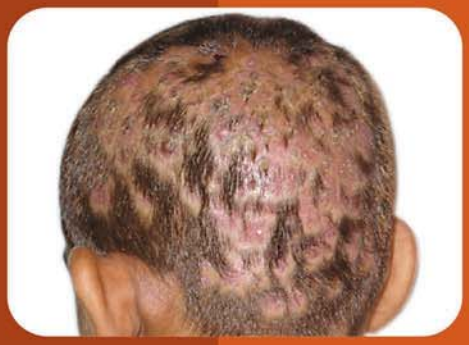

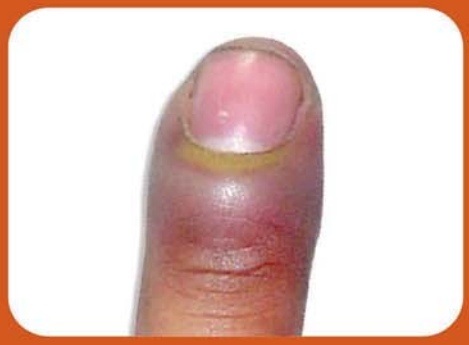

Atlas of Infections in Dermatology

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Foreword
Bhushan Kumar

2nd Edition

JAYPEE

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CHAPTER 8

Pox, Rubella, Coxsackie, and Other Viral Cutaneous Disorders

Introduction

With the advent of the human immunodeficiency virus (HIV) pandemic, ease of global travel, and mass migrations, dermatologists need to be aware regarding the various cutaneous presentations of both deoxyribonucleic acid (DNA) and ribonucleic acid (RNA) viruses. With recent reports of outbreaks of various earlier known zoonotic infections crossing over into humans, such as monkeypox (Mpox) and *buffalopox*, the need to mass educate healthcare providers becomes paramount to identify, manage and control such infections at the earliest. This chapter covers viral cutaneous disorders caused by miscellaneous viruses including pox, rubella, and coxsackie viruses.

Poxviruses

- Poxviruses are brick-shaped (240 × 300 nm) viruses, which are relatively larger.
- They have a complex internal structure including a double-stranded DNA genome (130–260 kb) and associated enzymes.

Smallpox

Smallpox has been eradicated since 1976, following worldwide vaccinations. However, remnants of the disease on the skin can still be seen in patients who suffered before that **(Fig. 1)**. Smallpox is caused by the *Variola virus*; a double-stranded deoxyribonucleic acid (dsDNA) linear enveloped virus that has been divided into two major groups:

1. *Variola major*: The severe and most common form of smallpox, which caused an extensive rash and high fever.
2. *Variola minor*

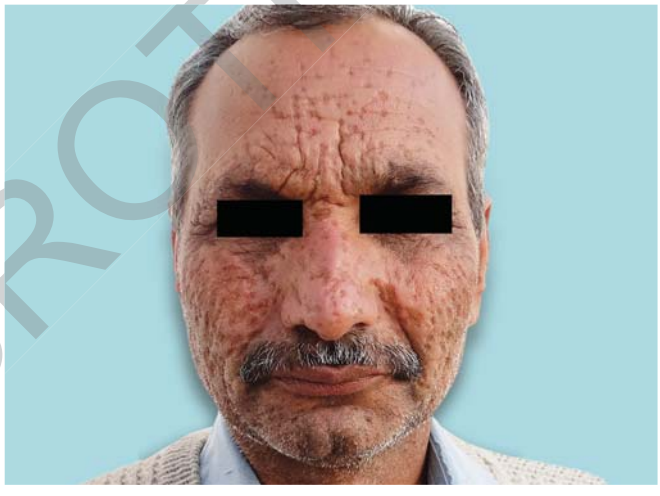


FIG. 1: Disfiguring well-defined hyperpigmented pitted scars (pockmarks) on the face of a patient post-smallpox. On the face due to larger and numerous sebaceous glands, scarring is the most prominent.

The disease transmission used to occur via respiratory droplets from an infected individual. It was seen more in winter and early spring (low humidity and temperature). Very young, elderly, and pregnant women used to develop severe infections.

Clinical Features

- High fever, myalgia, and severe headache develop within an average of 7–17 days of exposure.
- A rash (enanthem) emerged first as small red spots on the tongue, mouth, and oropharynx and broke open to discharge large amounts of the virus into the mouth and throat. At this time, the person was most contagious.
- Then rash (exanthem) appeared on the skin, starting on the face and spreading to the arms and legs and then to the hands and feet (centrifugal distribution),

within 24 hours. As the rash appeared, the fever usually resolved.
- By the third day of disease, the rash transformed into papular lesions that subsequently became varioliform. The palms and soles were frequently affected.
- By the end of 1 week, the scabs formed and were shed off leaving behind pockmarks as a disfiguring sequel of the infection **(Fig. 1)**.

Differential Diagnosis

- Varicella
- Disseminated herpes zoster
- Impetigo
- Erythema multiforme
- Secondary syphilis
- Molluscum contagiosum

Diagnosis

Characteristic intracytoplasmic Guarnieri bodies may be seen apart from ballooning degeneration of the epidermis. Papillary dermal edema and perivascular lymphohistiocytic infiltrate may be seen in histopathology. Electron microscopy and polymerase chain reaction (PCR) also can be done.

Treatment

Treatment is mainly supportive. Maintenance of general hygiene, management of fever, body ache, calamine/bland emollient application over lesions, and oral antihistamines.

Mpox (Monkeypox)

The Monkeypox virus is a zoonotic orthopoxvirus, incidentally causing disease in humans, which is similar to but much milder than smallpox. The first confirmed human case was in 1970 from a child in the Democratic Republic of Congo. The virus was endemic to western and central Africa, with outbreaks in Europe related to exotic pet trade and international travel. However, recent outbreaks have been reported with sexual transmission, more commonly in men having sex with men (MSM) population. Earlier coincidental immunity to the Mpox virus achieved with vaccinia vaccination has been ebbing with its discontinuation after the eradication of smallpox, making Mpox clinically relevant.

Transmission

- Mpox is a zoonosis, spreading from animals to humans. Animal reservoirs include squirrels, rats, monkeys, primates, prairie dogs, hedgehogs, pigs, and mice.
- Ongoing epidemic (since 2022) is driven by human-to-human transmission through respiratory droplets, fomites, and direct contact with lesions.
- Sexual transmission is a major route, with a presentation involving predominantly genital lesions. The ongoing outbreak may have a newly emerging clade.

Clinical Features

- Initial symptoms include fever, headache, myalgia, or fatigue.
- Lymphadenopathy is present, which helps differentiate it from smallpox.
- Mucosal lesions develop after 1–2 days **(Fig. 2)**.
- These are followed by cutaneous lesions involving the face **(Fig. 3)**, neck and extremities **(Fig. 4)**, palms, and

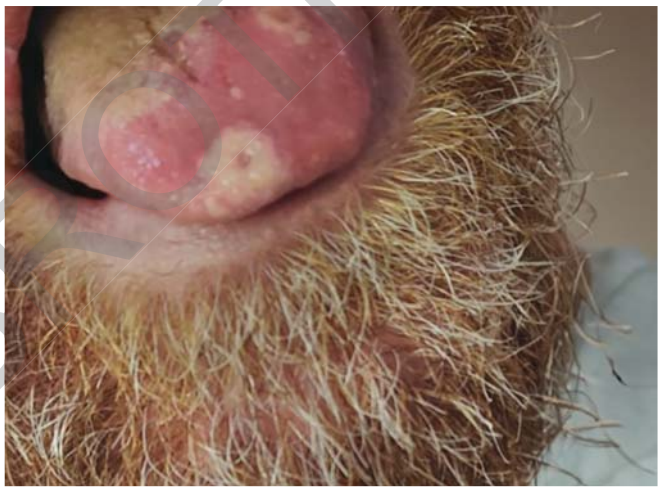


FIG. 2: Oral mucosal lesions in a patient with Mpox.
Courtesy: Dr Rajasekhar Reddy Rangareddy MD (DVL), Specialist Dermatologist, Lifecare Hospital, Abu Dhabi, UAE.

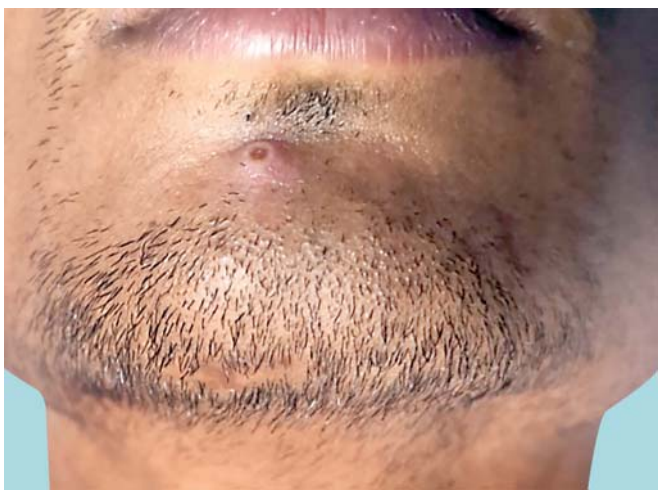


FIG. 3: Facial lesions in a patient.
Courtesy: Dr Rajasekhar Reddy Rangareddy MD (DVL), Specialist Dermatologist, Lifecare Hospital, Abu Dhabi, UAE.

soles **(Fig. 5)**, and are centrifugal in distribution. They may or may not spread.
- The number of lesions is also highly variable **(Fig. 6)**, and they keep evolving over 2–4 weeks in 1–2-day increments.
- Initial macular lesions evolve through papular, vesicular, and pustular phases, synchronously **(Fig. 7)**.
- They tend to be firm, deep-seated, and 2–10 mm in size.
- The pustular phase lasts for 5–7 days before crusting and desquamation over 7–14 days, rendering the patient non-infectious over 3–4 weeks **(Fig. 7)**.
- Umbilicated and pseudopustular lesions are classical **(Fig. 8)**.
- Genital **(Figs. 9 and 10)** and perianal area **(Fig. 11)** are commonly involved with lesion clustering and lymphadenopathy. The current epidemic is especially presenting with lesions in the genital area, especially in MSM.

Differential Diagnosis

- Smallpox and generalized vaccinia
- Disseminated zoster and chickenpox
- Eczema herpeticum and disseminated herpes simplex
- Syphilis and yaws
- Scabies
- Rickettsialpox

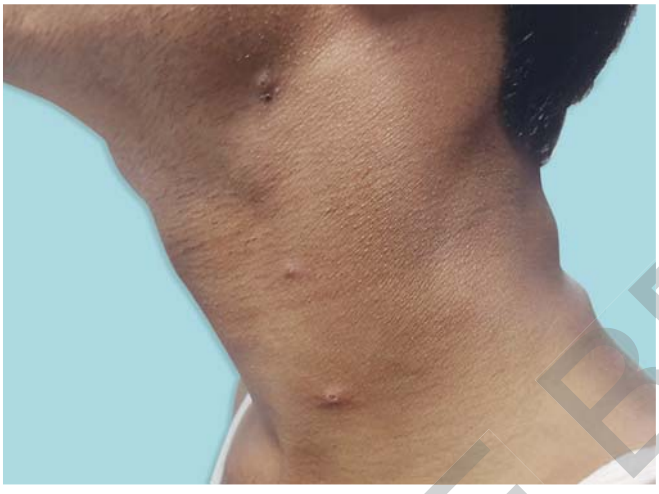


FIG. 4: Neck lesions, scarcely distributed.
Courtesy: Dr Rajasekhar Reddy Rangareddy MD (DVL), Specialist Dermatologist, Lifecare Hospital, Abu Dhabi, UAE.

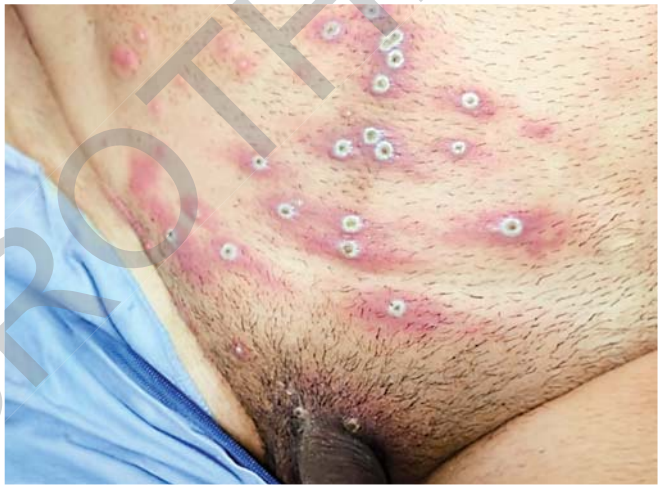


FIG. 6: A large number of lesions in an immunosuppressed patient with Mpox.
Courtesy: Dr Rajasekhar Reddy Rangareddy MD (DVL), Specialist Dermatologist, Lifecare Hospital, Abu Dhabi, UAE.

FIG. 5: Palmar lesions in a patient with Mpox.
Courtesy: Dr Rajasekhar Reddy Rangareddy MD (DVL), Specialist Dermatologist, Lifecare Hospital, Abu Dhabi, UAE.

FIG. 7: Typical umbilicated and pseudopustular lesions progressing to crusting and desquamation.
Courtesy: Dr Rajasekhar Reddy Rangareddy MD (DVL), Specialist Dermatologist, Lifecare Hospital, Abu Dhabi, UAE.

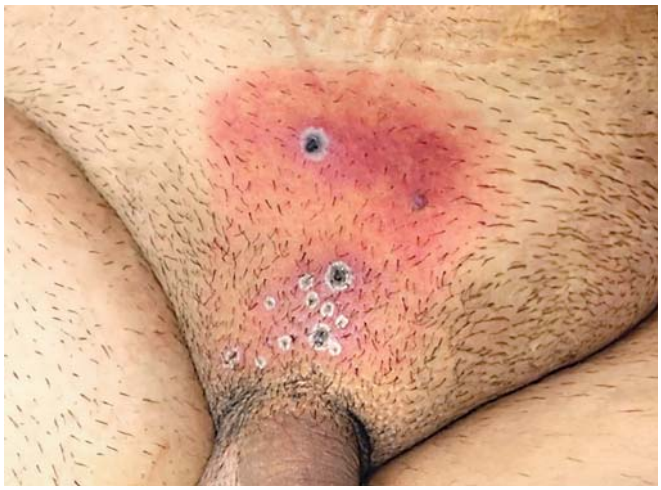


FIG. 8: Umbilicated lesions with peripheral erythema and significant inflammation, involving the genital area.
Courtesy: Dr Rajasekhar Reddy Rangareddy MD (DVL), Specialist Dermatologist, Lifecare Hospital, Abu Dhabi, UAE.

FIG. 10: Reactive edema due to multiple lesions in the same patient.
Courtesy: Dr Rajasekhar Reddy Rangareddy MD (DVL), Specialist Dermatologist, Lifecare Hospital, Abu Dhabi, UAE)

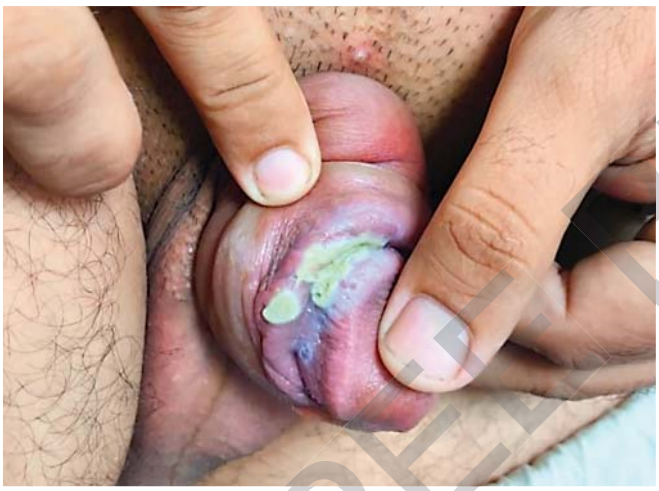


FIG. 9: Mpox lesions involving the coronal sulcus.
Courtesy: Dr Rajasekhar Reddy Rangareddy MD (DVL), Specialist Dermatologist, Lifecare Hospital, Abu Dhabi, UAE.

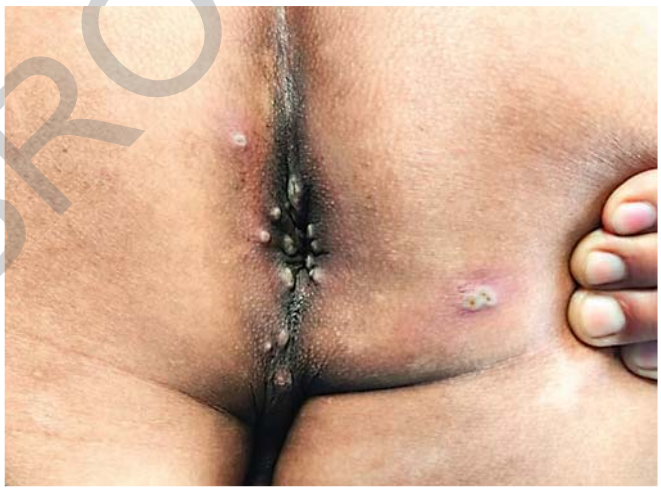


FIG. 11: Perianal lesions in a patient with Mpox.
Courtesy: Dr Rajasekhar Reddy Rangareddy MD (DVL), Specialist Dermatologist, Lifecare Hospital, Abu Dhabi, UAE.

- Measles
- Bacterial skin infections
- Drug rash

Diagnosis

The "Acute, Generalized Vesicular or Pustular Rash Illness Protocol" by the Centers for Disease Control and Prevention (CDC) is followed. Lymphadenopathy is a required primary criterion to determine the need for testing.

- Testing for the presence of *Orthopoxvirus* by electron microscopy, immunohistochemical staining for orthopoxvirus antigens, or serology for anti-orthopoxvirus antibodies can be sufficiently diagnostic if the clinical setting suggests so.
- Confirmation is based on isolation in viral culture or by PCR for Mpox virus DNA.

Complications

These include bacterial superinfection of lesions, scarring, loss of vision (corneal scarring), pneumonia, dehydration (poor oral intake or fluid loss due to lesions), sepsis, encephalitis, and death.

Management

- Treatment is largely supportive
- No specific antivirals exist. In severe cases, investigational use of drugs with benefits against

orthopoxviruses in animal studies or severe vaccinia vaccine complications, can be considered. These include

- *Brincidofovir*: Oral DNA polymerase inhibitor. It is approved for the treatment of smallpox. Normal saline and probenecid should be given concurrently.
- *Tecovirimat*: Oral intracellular viral release inhibitor. It inhibits viral envelope protein VP37, thus blocking viral maturation and release from infected cells.
- *Intravenous vaccinia immunoglobulin (VIG)*: It is licensed by the Food and Drug Administration (FDA) to treat complications of vaccinia vaccination.

Prevention and Postexposure Prophylaxis

- Infected individuals should remain in isolation, wear a surgical mask, and keep lesions covered till crusts fall off and epithelialization occurs.
- For individuals exposed to the virus, temperature and symptoms should be monitored twice daily for 21 days (upper limit of Mpox incubation period). Isolation is not needed as infectiousness starts with symptom onset.
- *Ankara vaccine*: It is replication-defective modified vaccinia (two shots, 4 weeks apart) and has a superior safety profile compared to first and second-generation smallpox vaccines. It does not create a skin lesion or pose a risk of spread. Postexposure vaccination is recommended, especially for high-risk exposure, defined as contact with broken skin or mucous membranes, infected patient's body fluids, respiratory droplets, or scabs. Vaccination within 4 days of exposure may prevent disease onset, and within 14 days may reduce disease severity.

Camelpox

Camelpox is an infectious and economically important contagious skin disease of camelids (members of the biological family Camelidae) caused by the *Camelpox virus* (CMLV). CMLV outbreaks have been reported from India, Pakistan, Middle East countries, Afghanistan, and North-East Africa **(Fig. 12)**. The incubation period of CMLV varies from 3 to 15 days.

Clinical Features

- In humans, lesions are mainly noticed on the hands.
- Initial signs include fever, itching with slight pain, and erythema at the affected site.

FIG. 12: Lesions of camelpox on a camel, multiple ulcerated papulovesicular lesions on hindquarters of a camel.

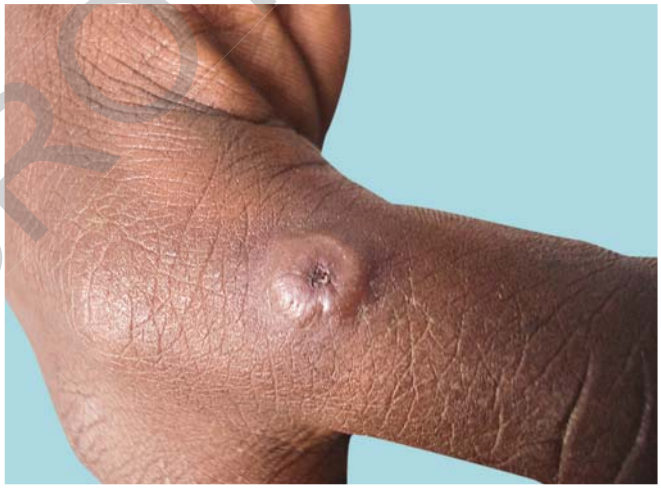


FIG. 13: A papulovesicular lesion on the finger showing central crusting, peripheral edema, and erythema.

- Local edema and vesicle may appear 4–7 days after infection **(Fig. 13)**.
- About 7–10 days later, these vesicles may rupture leaving a deep ulcerated wound surrounded by a red zone of inflammation.
- Subsequently, 2 weeks after the infection, lesions gradually heal by the development of crusts which later slough leaving a central residual scar.

Buffalopox

It was first described in 1934 in India. The causative agent *Buffalopox virus* (BPXV) was first isolated in 1967. BPXV is associated with sporadic outbreaks in Asian buffalos and cows in India, Pakistan, Bangladesh, Russia,

Indonesia, and Egypt. The incubation time in animals is about 2–4 days and in humans, it is 3–19 days. Humans can become infected by contact with diseased animals and infections are mainly noticed in animal attendees, milkers, and veterinarians.

Clinical Features

The symptoms are comparable to those of infections with cowpox virus (CPXV).

- In humans, the lesions are mostly confined to the hand, wrist, finger, face, forehead, buttock, and legs.
- Constitutional symptoms such as fever, axillary lymphadenopathy, general malaise, and an erythematous rash over the chest and abdomen may also be noted.
- Lesions are characterized by progressive stages of macules, papules, vesicles, and pustules **(Figs. 14 and 15)**. Lesions may even involve eyes **(Fig. 16)**. The disease is self-limiting, and lesions may heal within 3–6 weeks leaving a black eschar at the site of lesion.

Diagnosis

- It is mostly based on characteristic clinical features in appropriate settings.
- Swab and serum samples from cattle and humans can be subjected to viral isolation, cell culture, and plaque reduction neutralization test
- PCR may be undertaken.
- *Histopathology*: From the maculopapular stage, stratum malpighii shows multilocular vesicles due to cell vacuolization. Cytoplasmic eosinophilic inclusion bodies are seen. Reticulated vesicles and dilated capillaries, along with necrosis and dense inflammatory infiltrate are also seen **(Figs. 17 and 18)**.

Treatment

- No definite treatment is available.
- Washing the infected lesions with 1% potassium permanganate followed by topical broad-spectrum antibiotics is advisable.
- If symptoms are severe, oral antibiotics and paracetamol are advisable.

Prevention

Isolation of affected animals and wearing of gloves by animal handlers during washing or caring for infected animals is recommended.

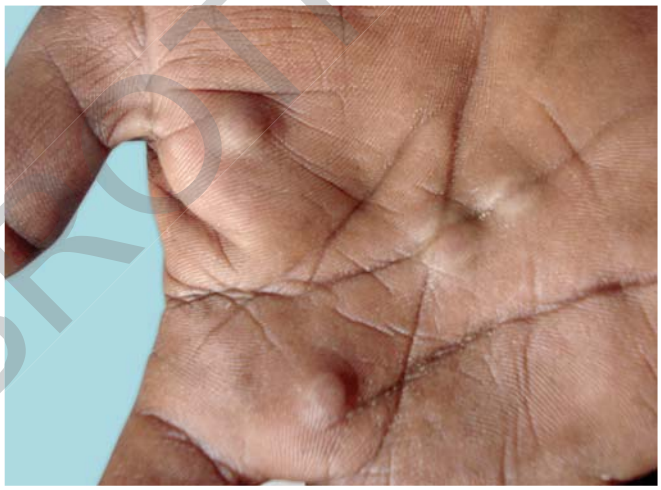


FIG. 15: Lesions on palms of the same patient, a relatively unusual site without a history of prior trauma.

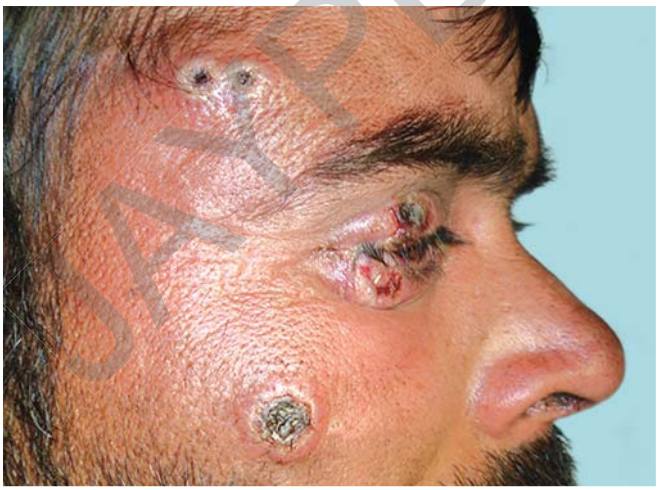


FIG. 14: Multiple well-defined centrally crusted papulovesicular lesions of buffalopox on the face of an infected individual with peripheral edema. Note the lesions on the lid margins.

FIG. 16: Conjunctival involvement of the right eye with a lesion on the lower lid margin of the left eye.

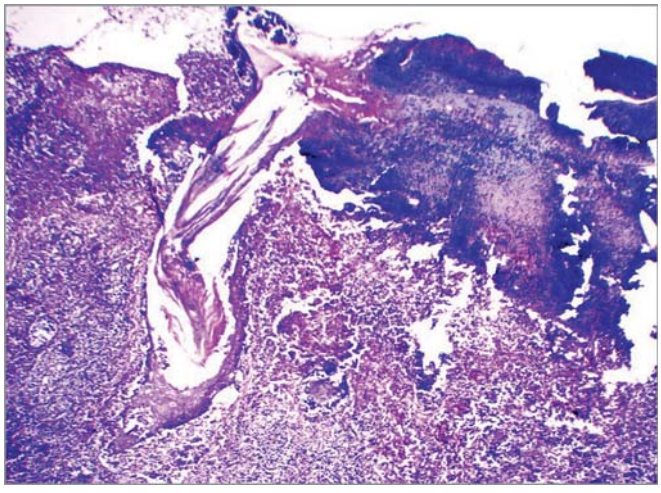
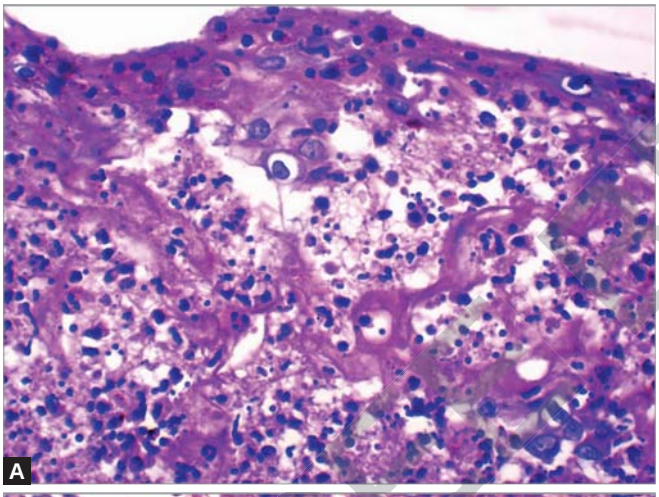
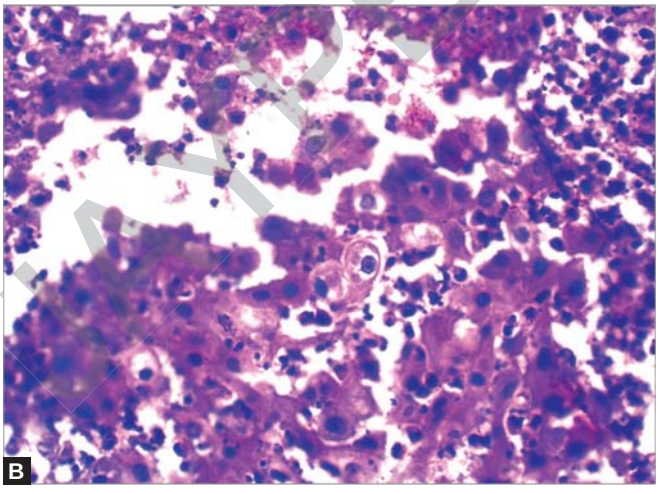


FIG. 17: Hematoxylin and eosin (H&E) (4 × 4) histopathological image of target stage section showing crusting, central hemorrhage, tissue necrosis, and edema. Inflammatory infiltrate in epidermis and dermis.





FIGS. 18A AND B: Hematoxylin and eosin (H&E) (4 × 10) magnification view showing Reticulated vesicles (a characteristic feature of viral infection of the epidermis), mixed infiltrate, and eosinophilic inclusion bodies in the cytoplasm of vacuolated epidermal cells.

Orf

It is caused by the dsDNA virus. It is also known as *contagious* ecthyma, sore mouth, or scabby mouth in sheep and goats. It has been reported from most sheep- or goat-raising areas including those in Europe, the Middle East, USA, Africa, Asia, Alaska, South America, Canada, New Zealand, and Australia.

Infection in humans occurs by direct contact or through fomites during contact with infected animals. It is more frequent among milkers, veterinarians, butchers, and people who come in contact with infected animals.

Clinical Features

- At 3–7 days, solitary papules are formed, usually on the hands.
- These are followed by vesicles, and finally wart-like nodules, which subside after 4–6 weeks with little or no scarring.
- The vesicular stage has a characteristic "target" appearance with a red center, white ring, and red halo which progresses to a weeping nodule **(Fig. 19)**.
- The nodule eventually dries creating small black dots on the surface and as it heals, papillomas may develop over the lesion surface.
- Symptoms such as pain, fever, lymphangitis, and erythema have been observed.
- Generalized disease and large progressive lesions ("giant" orf) have been reported in immunocompromised individuals.

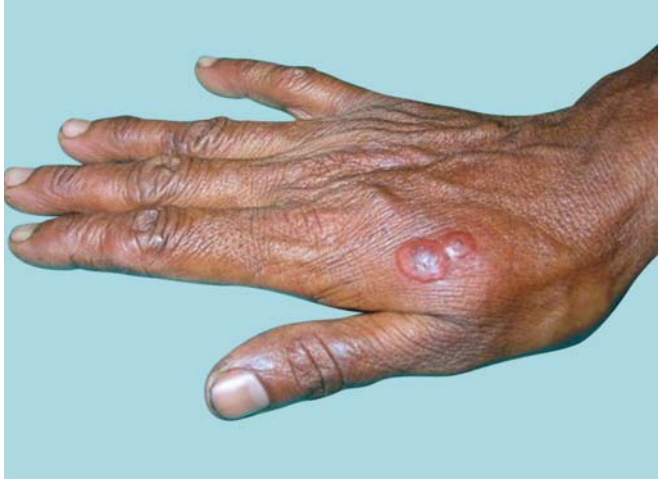


FIG. 19: Lesions of orf on the dorsum of right hand showing well-circumscribed nodular lesions with central dusky center and peripheral erythema and edema.

Diagnosis

The clinical signs are often typical. Laboratory diagnosis by electron microscopy, histology, and PCR offers the most accurate approach.

Treatment

- Moist dressings, local antiseptics, and finger immobilization are done for the lesions.
- Antibiotics, both topical and systemic to prevent bacterial superinfection.
- Topical imiquimod and cidofovir cream have also been used, successfully.

Prevention

The best preventive measure for animals is orf vaccination in every 6–8 months.

Milker's Nodule/Pseudocowpox/ Paravaccinia

Milker's nodule is caused by the *paravaccinia* virus. It is a localized benign cutaneous eruption known to occur in milkers, farm workers, butchers, and veterinary students, particularly new to their occupation with no previous immunity. It is prevalent worldwide, including in India. Transmission of disease within the cattle herds occurs by direct and indirect contact including calf suckling of multiple cows, flies, and milking equipment. The incubation period varies between 5 and 15 days.

Clinical Features

- A well-characterized clinical course progresses through the stages of macules, papules, vesicles, pustules, and scabs, lasting approximately 10–15 days.
- Skin lesions become opaque and grayish with small crusts and central depression.
- Some patients may develop lymphangitis or erythema multiforme secondary to the infection.
- Clinical signs spontaneously resolve in 6–8 weeks, leaving no scars.

Differential Diagnosis

- CPXV
- Herpes virus infection
- *Mycobacterium marinum* infection
- Orf
- Pyogenic granuloma
- Occasionally, syphilis and sporotrichosis

Diagnosis

In addition to typical clinical settings, viral culture, DNA hybridization, and PCR can be done for confirmation of diagnosis. The "pan-pox" assay (PCR assay) can be used for screening and diagnosis of human and animal poxvirus infections.

Treatment

Antiviral medications are not effective, only symptomatic treatment is given.

Prevention

Isolation of infected animals and wearing protective gear by their caretakers.

Molluscum Contagiosum

Molluscum contagiosum (MC) is caused by the dsDNA virus; *Molluscipoxvirus* (MCV). MCV commonly infects keratinized skin and infundibular portion of the hair follicle and mucosae, generating the characteristic Henderson–Patterson (HP) cytoplasmic inclusion bodies. The virus entry is facilitated by mechanical trauma to the skin and the incubation period is 2–7 weeks.

Clinical Features

The MCV affects three distinct populations: children, sexually active adults, and immunocompromised individuals.

- In children, the characteristic skin lesions are single or, more often, multiple, rounded, dome-shaped, pink, waxy papules that are 2–5 mm in diameter. The papules are umbilicated and contain a caseous plug **(Fig. 20)**. The common sites involved are the face, extremities, trunk, and chest.
- In immunocompetent adults, molluscum contagiosum most commonly is a sexually transmitted disease. Healthy adults tend to have few typical lesions, limited to the perineum, genitalia, lower abdomen, or buttocks **(Fig. 21)**.
- MC lesions in HIV-positive individuals, or immunosuppressed patients, are numerous, more widespread, larger (giant > 10 mm in diameter), and resistant to conventional therapy. The lesions most often involve the face, neck, and trunk. Disease states associated with widespread MC lesions include sarcoidosis, use of immunosuppressants, topical steroids, and also topical calcineurin inhibitors, suggesting a role of cell-mediated immunity.

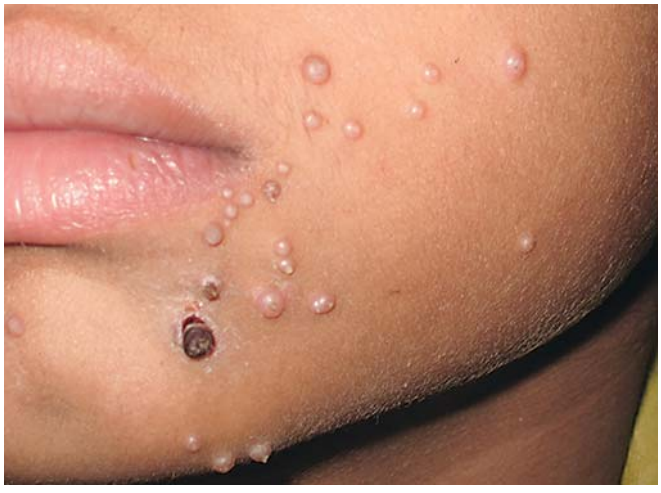


FIG. 20: Pearly white and umbilicated papular lesions of molluscum contagiosum on the face and back of a child.

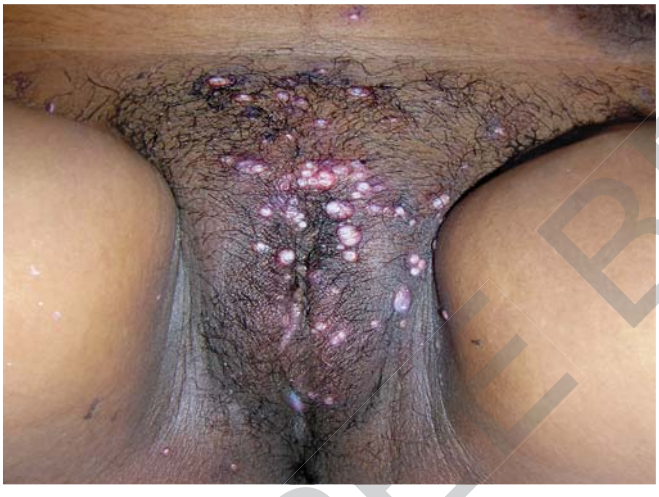


FIG. 21: Numerous papulonodular lesions of molluscum contagiosum (MC) on the vulva showing characteristic central umbilication and inflammation at places.

- Molluscum dermatitis refers to the erythema and eczematous changes occurring around the molluscum lesions. It is often seen in children with atopy.

Differential Diagnosis

- Cryptococcosis
- Histoplasmosis
- Coccidioidomycosis
- *Penicillium marneffei* infection
- Epitheliomas
- Basal cell carcinoma
- Sebaceous hyperplasia

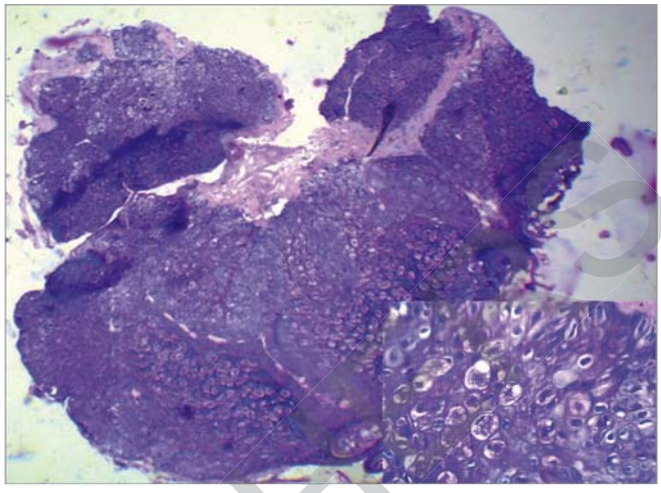


FIG. 22: Histopathological examination showing acanthosis, down the growth of infected epidermal cells having large eosinophilic intracytoplasmic inclusion bodies [Henderson–Paterson (HP) bodies] (H&E 40×). Inset: Close up higher magnification view of the HP bodies (H&E 100×).
(H&E: Hematoxylin and eosin)

Diagnosis

- Diagnosis of MC is mainly clinical.
- However, in atypical cases, squash preparation can be made. It involves a microscopic examination of cellular exudates prepared by manually extracting the cellular material from the lesion, crushing it between two glass slides, and staining with Giemsa.
- *Histopathology* reveals epidermal acanthosis with shells containing intracytoplasmic inclusion bodies (molluscum bodies) **(Fig. 22)**.
- Molecular diagnostics by in situ hybridization and PCR and MCV enzyme-linked immunosorbent assay (ELISA) are additional tools used for unclear cases.

Treatment

Molluscum contagiosum can be treated with various modalities that are discussed in **Table 1**.

Measles

Measles is caused by the single-stranded ribonucleic acid (ssRNA) virus of the genus *Morbillivirus* and family Paramyxoviridae. It is a common disease, for which the first live attenuated vaccine was licensed in the United States in 1963. In India, the measles, mumps, and rubella (MMR) vaccine (a live attenuated strain of Edmonston–Zagreb measles virus propagated on human diploid cell culture) is a part of the universal immunization program. The average incubation period is 10 days.

TABLE 1: Therapeutic modalities for molluscum contagiosum.

Destructive therapy	Curettage, trichloroacetic acid, 10% KOH application, cryotherapy, and CO_2 laser
Cytotoxic agents	Podophyllotoxin and 5-fluorouracil
Antiviral agents	Topical and systemic cidofovir and intralesional interferon-α
Immunomodulator	Topical imiquimod
Others	Photodynamic therapy, electron beam therapy, introduction of HAART can normally elicit resolution in HIV/AIDS patient

(AIDS: acquired immunodeficiency syndrome; HAART: highly active antiretroviral therapy; HIV: human immunodeficiency virus; KOH: potassium hydroxide)

Clinical Features

- There is a prodrome of high fever (often > 104°F), conjunctival congestion, and Koplik's spots, which appear as bluish-gray specks or "grains of sand" on a red base, on the buccal mucosa opposite the second molars.
- Fever typically lasts 4–7 days followed by a classic triad of conjunctivitis, cough, and coryza (the "3 Cs").
- This is followed by the appearance of a maculopapular rash that lasts 3–5 days. It is generalized, maculopapular, erythematous, and starts on the back of the ears. After a few hours, it spreads to the head and neck region and then covers the entire body, often associated with itching **(Figs. 23 and 24)**.
- Immunocompromised patients may not develop a rash.
- The entire course of uncomplicated measles lasts for 7–10 days. Natural immunity is then known to last as long as 65 years.

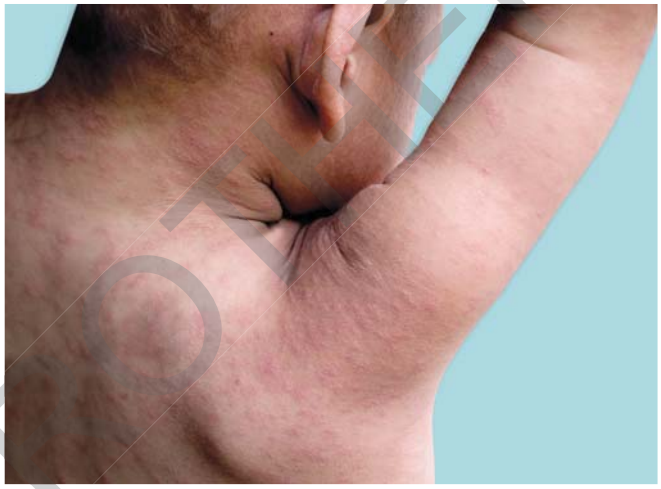


FIG. 23: A typical rash of measles in a child. The rash is typical maculopapular erythematous (morbilliform) in nature.

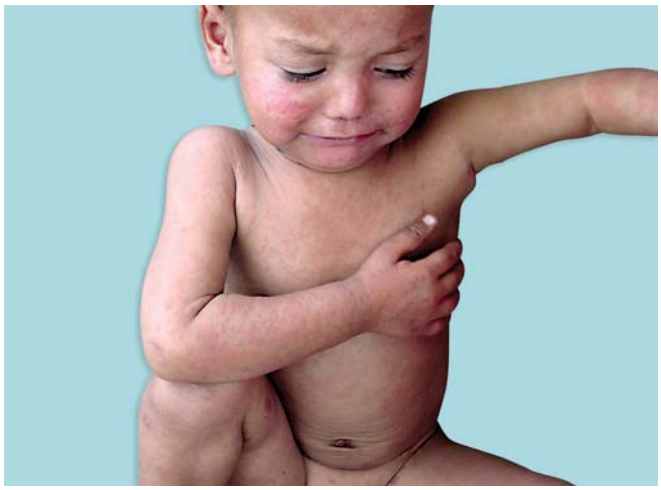


FIG. 24: The same patient with a generalized rash.

Complications

These include diarrhea, pneumonia, acute measles panencephalitis, subacute measles encephalitis, subacute sclerosing panencephalitis, and corneal ulceration. Case fatality rates are higher in malnourished individuals, vitamin A deficiency, pregnancy, and immunocompromised states.

Diagnosis

Clinical diagnosis may be aided by:

- *Microscopy of nasopharyngeal secretions*: It may reveal multinucleate giant cells with inclusion bodies, pathognomonic for measles.
- *Immunofluorescence*: Direct immunofluorescence (DIF) and indirect immunofluorescence (IIF).
- *Virus isolation*: It can be done from the throat or conjunctival washings, sputum, urinary sediment cells, and lymphocytes.
- *Serology*: A fourfold rise between the acute and the convalescent phase, in the measles-specific immunoglobulin M (IgM) antibodies, is diagnostic and is a commonly performed test.

Treatment

Most of the patients with uncomplicated measles will recover with rest and supportive treatment.

Prevention

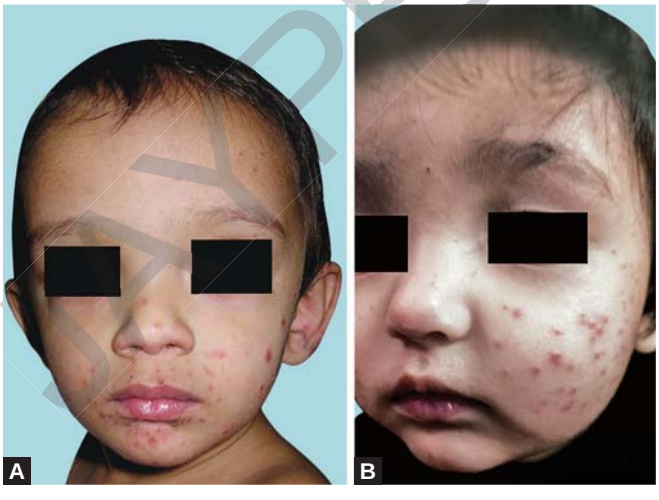
The best way to avoid contracting measles is to have the MMR vaccine. A patient hospitalized with measles should be isolated.

Hand, Foot, and Mouth Disease

It is a viral infection occurring as outbreaks mostly in spring and summer. In India, disease activity has been reported from different parts since 2004. It is a distinctive eruption of children caused by *Coxsackievirus* A16 and *Enterovirus* 71. It is highly contagious, and the route of spread is feco-oral, oral, and contact with skin lesions.

Clinical Features

- Initial prodrome consists of sore throat, dysphagia, mild fever, and abdominal pain.
- The enanthem consists of papules on the tongue and buccal mucosa which break down to form ulcers with an erythematous base.
- This is followed by exanthema with the sudden appearance of erythematous papulovesicular eruptions in crops. These are initially filled with clear fluid which rapidly turns turbid.
- The lesions are 2–10 mm in size with characteristic perilesional erythema appearing on the face, sides and dorsal surfaces of hands and feet, and perineum **(Figs. 25 and 26)**.
- Mucosal involvement is also known **(Figs. 27A to C)**.
- Palms, soles, proximal extremities, and trunk may also be involved.



FIGS. 25A AND B: Lesions of hand, foot and mouth disease (HFMD) on the face of children.

FIG. 26: Lesions on palms and soles of the child with hand, foot and mouth disease (HFMD).

- Lesions heal within 7–10 days without any major complications.
- Rare but fatal complications include cardiac and neurological complications, such as encephalitis, aseptic meningitis, and acute flaccid paralysis.
- Orange-yellow nail discoloration, Beau's lines, and onychomadesis may be seen in one-third to one-fourth of children 3–6 weeks after the lesions have subsided **(Fig. 28)**.

Differential Diagnosis

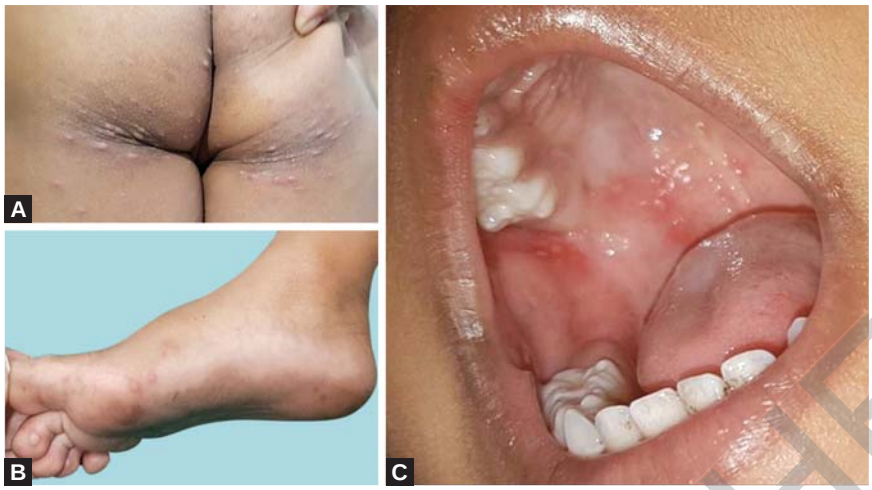
- Herpangina (lesions being more on the soft palate and tonsils here)
- Varicella
- Herpes stomatitis
- Drug eruptions
- Erythema multiforme
- Aphthous ulcers

Diagnosis

Mainly clinical but PCR is currently the diagnostic method of choice.

Treatment

There is neither an effective antiviral therapy nor a vaccine against HFMD. Good hygiene with proper cleaning of hands, safe drinking water, and avoiding direct contact with patients is of paramount importance.



FIGS. 27A TO C: Lesions on the buttocks, sole, and hard palate in a child with hand, foot and mouth disease (HFMD).

FIG 28: Orange to greenish discoloration of the nail plate, onychomadesis, and Beau's lines post hand, foot and mouth disease (HFMD).

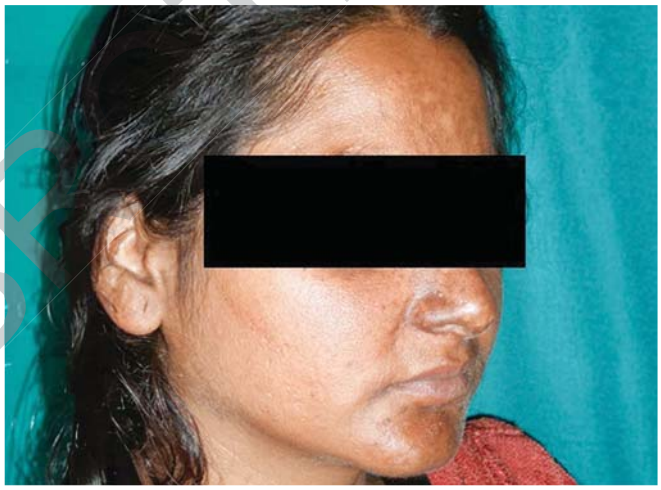


FIG. 29: Melasma-like pigmentation in a patient of chikungunya.

Chikungunya Virus

It was first isolated in India from Kolkata in 1963, followed by an outbreak in 1971 which disappeared in a few years. Recently, there has been a resurgence of this virus. The vector involved in the transmission of this virus is *Aedes aegypti*. Currently, it is an endemic disease in India, Africa, and South-East Asia. The incubation period is 2–12 days.

Clinical Features

- Acute phase manifests as high-grade fever with chills which lasts for 2–3 days. The fever remits for 1–2 days and then reappears.
- Severe polyarticular, migratory arthritis may occur predominantly affecting the small joints of the hand, wrist, ankle, and feet.
- Associated systemic features include nausea/vomiting, fatigue, headache, photophobia, or retro-orbital pain.
- Maculopapular skin rash affecting trunk and limbs manifests in over 50% of the cases. Pruritus occurs in 20–85% of cases. Palmoplantar peeling may occur in a few cases. Oral mucosa may be involved in the form of aphthous ulcers and gingivitis.
- Facial pigmentation may occur in various patterns, including malar, midfacial, and flagellate in up to 30% of cases with characteristic involvement of the nose (Chik sign) **(Figs. 29 and 30)**.
- Various unusual features include urticarial and purpuric rash, photosensitivity, edema of feet and legs, scrotal dermatitis, erythema multiforme-like lesions, diffuse pigmentation, etc.

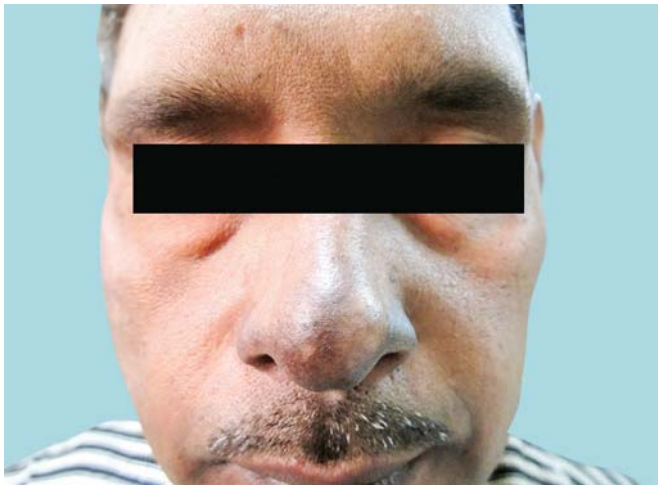


FIG. 30: Residual pigmentation of the nose in a patient of chikungunya (Chik sign).

Differential Diagnosis

- Dengue and dengue hemorrhagic fever
- Rubella
- Parvovirus

Diagnosis

Viremia is present in most patients during the first 48 hours of the disease. The antibodies appear with the cessation of viremia. Virus-specific IgM antibodies are detected by captured ELISA in patients recovering from infection. Virus isolation may also be done.

Treatment

The management is mainly symptomatic and supportive. Aspirin must be avoided in the acute phase.

Dengue

Dengue is perhaps the most important mosquito-borne viral disease in humans, affecting 50–100 million people annually worldwide. The principal vector is *Aedes aegypti*. Humans and mosquitoes both act as reservoirs of the virus. A temperature of >20°C and stagnant water, especially in artificial containers favor epidemic transmission. The incubation period ranges from 3 to 10 days.

Clinical Features

Dengue Fever

The patient presents with high-grade fever and chills, intense headache, and characteristic severe muscle and joint pains (bone-breaking fever). Fever remits for a few hours to 2 days to appear again (biphasic curve) and lasts for 5–7 days.

- A transient, generalized, blanchable macular rash may be seen during the first 24–48 hours of fever.
- A second skin eruption appears during the second febrile phase (in 80%) and lasts for 2 hours to several days.
- The lesions are maculopapular/morbilliform in approximately 50% of cases which appear confluent with the sparing of islands of normal skin sometimes referred to as "white islands in a sea of red."
- It may start on the legs and spread caudally or on the chest and trunk and spread to limbs. The rash is usually asymptomatic or mildly itchy. Facial flushing and hemorrhagic crusting of the lips may be seen in a few cases.
- Unusual clinical features include exfoliative dermatitis (palmoplantar), lymphadenopathy, jaundice, and encephalitis.

Dengue Shock Syndrome and Hemorrhagic Fever

Mucocutaneous features are present in approximately one-third of patients.

- Ecchymosis and petechiae are present in 30% and 13% of patients, respectively **(Fig. 31)**.
- Circumoral cyanosis is another characteristic feature.
- Conjunctival injection and hemorrhagic crusting of lips may be present.
- Hemorrhagic manifestations generally appear 4–5 days after onset of fever and include bleeding phenomenon (epistaxis, petechiae, and gingival bleeding) and rarely menorrhagia and gastrointestinal bleeding.

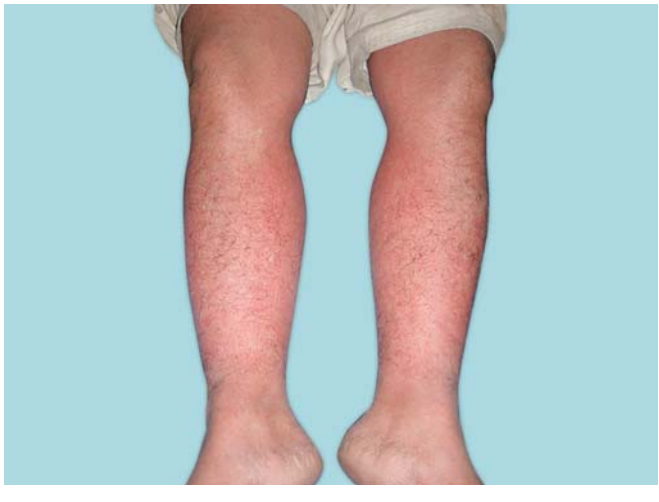


FIG. 31: Petechiae involving bilateral legs in a patient with dengue hemorrhagic fever.

Differential Diagnosis

- Chikungunya fever
- Measles
- Rubella
- Erythema infectiosum
- Exanthem subitum

Diagnosis

It is based on IgM antibody detection, which appears within 2–5 days of onset of illness and persists for 1–3 months. Nonstructural protein-1 (NS-1) antigen detection method is also available.

Treatment

Treatment is mainly symptomatic. Avoidance of aspirin is important. Dengue hemorrhagic fever and dengue shock syndrome are managed with appropriate intravenous fluids and oxygen along with close monitoring of vitals and laboratory parameters.

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