Vesicular rash

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Vesicles are circumscribed, fluid-filled epidermal elevations <1 cm in diameter that:

- Contain serous exudates or serum mixed with blood, or are seropurulent in character (vesicopustules).
- Are discrete, grouped (e.g., herpetic infection), irregularly distributed, or linear as in rhus dermatitis (e.g., poison ivy).
- Are short-lived, either breaking spontaneously or evolving into bullae through enlargement or coalescence.

Bullae are blisters >1 cm in diameter containing serous or seropurulent fluid. When bullae are located within the epidermis, they present with thin and flaccid walls, which rupture resulting in erosions, scale, and crusts.

Pustules, vesicles with purulent exudates, occur as a primary lesion or as secondary change in a vesicle or bulla.

Vesicles develop when fluid becomes trapped under the epidermis, the top layer of the skin. Most vesicles rupture easily and release the fluid onto the skin. When the fluid dries, it may turn yellow or crusty.

Causes of vesicles include:

- Allergic reactions that cause skin irritation.
- Dermatitis or eczema.
- Contact dermatitis, such as from poison ivy or poison oak.
- Autoimmune disorders, such as bullous pemphigoid (adult).
- Skin diseases that cause blistering, such as porphyria cutanea tarda.
- Impetigo (caused by infection with strep or staph bacteria).
- Chickenpox or shingles.
- Herpes (cold sores).
# Chickenpox (Varicella)

**What is it?**

Chickenpox is an acute viral infectious disease.

It is caused by varicella-zoster virus (VZV), VZV is a DNA virus.

It is a member of Herpes virus group.

Primary infection results in Varicella. Recurrent infection results in herpes zoster (shingles).

**Incubation period:**

Usually 8 - 21 days from contact with an infected person.

**Signs and symptoms of chickenpox may include:**

- **Fever, malaise** (for 1-2 days) which may be present before a rash develops.

- **Rash** usually first appears on body, face, and scalp, it then spreads to limbs.

  Rash begins as small, red, flat spots that develop into itchy fluid-filled blisters.

  - Blisters are usually less than ¼” wide and have a red base.

  After the blister breaks, open sores will crust over to form dry, brown scabs.

- For some people, the virus can become active again later in life and cause shingles.

  - Each lesion progresses through a series of characteristic stages over about a week.

  - Papules and vesicles develop into pustules, which then crust over prior to healing.

- A prominent feature of chickenpox is the development of several crops of spots.

  The peak of the illness, 3-4 days after first appearance of the rash, there are lesions at **all stages** of development, from new vesicles through to crusts.

**How is it spread?**

Chickenpox is an airborne disease spread easily through coughing or sneezing of ill individuals or through direct contact with an infected person’s blisters or fluid from the blisters.

Contact with an infected person’s saliva.

A pregnant woman with chickenpox can pass it on to her baby during pregnancy.

A mother with chickenpox can pass it on to her newborn.

The virus does **not live** on objects like sheets, counters, or toys.
When is the person contagious?

From 2 days before spots appear and until all blisters have crusted over (which is usually 5 days after the first blisters appear). Child is most infectious 12 - 24 hours before the rash appears.

Prevention:

Chickenpox vaccine is available for all who are over one year of age and are susceptible to chickenpox.

Chickenpox vaccine given within 5 days of exposure to chickenpox disease is effective in preventing or reducing the severity of chickenpox.

Pregnant women with chickenpox:

For pregnant women, antibodies produced as a result of immunization or previous infection are transferred via the placenta to the fetus.

Women who are immune to chickenpox cannot become infected and do not need to be concerned about it for themselves or their infant during pregnancy.

Varicella infection in pregnant women could lead to viral transmission via the placenta and infection of the fetus.

If infection occurs during the first 28 weeks of gestation, this can lead to fetal varicella syndrome (also known as congenital varicella syndrome).

Effects on the fetus can range in severity from underdeveloped toes and fingers to severe anal and bladder malformation.

Congenital Varicella Syndrome:

Results from maternal infection during pregnancy.

Period of risk may extend through first 20-28 weeks of pregnancy.

Atrophy of extremity with skin scarring, low birth weight, eye and neurologic abnormalities.

Risk appears to be small (<2%).

Newborn infected with Varicella:

The risk of the baby developing the disease is greatest following exposure to infection in the period 7 days prior to delivery and up to 7 days following the birth.
Possible problems to newborn include:

Damage to brain: encephalitis, microcephaly, hydrocephaly, aplasia of brain.

Damage to the eye: optic stalk, optic cup, and lens vesicles, microphthalmia, cataracts, chorioretinitis, optic atrophy.

Other neurological disorder: damage to cervical and lumbosacral spinal cord, motor/sensory deficits, absent deep tendon reflexes, anisocoria/Horner's syndrome.

Damage to body: hypoplasia of upper/lower extremities, anal and bladder sphincter dysfunction.

Skin disorders: (cicatricial) skin lesions, hypopigmentation.

Laboratory Diagnosis:

Laboratory diagnosis is not routinely required.

Useful if confirmation of the diagnosis or determination of susceptibility is necessary.

Most frequent source of isolation is vesicular fluid.

Stained smears from vesicular scrapings (Tzanck Smear).

ELISA and Latex Agglutination (LA) useful in screening for varicella immunity.

Serology Tests for Varicella IgM antibody and PCR.

Treatment:

Varicella treatment mainly consists of easing the symptoms as there is no actual cure of the condition. If oral acyclovir is started within 24 hours of rash onset it decreases symptoms by one day but has no effect on complication rates.

Use of acyclovir therefore is not currently recommended for immunocompetent individuals.

Although there have been no formal clinical studies evaluating the effectiveness of topical application of calamine lotion, a topical barrier preparation containing zinc oxide and one of the most commonly used interventions, it has an excellent safety profile.

It is important to maintain good hygiene and daily cleaning of skin with warm water to avoid secondary bacterial infection. Scratching may also increase the risk of secondary infection.

To relieve the symptoms of chickenpox, people commonly use anti-itching creams and lotions, these lotions are not to be used on the face or close to the eyes.

Also, staying in a cold surrounding can help in easing the itching as heat and sweat makes it worse.

Aspirin is highly contraindicated.
Herpes labialis

Cold sores on the mouth are usually caused by herpes simplex type 1 virus.

This is the most common recurrent disease produced by type 1.

Clusters of localized vesicles occur, usually at the mucocutaneous junction of the lips. The vesicle ruptures, leaving a painful ulcer that heals without scarring.

The lesions may recur, repeatedly and at various intervals of time, in the same location.

Herpes labialis (cold sore) is a recurrence of oral HSV, a prodrome of tingling, warmth or itching at the site usually heralds the recurrence. About 12 hours later, red-ness appears followed by papules and then vesicles.

The acute phase is characterized by painful sores or blisters on the lips, mouth, face and sometimes in the throat, after the blisters appear, the cold sores usually break open, leak a clear fluid, and then crust over and disappear after several days to 2 weeks.

Recurrent infection on the lips is usually less serious than the first infection.

The permanent site of latent herpes simplex virus is the trigeminal ganglion.

HSV also infects neurons that innervate the epithelial tissue. The virus travels along the neuron (retrograde transport).

Signs and symptoms of cold sores may include:

- Superficial clear blisters with a red base which crust over.
- Blisters heal within days.
- Sore mouth that makes eating, drinking, and sleeping uncomfortable.
- Fever.
- Sore throat.
- Swollen lymph glands in neck.
- Some patient have the virus but don't get cold sores. They have no symptoms.

The virus replicates in the epithelial tissue yielding a characteristic “fever blister” or “cold sore”.

The fluid in this blister is full of infectious virus. The blister ulcerates and forms a crusted lesion that heals without a scar.

Acute Gingivostomatitis: The patient experiences pain and bleeding of the gums. 1 - 8 mm ulcers with necrotic bases are present. Neck glands are commonly enlarged accompanied by fever.
**Incubation period:**

Usually 2 – 12 days from date of contact with infected person.

**When is the person contagious?**

When the cold sore is open.

**Transmission:**

Generally through direct contact with person shedding virus (from a kiss), as the virus is shed in saliva, tears, genital and other secretions.

Some people shed virus despite absence of lesions. Virus enters through mucosal tissues; cannot penetrate healthy skin.

There are 2 peaks of incidence, the first at 0 - 5 years and the second in the late teens, when sexual activity commences.

Following primary infection, 45% of orally infected individuals and 60% of patients with genital herpes will experience recurrences.

**How to prevent spread of the illness to other children:**

Exclude children from the child care facility when it is their first attack with a cold sore and they drool or have a weeping or open cold sore. They can return when the cold sore is crusted over.

Keep children with cold sores away from newborn babies, children with eczema or burns, and people with weakened immune systems.

Avoid kissing a child or adult with cold sores. Ensure children do not kiss each other when they have cold sores or uncontrollable drooling.

Ensure child with cold sores does not share toys (that are put in the mouth) with other children.

**HSV- Latency and Reactivation:**

HSV infects neurons that innervate the epithelial tissue. The virus travels along the neuron (retrograde transport)

- oral mucosa → trigeminal ganglia
- genital mucosa → sacral ganglia

A latent infection is established in the nervous tissue.

The virus can travels back down the nerve axon and arrives at the mucosa that was initially infected.
Vesicles containing infectious virus are formed on the mucosa and the virus spreads.

Recurrent infections are usually less pronounced than the primary infection (asymptomatic) and resolve more rapidly.

**Several agents may trigger recurrence:**

1- UV-B radiation (skiing, tanning)
2- Fever (hence the name "fever blister")
3- Emotional stress (e.g. final examinations, big date)
4- Physical stress (irritation)
5- Menstruation
6- Foods: Spicy, acidic, allergies
7- Immunosuppression:
   - Transient (stress related)
   - Chemotherapy, radiotherapy
   - Human immunodeficiency virus

**Laboratory Diagnosis:**

Samples: swabs, vesicle fluid, tissue, serum.

Tzanck prep

Culture (fresh ulcer or vesicle)

Immunofluorescence test

Serology

**Treatment:**

Cold sores will usually start to heal on their own within a few days.

Treatment may include skin creams, ointments, or pills.

At present, there are only a few indications of antiviral chemotherapy, with the high cost of antiviral drugs being a main consideration.

Generally, antiviral chemotherapy is indicated where the primary infection is especially severe, where there is dissemination, where sight is threatened, and herpes simplex encephalitis.