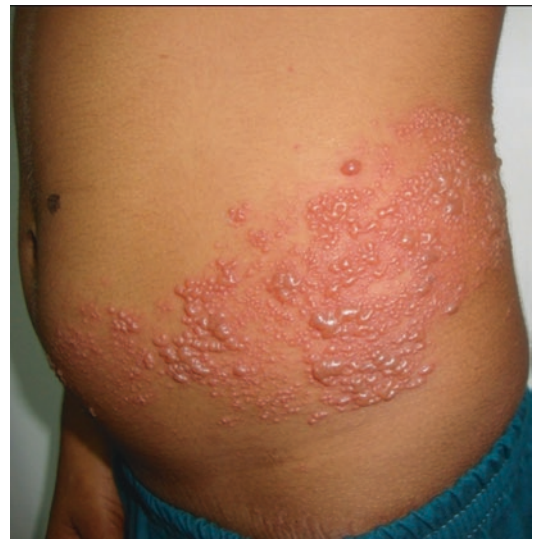


Fig. 16.11 Herpes zoster of the thoracic dermatome (photographed by Dr. Ranthilaka R. Ranawaka)

16.2.2.3 Eczema Herpeticum/Kaposi Varicelliform Eruption

Causative Organisms HSV-1, HSV-2, Coxsackie A6 and A16 and vaccinia have all been implicated in similar eruption.

A widespread cutaneous eruption most commonly due to HSV-1 (which usually cause mild localized disease), occurring in patients with pre-existing skin disease. Vesicles erupt in crops, confined to abnormal skin, but often widely disseminated simulating chickenpox.

Commonest predisposing condition is atopic eczema. Eczema herpeticum results from primary or recurrent infection. It has followed trauma or cosmetic procedures.

Treatments Less ill patients respond to oral antiviral therapy. Severe cases need intravenous acyclovir therapy as early as possible (Fig. 16.18).

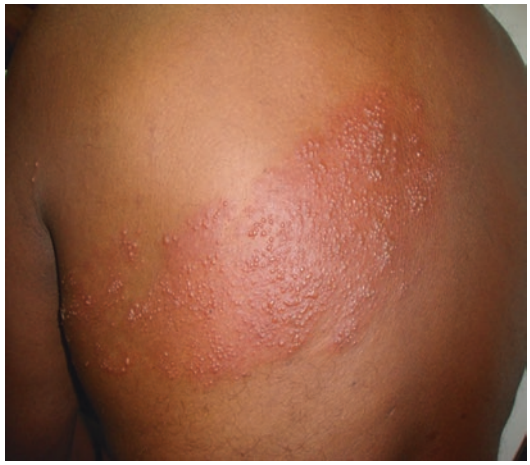


Fig. 16.12 Herpes zoster of thoracic dermatome distribution (photographed by Dr. Ranthilaka R. Ranawaka)

16.3 Human Papilloma Virus

Human papilloma viruses (HPV) commonly cause benign papillomas and warts.

HPV-16 and HPV-18 are recognized as the etiologic agents for cervical cancer.

Clinical Features

Cutaneous HPV types infect the skin and induce common warts, palmar and plantar warts, mosaic warts, flat warts and butcher's warts.

Epidermodysplasia verruciformis is a rare genetic disease characterized by a particular susceptibility to cutaneous infections with HPV.

Bowenoid papulosis presents as multiple red-brown papules or confluent plaques on the external genitalia and on the perineum. These lesions resemble genital warts, but histopathology shows

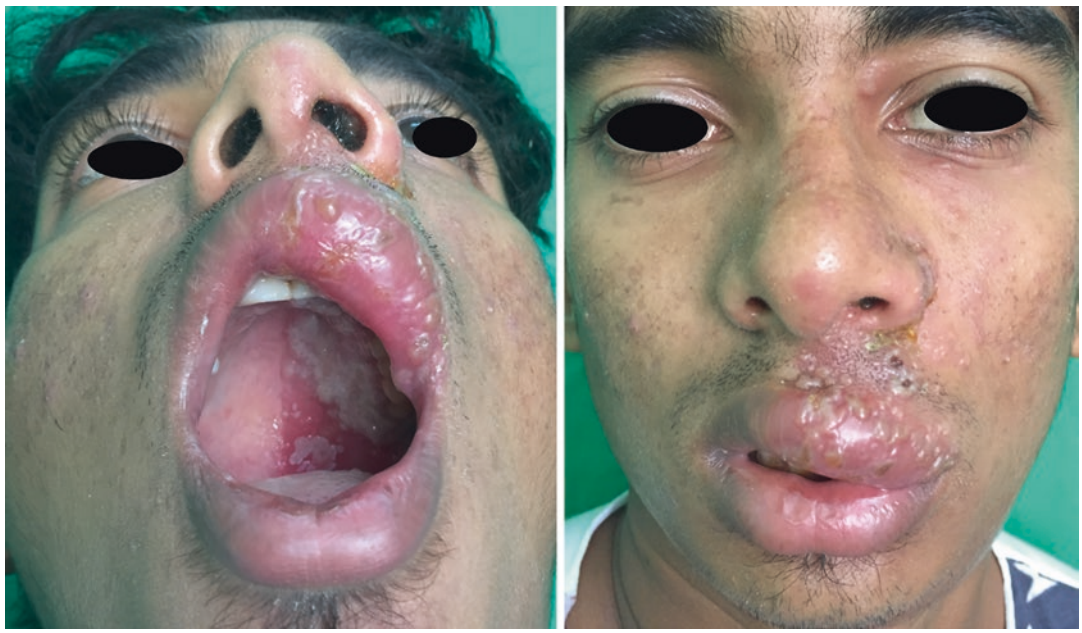


Fig. 16.13 Herpes zoster of maxillary division of trigeminal nerve (photographed by Dr. Premini Rajendiran)

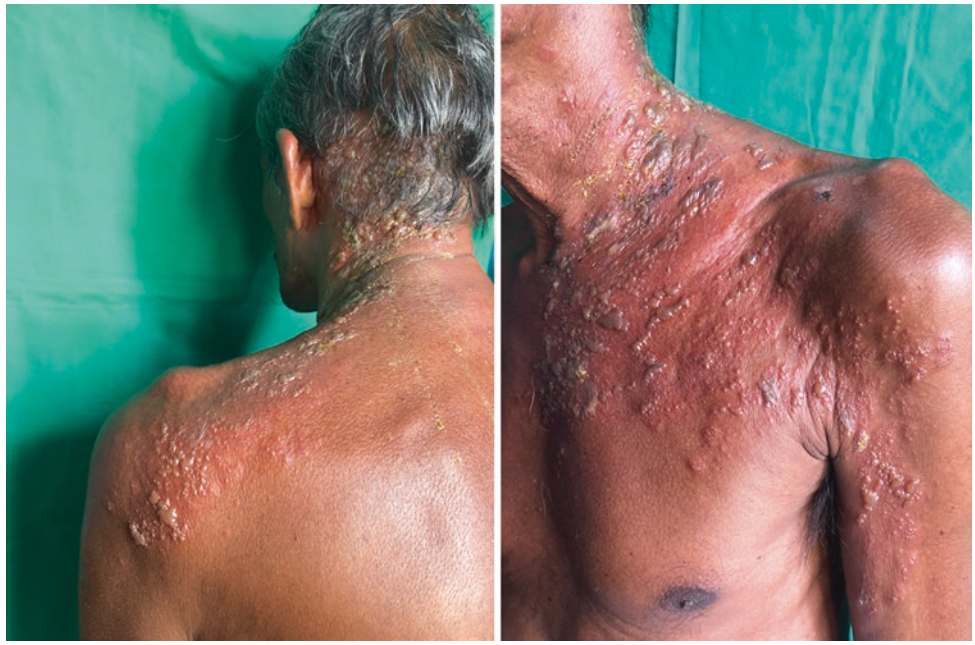


Fig. 16.14 Herpes zoster of cervical dermatomes (photographed by Dr. Premini Rajendiran)

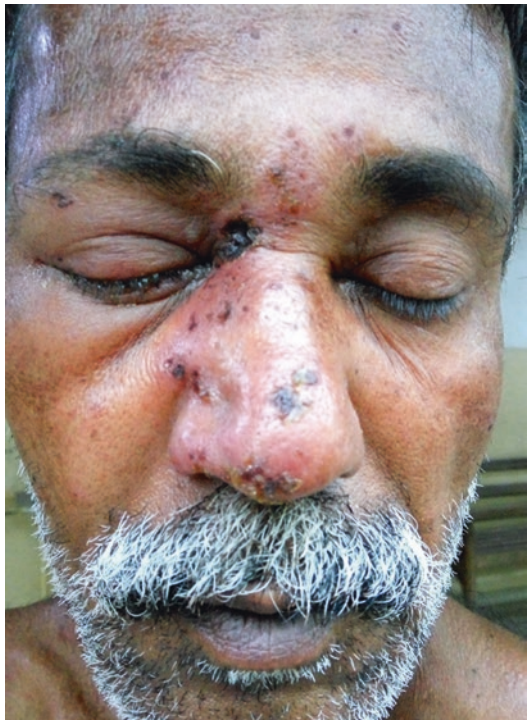


Fig. 16.15 Herpes ophthalmicus in a 67-year-old fisherman. When vesicles are on the side of the nose that indicates involvement of the nasociliary nerve (Hutchinson sign) which indicate high risk of ophthalmic complications. (photographed by Dr. Ranthilaka R. Ranawaka)

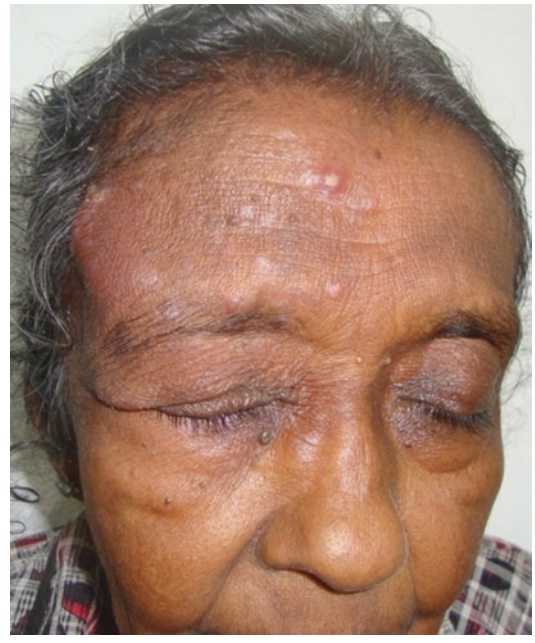


Fig. 16.16 Healed zoster on the ophthalmic division of the trigeminal nerve. Note unilateral blepharochalasis on the affected side. In this patient it is a localized form of post-inflammatory elastolysis. (Blepharochalasis is laxity of the eyelid skin due to a defect in the elastic tissue.) (Photographed by Dr. Ranthilaka R. Ranawaka)

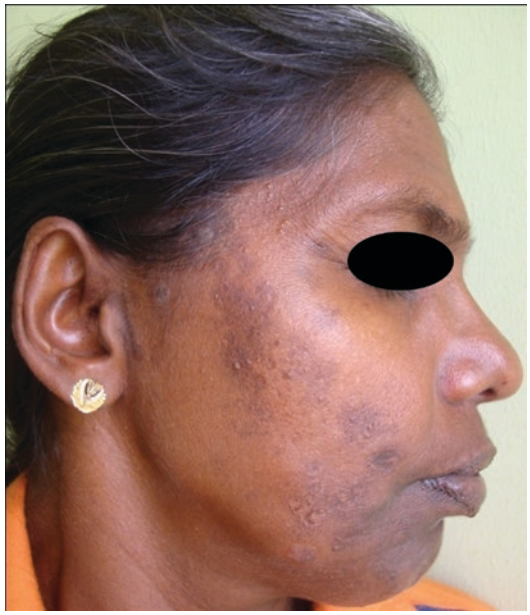


Fig. 16.17 Post-inflammatory hyperpigmentation following a herpes zoster along the mandibular division of the trigeminal nerve. This woman came to us with postherpetic neuralgia (photographed by Dr. Ranthilaka R. Ranawaka)

a high-grade squamous intraepithelial lesion (HSIL) or squamous cell carcinoma in situ.

Erythroplasia of Queyrat manifests as a well-demarcated velvety erythematous plaque on the glabrous skin of the penis or vulva that histologically is of HSIL. Both Bowenoid papulosis and erythroplasia are premalignant lesions, which predominantly contain high risk HPV-16. (Sterling 2016, Kimbauer and Lenz 2012).

Differential diagnosis

Common warts—seborrheic keratoses, cutaneous horns, actinic keratoses, keratoacanthomas, Spitz naevi, tuberculosis verrucosa cutis and hypertrophic lichen planus

Anogenital lesions (condylomata acuminate)—condylomata lata, molluscum contagiosum, pearly penile papules, vestibular papillomatosis and angiokeratomas

Erythroplasia of Queyrat—erosive lichen planus and Zoon's balanitis

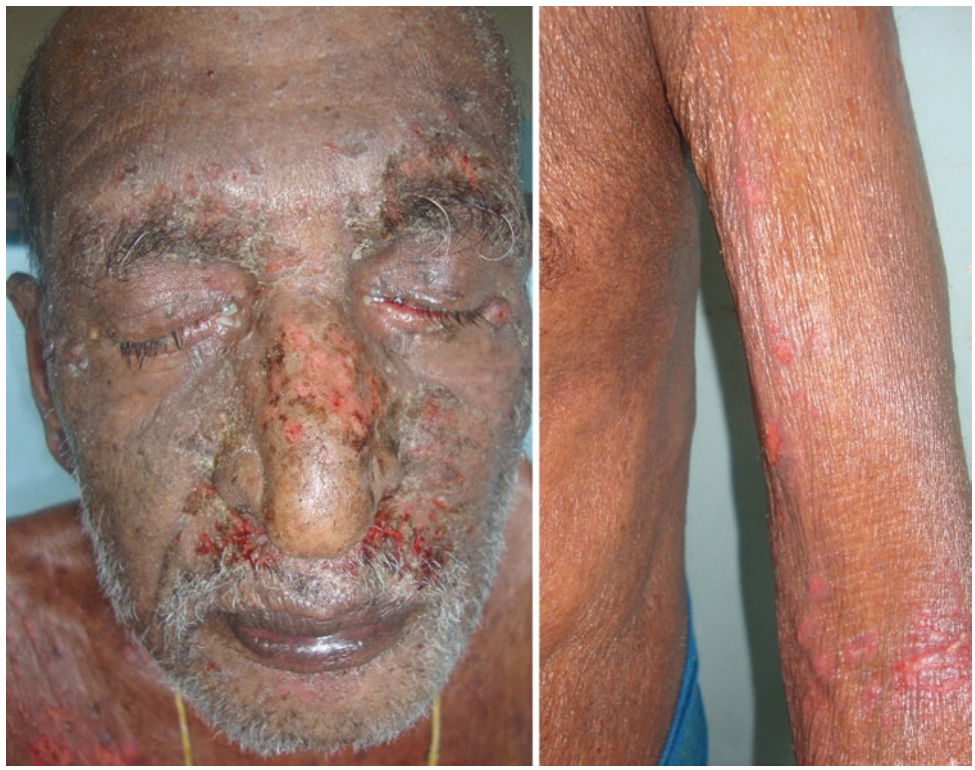


Fig. 16.18 Eczema herpeticum developed in a 74-year-old man who had exfoliative dermatitis following anti-tuberculous therapy. Note the discrete punched-out ulcers on the arm and the elbow which were clue to the clinical diagnosis. (photographed by Dr. Ranthilaka R. Ranawaka)

Treatment

Currently there is no specific treatment available to cure HPV infection. Existing treatment modalities aim on the destruction of visible lesions or induction of cytotoxicity against infected cells.

Local destructive therapy—cryotherapy, trichloroacetic acid, electrosurgery, curettage, scalpel or scissor excision, laser vaporization and photodynamic therapy.

Topical and intralesional cytotoxic therapy—podophyllotoxin, topical 5-fluorouracil and intralesional bleomycin.

Topical immunomodifiers—Imiquimod cream, sinecatechins ointment.

Systemic immunomodifiers—interferons.

16.3.1 Common Warts, Verrucae Vulgaris, Verrucae Palmares et Plantares (Figs. 16.19–16.25)

16.3.2 Plane Warts/Verrucae Planae

Plane warts are smooth, flat or slightly elevated and are usually skin coloured or greyish yellow but may be pigmented. They are round or polygonal in shape and vary in size from 1 to 5 mm diameter (Figs. 16.26a and b).

Differential Diagnosis Lichen planus.

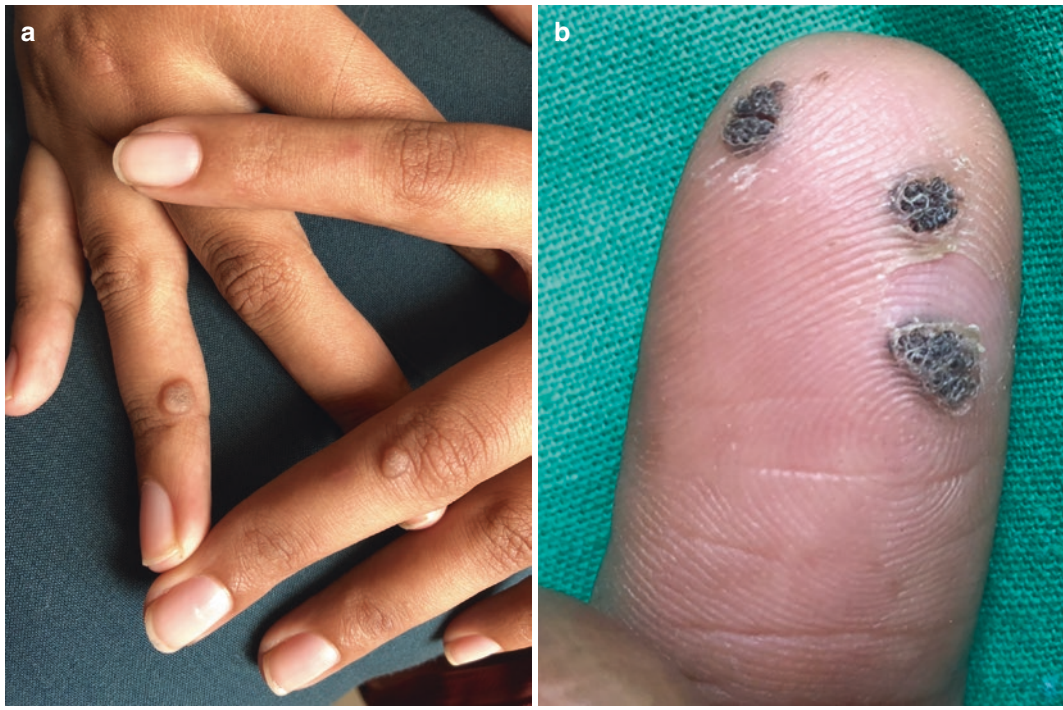


Fig. 16.19 (**a**, **b**) Common warts on the fingers (photographed by Dr. Premini Rajendiran)

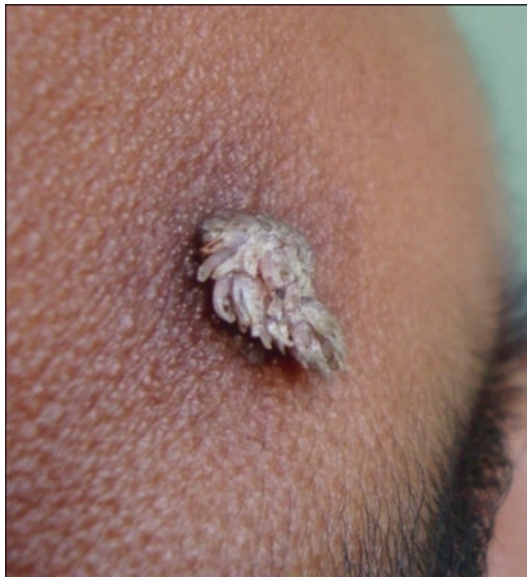


Fig. 16.20 Filiform wart/digitate wart on the forehead of a man. Filiform wart is a clinical variant of common wart (photographed by Dr. Ranthilaka R. Ranawaka)

16.3.3 Anogenital Warts (Fig. 16.27a and b)

16.3.4 Epidermodysplasia Verruciformis

Epidermodysplasia verruciformis (EV) is an inherited disorder in which widespread and persistent infection with HPV occurs. There is a combination of plane warts, pityriasis versicolor-like lesions and reddish plaques. Malignant change is very common in adult life, but metastasis is rare.

Acquired EV occurs in patients with organ and bone marrow transplant recipients and patients receiving long-term immunesuppression for inflammatory disease and disorders of immune compromise such as primary immunodeficiencies, lymphoma and in HIV/AIDS (Fig. 16.28).

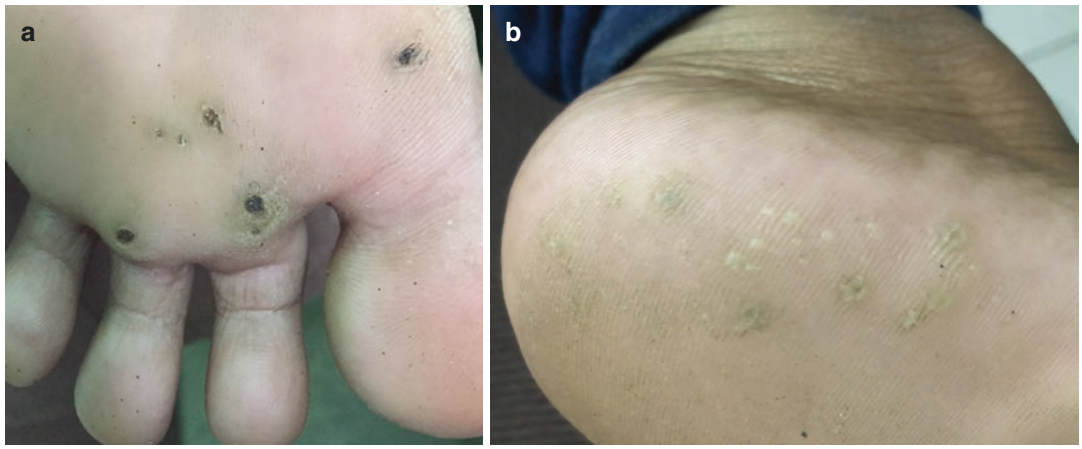


Fig. 16.21 (**a**, **b**) Plantar warts (photographed by Dr. Premini Rajendiran)

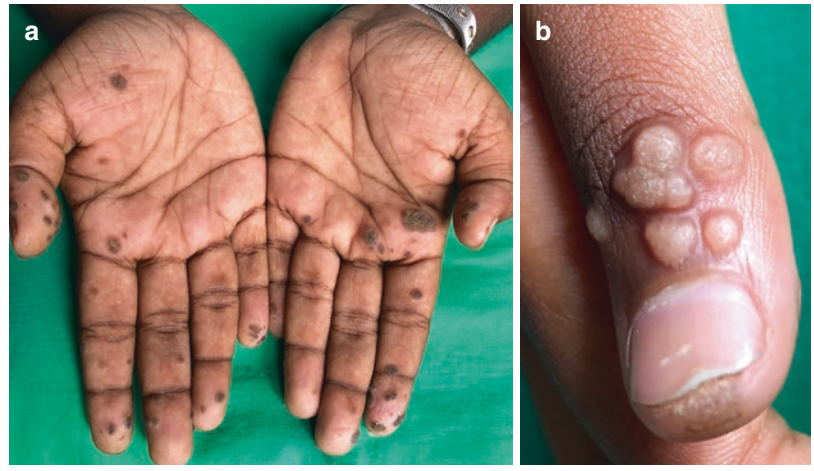


Fig. 16.22 (**a**, **b**) Common warts on the fingers and palm (photographed by Dr. Premini Rajendiran)

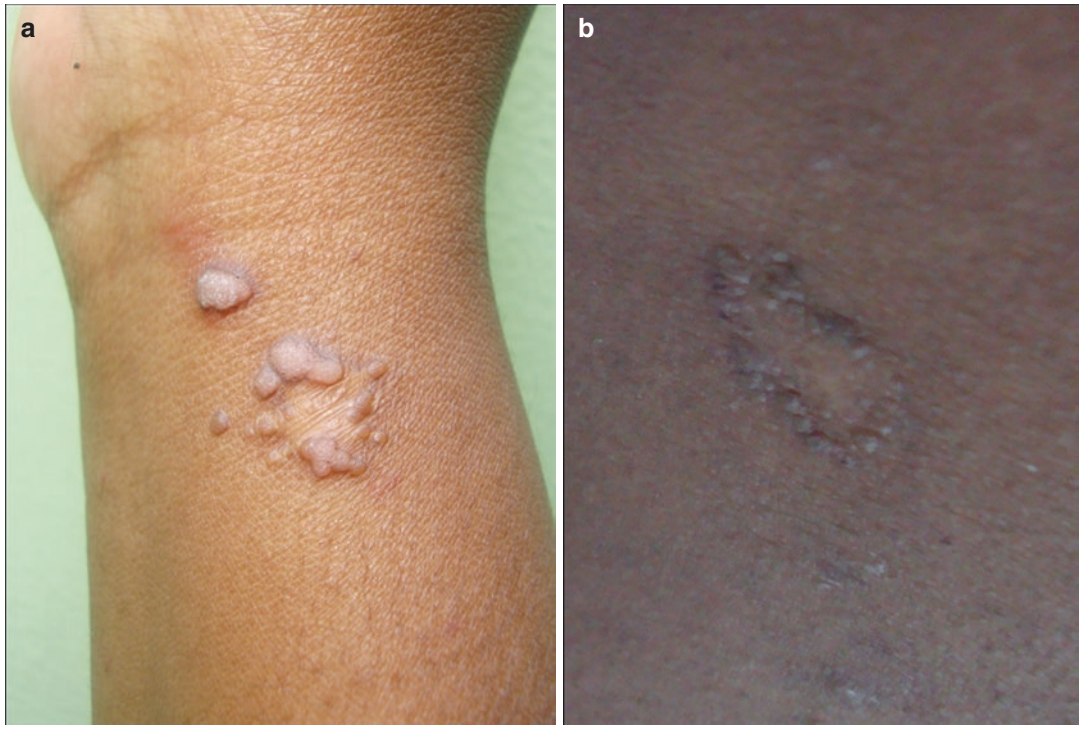


Fig. 16.23 (**a**, **b**) Following surgical excision of a single viral wart, multiple viral warts had aroused around the scar. Therefore surgical excision of viral warts is not recommended (photographed by Dr. Ranthilaka R. Ranawaka)

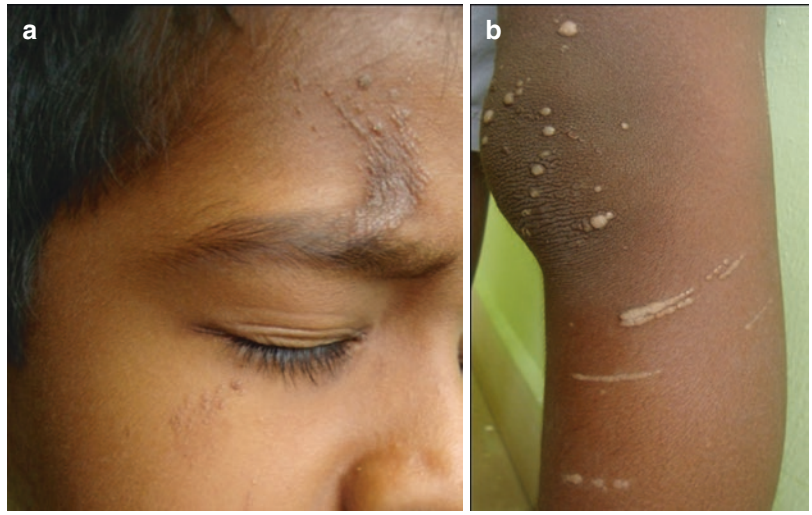


Fig. 16.24 (**a**, **b**) Viral warts appearing along the scratch marks (Koebner phenomena) (photographed by Dr. Ranthilaka R. Ranawaka)

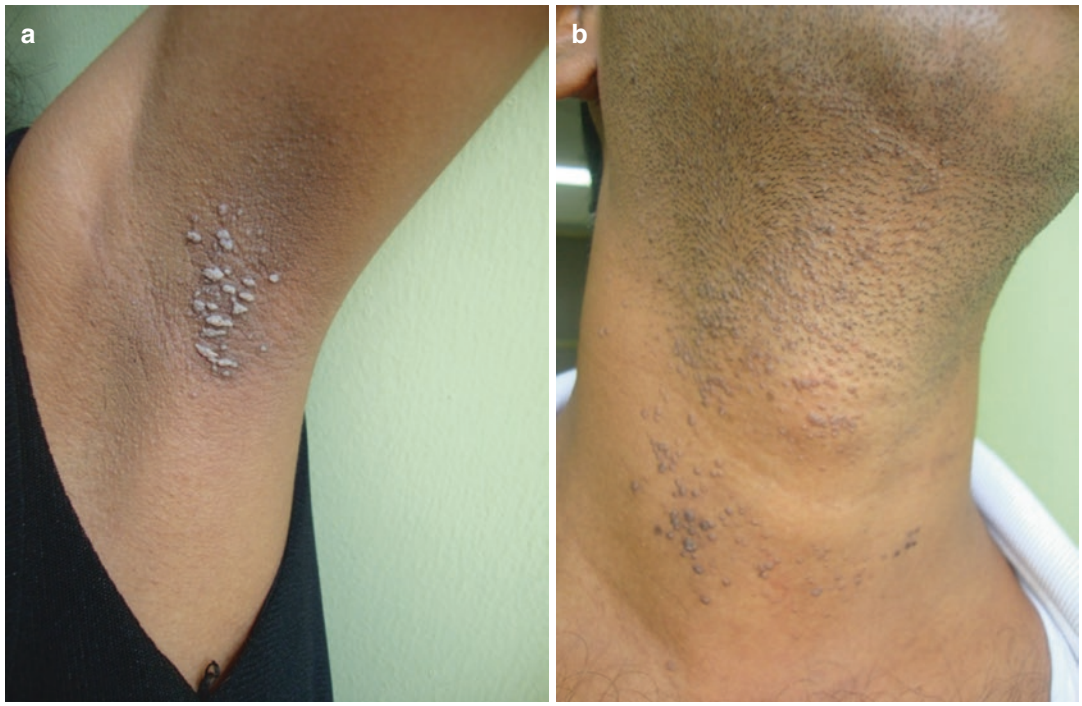


Fig. 16.25 (**a**, **b**) Shaving has spread warts in axilla and beard area (Koebner phenomena) (photographed by Dr. Ranthilaka R. Ranawaka)

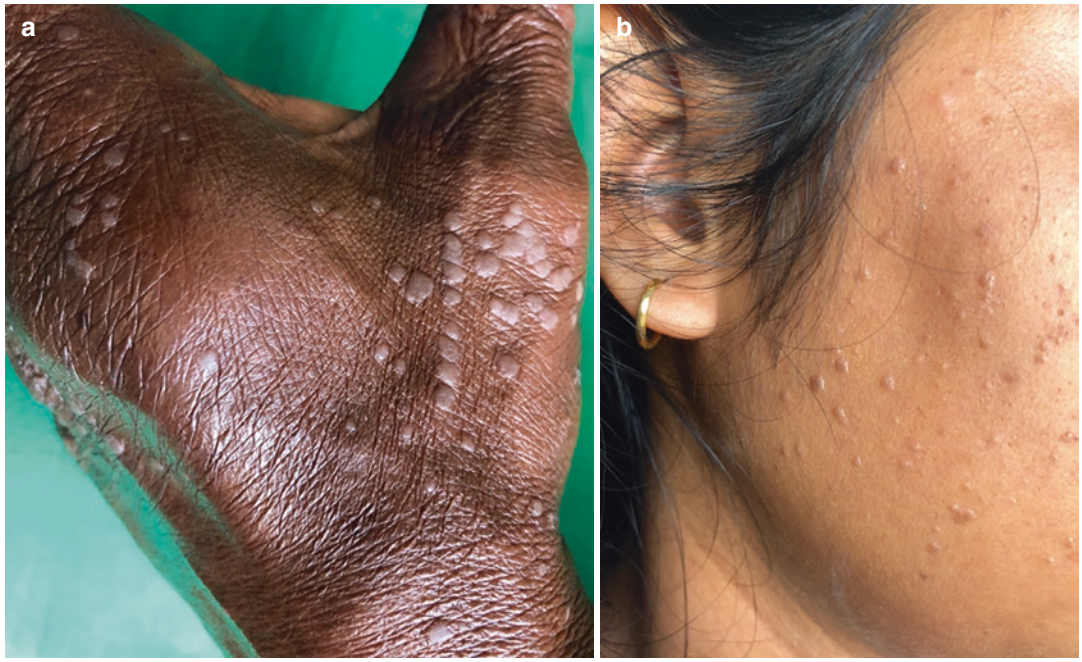


Fig. 16.26 (**a**, **b**) Plane warts on the hand and face (photographed by Dr. Premini Rajendiran)

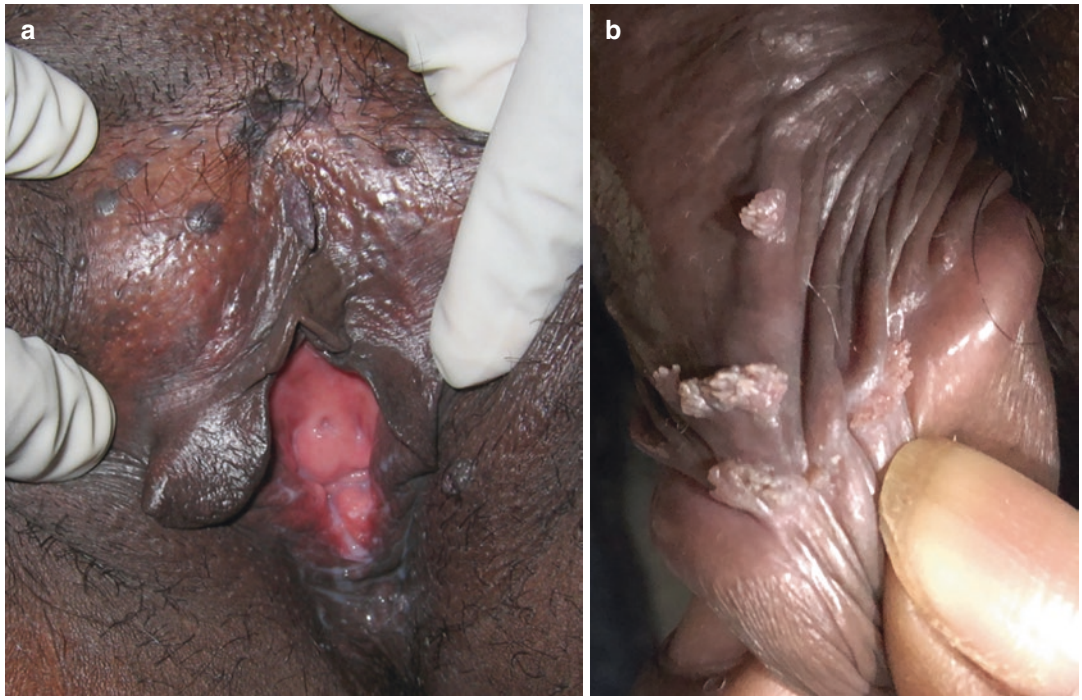


Fig. 16.27 (**a**, **b**) Genital warts on labia majora and penile skin (photographed by Dr. Premini Rajendiran)

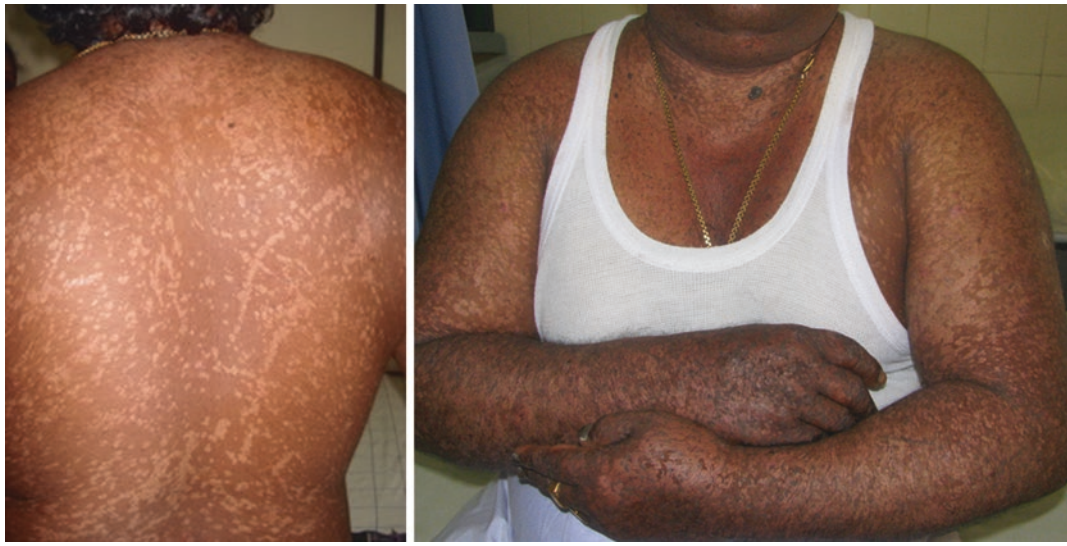


Fig. 16.28 Epidermodysplasia verruciformis. Widespread flat hypopigmented lesions on the trunk (**a**). Note some hyperpigmented lesions on the forearm (picture courtesy Dr. Felicia Srisaravanapavananthan, consultant dermatologist, Teaching Hospital Jaffna, Sri Lanka)

16.4 Flavivirus Infections

This family comprises antigenically related types of both mosquito and tick-borne viruses.

16.4.1 Dengue/Breakbone Fever

Dengue is mosquito-borne virus disease in humans. The virus causes a range of manifestations from asymptomatic to dengue haemorrhagic fever which carries a high mortality.

Causative Organism There are four antigenically distinct dengue viruses, serotypes 1–4. The virus is spread via mosquitoes, *Aedes aegypti*.

Clinical Features Dengue is a flu-like illness with an incubation period of 2–8 days. The characteristic exanthem of dengue fever is estimated to occur in 50–82% of patients with dengue fever. The initial rash in dengue fever is transient flushing erythema of face that typically occurs shortly before or 24–48 hours after the onset of symptoms. The second rash asymptomatic maculopapular morbilliform rash occurs 3–6 days after the onset of fever. In some cases, there is generalized confluent erythema with petechiae with rounded islands of sparing "white islands in a sea of red". Pruritus is reported in 16–27% of patients in different studies. Haemorrhagic manifestations on the skin petechiae, purpura and ecchymosis with positive tourniquet test are seen in dengue haemorrhagic fever and dengue shock syndrome (Thomas et al 2010).

Differential Diagnosis Measles, rubella, roseola, infectious mononucleosis, chikungunya fever and Rocky Mountain spotted fever.

Management In endemic areas, vector control is important. There is no specific antiviral treatment; management is symptomatic and supportive (Fig. 16.29).

16.4.2 Chikungunya Fever

Chikungunya fever is an emerging infection in Reunion Island, India, Sri Lanka, Europe and

China. It is a mosquito-borne disease. The virus can be transmitted from mother to child. *Aedes aegypti* and *Aedes albopictus* are the common vectors.

Clinical Features It is a flu-like illness with acute onset of fever and joint pains. A rash develops in half of those affected. The eruption is usually maculopapular and occasionally purpuric. Anogenital and oral ulceration occasionally observed. In children the lesions may be vesicular and pigmented.

Management General supportive measures as for a virus induced fever are used.

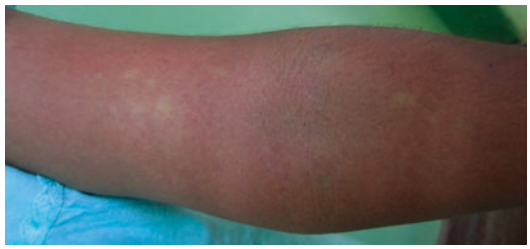


Fig. 16.29 Exanthem of dengue fever. "White islands in a sea of red" (photographed by Dr. Premini Rajendiran)

16.5 Enterovirus Infection

16.5.1 Hand, Foot and Mouth Disease

Causative organism Coxsackie virus.

Children less than 10 years of age are commonly affected. Adults are also frequently affected.

Clinical Features In children, the illness starts with a low-grade fever, sore throat and malaise. Blisters in the mouth soon break down leaving superficial ulcers. Flaccid greyish 2–5 mm oval blisters appear on the palms and soles. Rarely, a widespread eruption occurs over trunk, buttock and perioral area. In some, the eruption may be papular or maculopapular without vesicles.

Differential Diagnosis Herpes simplex, Stevens-Johnson syndrome, chickenpox, Kaposi varicelliform eruption

Management Supportive (Figs. 16.30, 16.31, 16.32).

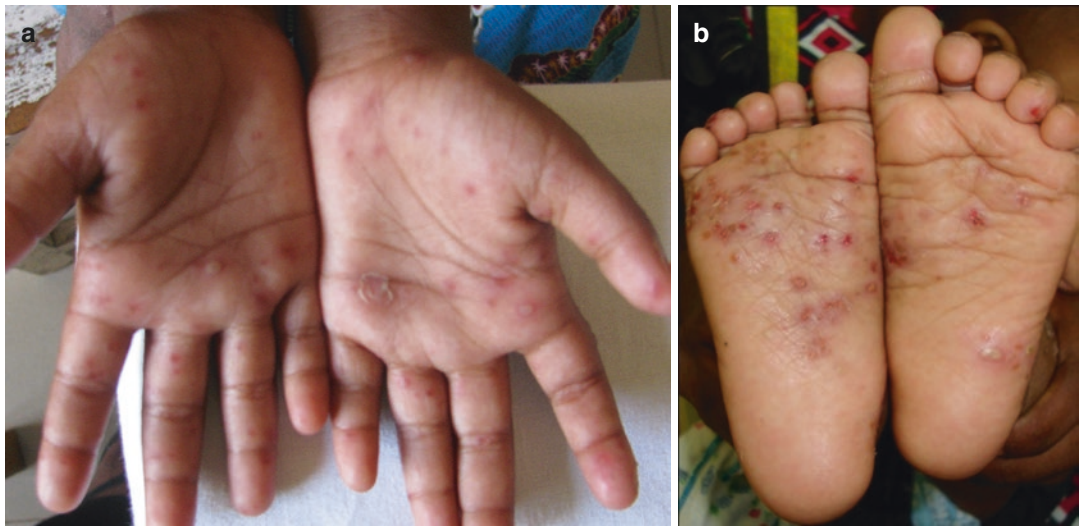


Fig. 16.30 (**a**, **b**) Hand, foot and mouth disease in a child. Note the erythematous macules and greyish oval vesicles on palms and soles (photographed by Dr. Premini Rajendiran and Dr. Ranthilaka R. Ranawaka)

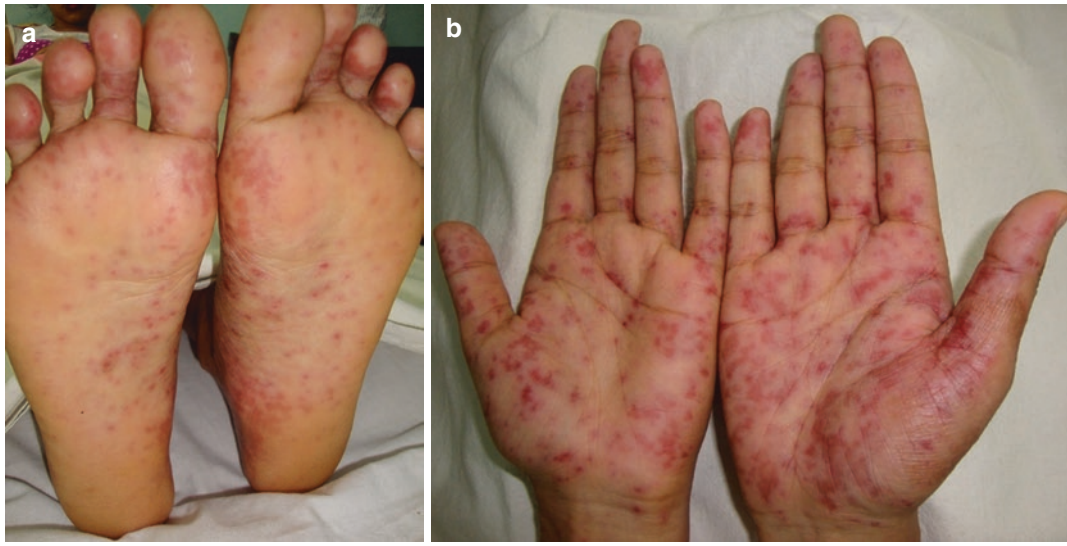


Fig. 16.31 (**a**, **b**) Hand, foot and mouth disease in a 24-year-old woman. The erythematous macules on palms and sole (sparing other areas of the body) were the clue to diagnosis and she responded dramatically to oral acyclovir (photographed by Dr. Ranthilaka R. Ranawaka)

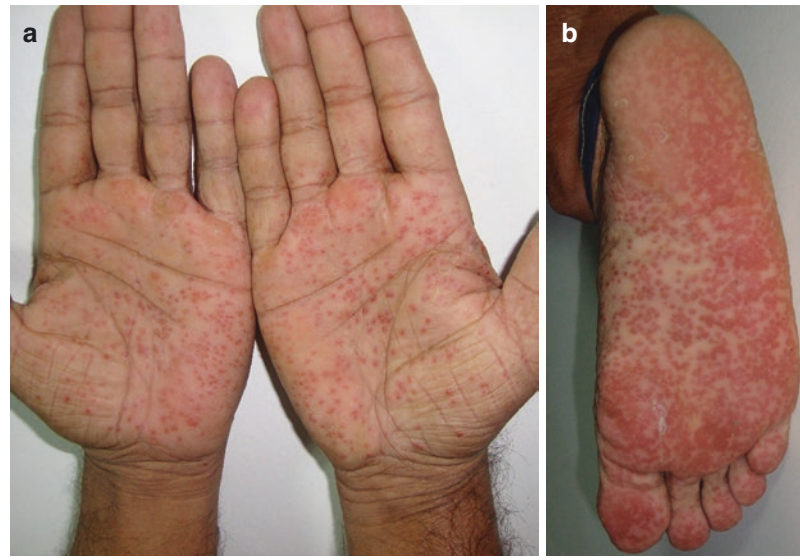


Fig. 16.32 (**a**, **b**) Hand, foot and mouth disease in a 40-year-old man (photographed by Dr. Ranthilaka R. Ranawaka)

16.6 Myxovirus Infections and Related RNA Viruses

16.6.1 Measles/Rubeola

Measles is a highly infectious disease caused by measles virus. Measles infection occurs from human to human via upper respiratory tract. The incidence of measles has markedly decreased since the introduction of the live vaccine in 1963.

Clinical Features Measles classically presents with a prodrome of fever, cough, nasal congestion and rhinoconjunctivitis. The pathognomonic enanthem, Koplik spots and grey-white papules

on the buccal mucosa appear during the prodrome. The exanthem consists of erythematous macules and papules which appear on the hairline and behind the ear and then spread cephalocaudally (Mancini and Shani-Adir 2012).

Complications of measles are otitis, pneumonia, encephalitis, myocarditis and subacute sclerosing panencephalitis.

Differential Diagnosis Other viral infections, drug eruption, Kawasaki disease

Treatment No specific antiviral therapy

16.6.2 Pityriasis Rosea

Pityriasis rosea is an acute self-limiting disease, probably infective in origin, and characterized by a distinctive skin eruption and minimal constitutional symptoms. Although the cause of pityriasis rosea is uncertain, many epidemiological and clinical features suggest that an infective origin most speculation now centres on a viral aetiology.

This affects mainly children and young adults between the ages of 10 and 35 years.

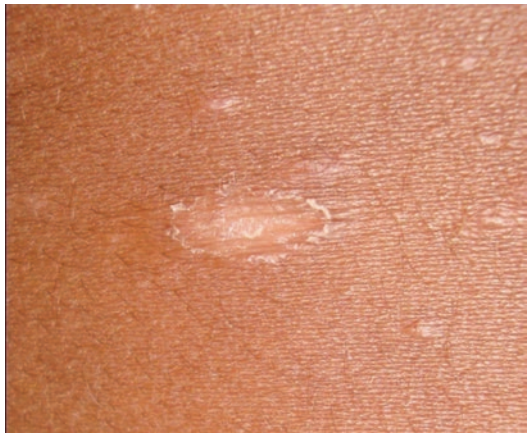


Fig. 16.33 Pityriasis rosea: Note the marginal collarette of scale attached peripherally, with the free edge of the scale internally

Clinical Presentation The first manifestation of the disease is usually the appearance of the herald patch, which is larger and more prominent than the later eruption. In its classical form, the eruption consists of discrete oval lesions, dull pink in colour and covered by fine dry silvery-grey scales. Marginal collarette of scale attached peripherally, with the free edge of the scale internally.

Differential Diagnosis Drug reaction, guttate psoriasis, secondary syphilis

Management This is usually asymptomatic and self-limiting, If itchy, mild topical steroid, UVB phototherapy (Figs. 16.33, 16.34, 16.35, 16.36).

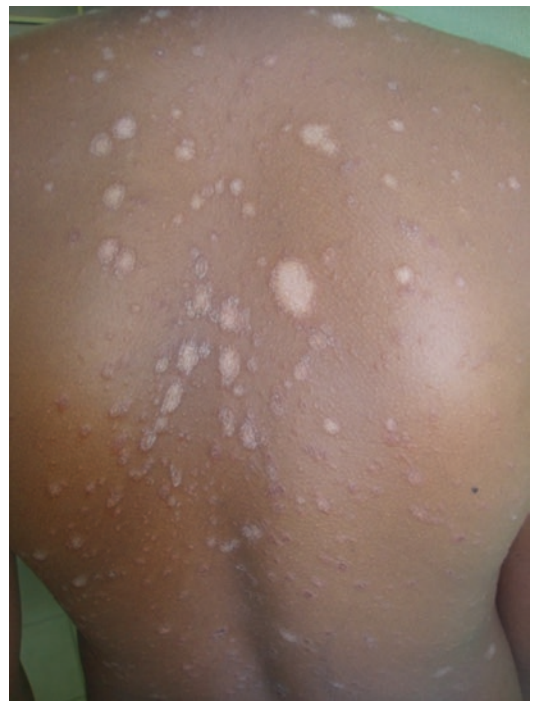


Fig. 16.34 Pityriasis rosea in a 13-year-old boy. Note the herald patch, which is larger and more prominent. It is the first lesion to appear (photographed by Dr. Ranthilaka R. Ranawaka)

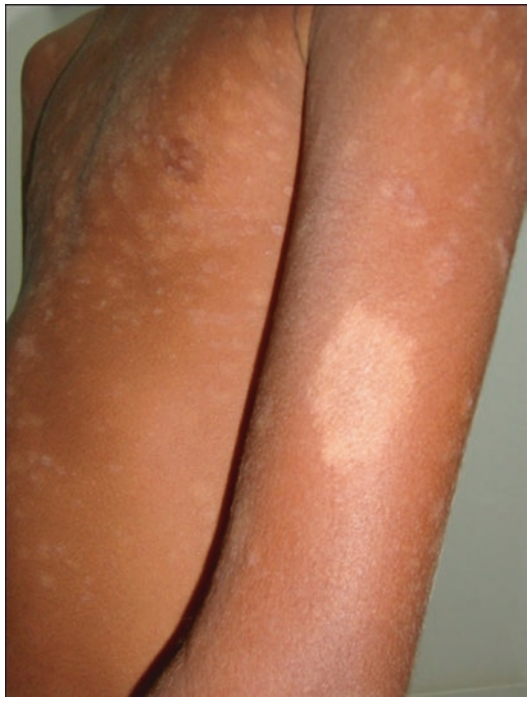


Fig. 16.35 Pityriasis rosea in an 11-year-old boy. Note the herald patch

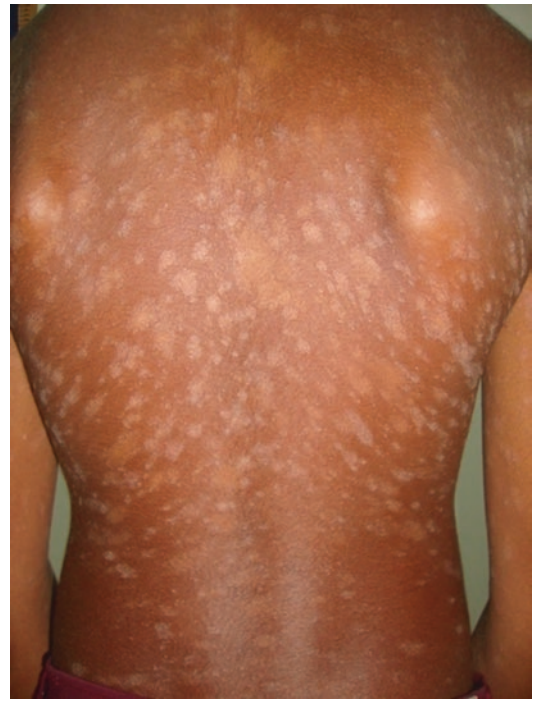


Fig. 16.36 Pityriasis rosea. Christmas tree pattern of eruption is classical in widespread eruption on the back of the trunk (photographed by Dr. Ranthilaka R. Ranawaka)

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