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**Common pediatric viral infection**

* **Chicken pox**
* **Herpes**
* **Roseola infantum**
* **Measles**
* **Rubella**
* **Mumps**
* **COVID-19**

**Chickenpox (varicella)**

* VZV is a neurotropic double stranded DNA herpesvirus
	+ Persons with varicella are contagious 1-2 day before the rash and until vesicles are crusted, usually 3-7 days after onset of rash.
* VZV is transmitted by contact with oropharyngeal secretions and the fluid of skin lesions of infected individuals, either by airborne spread or through direct contact.
	+ I.P. is 10-21 days.

 

* Prodromal symptoms begin 1-2 days before the

rash as **fever, malaise, anorexia, headache**, and occasionally mild abdominal pain; these symptoms usually resolve within 2-4 days after the rash.

* Varicella lesions often appear first on the scalp, face, or trunk. Initial exanthem consists of intensely pruritic erythematous macules that evolve through the papular stage to form clear, fluid-filled vesicles. While the initial lesions are crusting, new crops form on the trunk and then the extremities; the simultaneous presence of lesions in various stages of evolution is characteristic of varicella. The distribution of the rash is predominantly central with the greatest concentration on the trunk and proximally on the extremities. Ulcerative lesions involving the mucosa of the oropharynx and vagina are also common.

**Differential Dx**. Vesicular rashes caused by other infectious agents e.g. herpes simplex virus, enterovirus, rickettsial pox, and S. aureus; drug reactions; disseminated herpes zoster; contact dermatitis; and insect bites.

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**Complications: -**

* **Secondary bacterial infections** of the skin are usually caused by group A Streptococcus and S. aureus.
* **Encephalitis and Cerebellar Ataxia** are well-described neurologic complications that are highest among patients <5 yr and >20 yr.
* **Meningoencephalitis** usually begin 2-6 days after the onset of the rash but may occur during the incubation period or after resolution of the rash; clinical recovery is typically rapid occurring within 1-2 days, and is usually complete.
* **Varicella pneumonia** is a severe Cx that manifests as cough, dyspnea, cyanosis, pleuritic chest pain, and hemoptysis.
* **Progressive varicella** with visceral organ involvement, coagulopathy, severe hemorrhage, and continued vesicular lesion development after 1 week. Immunocompromised children, pregnant women, and newborns may herald severe, and potentially fatal, diseases.
* **Others**: mild hepatitis, mild thrombocytopenia, nephritis, nephrotic syndrome, HUS, arthritis, myocarditis, pericarditis, pancreatitis, orchitis, and acute retinal necrosis.

Herpes zoster VZV is transported in a retrograde manner through sensory axons to the dorsal root ganglia throughout the spinal cord. Herpes zoster is caused by the reactivation of latent VZV → vesicular rash that usually is dermatomal in distribution. HZ is rare in healthy children <10 yr of age, except immunocompromised patients or those infected with VZV in utero or 1st year of life.HZ in children tends to be milder than that in adults.

**Diagnosis:**

is mainly clinical; however, tests are used for confirmation in atypical cases including

• CBP: Leukopenia followed by relative and absolute lymphocytosis.

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 • Liver function tests mildly elevated.

• CSF shows a profile of viral meningoencephalitis in neurological Cx.

 • Serology: 4-fold or greater rise in VZV IgG antibodies is confirmatory of acute infection (but this requires a 2-3 wk. delay to collect a convalescent specimen)

• VZV can be identified quickly by direct fluorescence assay of cells from cutaneous lesions (vesicular fluid) in 15-20 min, by PCR amplification testing (vesicular fluid, crusts) in hours to days, and by rapid culture with specific immunofluorescence staining in2-3 days.

**Treatment: -**

* Analgesia for neuritis
* Antipyretic.
* Oral therapy with acyclovir (20 mg/kg/dose) is given as 4 doses/day for 5 days.
* Famciclovir or valacyclovir in older children who can swallow tablets (these drugs are better absorbed by the oral route than acyclovir).

 Note: Acyclovir therapy is not recommended routinely for treatment of uncomplicated varicella in the otherwise healthy child.

**Prognosis: -**

The lowest case fatality rates are among children 1-9 yr of age. Infants have a 4 times greater risk of death, whereas adults have a 25 times greater risk of dying.

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**The human herpesviruses**

There are currently eight known human herpesviruses: herpes simplex virus 1 and 2 (HSV1 and HSV2), varicella zoster virus (VZV), cytomegalovirus (CMV), Epstein–Barr virus (EBV), and human herpesviruses 6, 7 and 8 (HHV 6–8). HHV8 is associated with Kaposi sarcoma in HIV-coinfected individuals. The hallmark of the herpesviruses is that, after primary infection, latency is established and there is long-term persistence of the virus within the host, usually in a dormant state. After certain stimuli, reactivation of infection may occur.

**Herpes simplex infections**

Herpes simplex virus (HSV) usually enters the body through the mucous membranes or skin, and the site of the primary infection may be associated with intense local mucosal damage. HSV1 is usually associated with lip and skin lesions, and HSV2 with genital lesions, but both viruses can cause both types of disease.

**Asymptomatic:** Herpes simplex infections are very common and are mostly asymptomatic.

**Gingivostomatitis:** This is the most common form of primary HSV illness in children. It usually occurs from 10 months to 3 years of age. There are vesicular lesions on the lips, gums and anterior surfaces of the tongue and hard palate, which often progress to extensive, painful ulceration with bleeding. There is a high fever, and the child is very miserable. The illness may persist for up to 2 weeks. Eating and drinking are painful, which may lead to dehydration. Management is symptomatic, but severe disease may necessitate intravenous fluids and acyclovir.

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Eczema herpeticum – In this serious condition, wide­ spread vesicular lesions develop on eczematous skin. This may be complicated by secondary bac­terial infection, which may result in septicemia.

**Skin manifestations**

Mucocutaneous junctions and damaged skin are par­ticularly prone to infection. ‘Cold sores’ are recurrent HSV1 lesions on the gingival (lip) margin.



Herpetic whitlows – These are painful, erythematous, edematous white pustules on the site of broken skin on the fingers. Spread is by auto­inoculation from gingivostomatitis and infected adults kissingtheir children’s fingers. In sexually active adolescents, HSV2 may be the cause



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**Eye disease**

Eye disease may cause a blepharitis or conjunctivitis. It may extend to involve the cornea, producing dendritic ulceration. This can lead to corneal scarring and ultimately loss of vision. Any child with herpetic lesions near or involving the eye requires ophthalmic investi­gation of the cornea by slit lamp examination.



**Treatment**

 For other cutaneous & superficial infections (including herpes labialis), oral acyclovir, valacyclovir, or famciclovir are used.

* For herpetic gingivostomatitis; oral acyclovir; 15 mg/kg/dose 5 times a day for 1 wk
* In severe, disseminated or CNS infections, IV Acyclovir are used (10 mg/kg every 8 hr for 2-3 wk).

**Prevention**

* Good handwashing and the use of gloves (by healthcare workers) provide excellent protection.
* C/S delivery for pregnant women with active genital herpes.
* In immunocompromised patients can be prevented by the daily use of oral acyclovir, valacyclovir, or famciclovir.

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**Roseola Infantum (Exanthem Sabitum)**

**Typical features: -**



 High fever in a child aged 6–36 months.

 Minimal toxicity.

 Rose-pink maculopapular rash appears when fever subsides.

**General Considerations:-**

Roseola infantum is a benign illness caused by human herpesviruses 6 (HHV-6) or 7 (HHV-7). HHV-6 is a major cause of acute febrile illness in young children. Its significance is its ability to mimic more serious causes of high fever and its role in inciting febrile seizures.

**Clinical Findings:**-

The most prominent historical feature is the abrupt onset of fever, often reaching 40.6 °C, which lasts up to 8 days (mean, 4 days) in an otherwise mildly ill child. The fever then ceases abruptly, and a characteristic rash may appear. Roseola occurs predominantly in children aged 6 months to 3 years, with 90% of cases occurring before the second year. It is the most common recognized cause of exanthematous fever in this age group and is responsible for 20% of emergency room visits by children aged 6–12 months.

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**Symptoms and signs:**

Mild lethargy and irritability may be present, but generally there is a dissociation between systemic symptoms and the febrile course. The pharynx, tonsils, and tympanic membranes may be injected. Conjunctivitis and pharyngeal exudate are notably absent. Diarrhea and vomiting occur in one third of patients. Adenopathy of the head and neck often occurs. The anterior fontanelle is bulging in one quarter of HHV-6-infected infants. If rash appears (10–20% incidence), it begins on the trunk and spreads to the face, neck, and extremities. Rose-pink macules or maculopapules, 2–3 mm in diameter, are nonpruritic, tend to coalesce, and disappear in 1–2 days without pigmentation or desquamation. The rash may occur without fever.

**Laboratory findings:**

Leukopenia and lymphocytopenia are present early. Laboratory evidence of hepatitis occurs in some patients, especially adults.

**Differential Diagnosis:**

The initial high fever may require exclusion of serious bacterial infection. The relative well-being of most children and the typical course and rash soon clarify the diagnosis. The erythrocyte sedimentation rate is normal. If the child has a febrile seizure, it is important to exclude bacterial meningitis. The CSF is normal in children with roseola. In children who receive antibiotics or other medication at the beginning of the fever, the rash may be attributed incorrectly to drug allergy.

**Complications & Sequelae:**

Febrile seizures occur in 10% of patients. There is evidence that HHV-6 can directly infect the central nervous system, causing meningoencephalitis or aseptic meningitis. Multiorgan disease (pneumonia, hepatitis, bone marrow suppression, encephalitis) may occur in immunocompromised patients.

**Treatment & Prognosis:**

Fever is managed readily with acetaminophen and sponge baths. Fever control should be a major consideration in children with a history of febrile seizures. Roseola infantum is otherwise entirely benign.

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**Measles ( Rubuola)**

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* It is a single-stranded, lipid-enveloped RNA virus; human is the only host for measles.
* Measles is **highly contagious** & serious infection; transmission of virus is through the respiratory tract or conjunctivae following contact with large- or small-droplet aerosols. Face-to-face contact is not necessary, because viable virus may be suspended in air for as long as **1 hr** after the patient leaves a room
* **Measles consists of 4 phases:** incubation period, prodromal illness, exanthematous phase, and recovery. Fusion of infected cells results in multinucleated (up to 100 nuclei) giant cells called "**WarthinFinkeldey giant cells**" which are pathognomonic for measles, with intracytoplasmic and intranuclear inclusions (represent the virus).
* I.P. is 8-12 days.
* The prodromal phase begins with a mild fever followed by conjunctivitis, coryza, cough, and increasing fever**. Koplik spots** are the pathognomonic sign of measles, it is an enanthem appearing 1-4 days before the onset of the rash; it first appears as discrete red lesions with bluish-white spots in the center on the inner aspects of the cheeks at the level of the premolars. They may spread to involve the lips, hard palate, and gingiva.



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* The rash begins on the forehead (around the hairline), behind the ears, and on the upper neck as a red maculopapular eruption. It then spreads downward to the torso and extremities and may reach to palms and soles. The exanthem frequently becomes confluent on the face and upper trunk. With the onset of the rash, symptoms begin to subside. The rash fades over ≈ 1 wk in the same progression as it evolved. Cough lasts longer, often up to 10 days. In more-severe cases, generalized LAP may be present.

**Diagnosis**

 is almost always based on **clinical** and epidemiologic findings.

• CBP; Leukopenia. ESR & CRP are normal.

• Serology, viral isolation, & PCR may be needed to confirm Dx.

**Differential diagnosis**

 Rubella, Adenovirus infection, Enterovirus infection, EBV infection, Exanthem subitum (in infants) and Erythema infectiosum (in older children), Mycoplasma pneumoniae and group A streptococcus infections, Kawasaki syndrome, and Drug eruptions.

**Complications: -**

Risk factors:- age <5 yr (especially <1 yr) and >20 yr, crowding, severe malnutrition, immunodeficiency, & low serum retinol levels (vit. A deficiency).

**• Respiratory Cxs**: Pneumonia is the most common cause of death in measles. It may manifest as giant cell pneumonia caused direct viral infection or as superimposed bacterial infection e.g. Streptococcus pneumoniae, Haemophilus influenzae, & Staphylococcus aureus. Croup, tracheitis, and bronchiolitis are common in infants and toddlers.

**• ENT Cxs** e.g. acute otitis media, mastoiditis, & sinusitis.

**• GIT Cxs:** Diarrhea and vomiting with dehydration are common symptoms associated with measles; appendicitis or abdominal pain may occur.

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**• Neurological Cxs**: Febrile seizures occur in <3%. Encephalitis is mainly occurring in adolescents and adults. It is due to postinfectious, immunologically mediated process and is not the result of a direct effect of the virus. Clinical onset begins during the exanthem and manifests as seizures, lethargy, coma, and irritability. Death occurs in 15% of patients with measles encephalitis; others may suffer long-term sequelae, including cognitive impairment, motor disabilities, and deafness. Subacute Measles Encephalitis manifests 1-10 mo. after measles in immunocompromised patients; it results from direct damage to the brain by the virus and manifest as seizures, myoclonus, stupor, and coma; progressive disease and death almost always occur.

Subacute Sclerosing Panencephalitis (SSPE) is a rare disease but nearly always fatal. SSPE begins insidiously 7-13 yr after primary measles infection when the virus apparently regains virulence and attacks the CNS cells → inflammation and cell death. It is passed into 4 stages; the 1st stage manifests as subtle changes in behavior, irritability, reduced attention span, and temper outbursts.

**• Rare Cxs include**: Hemorrhagic or “black” measles which is often fatal and manifests as hemorrhagic skin eruption. Other rare Cxs are keratitis, myocarditis, thrombocytopenia, bacteremia, cellulitis, and toxic shock syndrome.

* Measles during pregnancy is associated with high rates of maternal morbidity, fetal wastage, and stillbirths; congenital malformations occur in 3% of liveborn infants.

**Treatment**

Management of measles is supportive e.g. maintenance of **hydration, oxygenation**, and comfort. **Antipyretics** are useful for comfort and fever control. Oral rehydration is effective in most cases, but severe dehydration may require IV therapy. For patients with respiratory tract involvement, airway humidification and supplemental oxygen may be of benefit. Respiratory failure from croup or pneumonia may require **ventilatory support**. **Vitamin A** therapy is indicated for all patients with measles.

Vitamin A should be administered once daily for 2 days at doses of 50,000 IU for infants <6 mo; 100,000 IU for infants between 6 mo-1 yr; and 200,000 IU for children >1 yr. In children with signs and symptoms of vitamin A deficiency, a 3rd dose is recommended 2-4 wk after the 2nd dose.

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 **Note**: Antiviral therapy is not effective in Rx of measles; likewise, prophylactic antimicrobial therapy to prevent bacterial infection is not indicated.

**Prevention**

 Patients are infectious from 3 days before rash (≈ 1 wk. after exposure) up to 4-6 days after the onset of rash, whereas immunocompromised patients will shed the virus throughout the illness. Therefore, these patients should be isolated during this period.

Measles vaccine is usually given as MMR in 2 doses; 1st at 12-15 mo.; the 2nd at 4-6 yr. (or any time after 1 mo. following the 1st dose).

Note: Passively administered immune globulin may inhibit the immune response to live measles vaccine, and administration should be delayed for variable amounts of time based on the dose of Ig.

Post-exposure prophylaxis can be given to prevent or modify infection. It is done by either vaccine if given within 72 hr of exposure, or Immune globulin up to 6 days after exposure. Immune globulin is indicated for infants <6 mo, pregnant women, and immunocompromised persons.

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**RUBELLA (German measles)**

* It is a single-stranded RNA virus; humans are the only known host. Transmission of rubella is in same manner as that of measles.
* Postnatal infection with rubella is a mild disease especially in children.
* I.P. is 2-3 wk.
* The prodrome consist of low-grade fever, sore throat, red eyes, headache, malaise, anorexia, and LAP (especially suboccipital, postauricular, and anterior cervical LNs).
* In children, the first manifestation of rubella is usually the rash, although it is variable and not distinctive but has similar manner as that of measles.
* The duration of the rash is generally 3 days (thus rubella also called 3-day measles).
* About the time of onset of the rash, an enanthem develop called "Forchheimer spots" as tiny, rose-colored lesions on oropharynx or petechial hemorrhages on soft palate.

**Diagnosis**

it is mainly **clinical**; other tests include:

* Rubella IgM enzyme immunosorbent assay which is typically present about 4 days after the appearance of the rash is the most common diagnostic test.
* PCR & viral culture.
* CBP: Leukopenia and mild thrombocytopenia.

**Complications**

usually self-limited, and generally not life-threatening; they include:

 \* Postinfectious Thrombocytopenia; about 2 wk after the rash.

\*Arthritis about 1 wk after the rash; more commonly among adult women.

\* Encephalitis is the most serious Cx; it occurs in 2 forms: a postinfectious syndrome following acute rubella and a rare progressive rubella panencephalitis (PRP) manifesting as a neurodegenerative disorder year following rubella (similar to SSPE of measles).

\* Other rare Cxs include Guillain-Barre syndrome, peripheral neuritis, & myocarditis.

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**Congenital Rubella Syndrome**

 The major clinical significance of Rubella is the transplacental infection and fetal damage. The most important risk factor for severe congenital defects is the **stage of gestation** at the

time of infection. The risk for congenital defects decrease as the gestational age increase. Maternal infection during the 1st 8 wk of gestation results in the most severe and widespread defects; whereas defects occurring after 16 wk of gestation are uncommon, even if fetal infection occurs.

may involve nearly every organ system including:

• **CVS**: PDA (most common), Pulmonary artery stenosis, VSD, Myocarditis

**• CNS**: Chronic meningitis, Parenchymal necrosis, Vasculitis with calcification

**• Eye:** microphthalmia, cataract, iridocyclitis, ciliary body necrosis, glaucoma, retinopathy.

**• Ear:** cochlear hemorrhage, endothelial necrosis. Note: Nerve deafness is the single most common finding among infants with CRS.

**• Lung**: chronic mononuclear interstitial pneumonitis.

**• Liver:** hepatic giant cell transformation, fibrosis, lobular disarray, bile stasis.

**• Kidney:** interstitial nephritis.

**• Adrenal gland:** cortical cytomegaly.

**• Bone:** malformed and poor mineralization of osteoid, thinning cartilage.

**• Spleen, lymph nodes:** extramedullary hematopoiesis.

**• Thymus:** histiocytic reaction, absence of germinal centers.

**• Skin:** erythropoiesis in dermis.

**• Late-onset manifestations** of CRS include: PRP, diabetes mellitus, thyroid dysfunction.

**Treatment**

* There is no specific Rx available for either acquired rubella or CRS.
* Postnatal rubella is generally a mild illness that requires no care.

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* beyond antipyretics and analgesics. IVIG or corticosteroids can be considered for severe, non-remitting thrombocytopenia.

Management of children with CRS is more complex and requires pediatric, cardiac, audiologic, ophthalmologic, and neurologic evaluation and follow-up because many manifestations may not be readily apparent initially or may worsen with time. Hearing

screening is of special importance, because early intervention may improve outcomes in children with hearing problems caused by CRS.

**Prevention**

Rubella vaccination is given as MMR or MMRV in a 2-dose regimen at 12-15 mo. then at 4-6 yr. of age. Post-exposure prophylaxis of vaccine administered within 3 days of exposure. Vaccination of women in the child-bearing age (not during pregnancy) is highly effective in prevention of CRS.

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**MUMPS**

**Etiology: -**

 It is a single-stranded pleomorphic **RNA** virus; humans are the only natural host. It targets the salivary glands, CNS, pancreas, testes, and, to a lesser extent, thyroid, ovaries, heart, kidneys, liver, and joint synovia.

**Epidemiology: -**

 Mumps is spread from person to person by respiratory droplets. The period of maximum infectiousness is **1-2 days** before to **5 days** after onset of parotid swelling.



 **Clinical manifestations: -**

 Incubation period usually **16-18** days. Clinical presentation ranging from asymptomatic or nonspecific symptoms to the typical illness associated with parotitis +/\_ Cxs involving several body systems. The prodrome lasts 1-2 days and consisting of fever, headache vomiting, and achiness.,

Parotitis then appears and may be unilateral initially but becomes bilateral in ≈ **70%** of cases. The parotid gland is tender, and may be accompanied by ear pain. Note: Ingestion of sour or acidic foods or liquids may enhance pain in the parotid area. As swelling progresses, the angle of the jaw is obscured. The opening of the Stensen duct in the mouth may be red and edematous. The parotid swelling peaks in ≈ 3 days and then gradually subsides over 7 days. Fever and the other systemic symptoms resolve in 3-5 days. Submandibular salivary glands may also be involved (or may be enlarged without parotid swelling). Edema over the sternum as a result of lymphatic obstruction may also occur.

**Investigations:-**

 It is mainly clinical; other tests include:

• CBP: Leukopenia with a relative lymphocytosis.

 • ↑ serum amylase (due to parotitis).

 • Serological studies, viral isolation & culture, PCR.

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**Differential Diagnosis:-**

Viral and bacterial infection of parotid gland, Submandibular or anterior cervical LAP, obstruction of Stensen duct, tumors, and collagen vascular diseases e.g. Sjögren syndrome & SLE

**complications**

1. **Meningitis and Meningoencephalitis**: Symptomatic CNS involvement occurs in 10-30% of infected individuals, but CSF pleocytosis has been found in 40-60% of patients with mumps parotitis. The meningo-encephalitis may occur before, along with, or most commonly 5 days after the parotitis. Infants and young children have fever, malaise, and lethargy, whereas older children, adolescents, and adults complain of headache and demonstrate meningeal signs. Symptoms usually resolve in 7-10 days. Less-common CNS Cxs of mumps include: transverse myelitis, acute disseminated encephalomyelitis (ADEM), aqueductal stenosis, facial palsy and rarely sensorineural hearing loss.
2. **Orchitis and Oophoritis:** Involvement in prepubescent boys is extremely rare, but after puberty, orchitis occurs in 30-40% of males. It is manifested as fever, chills, and exquisite pain and swelling of the testes. Atrophy of the testes may occur, but sterility is rare even with bilateral involvement. Oophoritis is uncommon in post pubertal females but may cause severe pain and may be confused with appendicitis.
3. **Pancreatitis** may occur with or without parotid involvement. Severe disease is rare, but fever, epigastric pain, and vomiting are suggestive.
4. **Uncommon and rare Cxs** of mumps include conjunctivitis, optic neuritis, pneumonia, nephritis, myocarditis, arthritis, thyroiditis, and thrombocytopenia.
5. Maternal infection with mumps during the 1st trimester of pregnancy results in ↑ fetal wastage. No fetal malformations have been associated with intrauterine mumps infection.

**Treatment: -** No specific antiviral therapy is available for mumps. **Analgesics** can be given for pain of meningitis or orchitis; **antipyretics** may be given for fever; maintain **adequate hydration**. The outcome of mumps is nearly always **excellent.**

**Prevention:-**Isolation of patients at least 5 days after onset of parotid swelling. Mumps vaccination is usually given as MMR or MMRV in a 2-dose regimen at 12-15 mo & then at 4-6 yr of age (or at 1 mo apart).

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**Clinical Manifestations:**

 The signs and symptoms of SARS-CoV-2 infection in children may be like those in adults, but most children may be asymptomatic or only have a few symptoms. The most common signs and symptoms of COVID-19 in hospitalized children are fever, nausea, vomiting, cough, shortness of breath, and upper respiratory symptoms. Signs and symptoms of COVID-19 may overlap significantly with those of other viral infections, including influenza and other respiratory and enteric viral infections. Although the true incidence of asymptomatic SARS-CoV-2 infection is unknown, asymptomatic infection was reported in up to 45% of children who underwent surveillance testing at the time of hospitalization for non-COVID-19 indication.

**Risk Factors:** Most hospitalized children with acute COVID-19 had underlying conditions e.g. prematurity, chronic lung disease, congenital heart disease, immune deficiency, obesity, neurologic and developmental disorders.

**Diagnosis:**

The diagnosis of COVID-19 infection can be confirmed by serologic testing, detection of viral RNA using RT-PCR, or isolation of the virus in cell culture. Antibodies are not detectable until 10 days after the onset of symptoms, and IgG sero-conversion may be delayed for up to 4 wk.

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**Multisystem Inflammatory Syndrome in Children (MIS-C)**

A small subset of children and young adults with SARS-CoV-2 infection de velop MIS-C.

This is an immune mediated condition. Most MIS-C patients have serologic evidence of previous SARS-CoV-2 infection, but only a minority are RT-PCR positive for SARS-CoV-2 at presentation. The peak incidence of MIS-C lags about 4 weeks behind the peak of acute pediatric COVID-19 hospitalizations. Unlike in children with acute COVID-19, the majority of children who present with MIS-C do not seem to have underlying comorbid conditions other than obesity.

Case definition for MIS-C includes:

1. An individual aged <21 years presenting with fever, laboratory evidence of inflammation, and evidence of clinically severe illness requiring hospitalization with multisystem of > 2 organ involvement e.g., cardiac, renal, respiratory, hematologic, gastrointestinal, dermatologic, or neurological
2. No alternative plausible diagnoses
3. Positive for current or recent SARS-CoV-2 infection by RT-PCR, antigen test, or serology; or COVID-19 exposure within the 4 weeks prior to the onset of symptoms.

Patients with MIS-C are often critically ill and up to 80% of children require ICU admission Supportive care remains the mainstay of therapy.

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