MEASLES (RUBEOLA)

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Essentials of diagnosis & typical features:

- **Exposure** to measles 9–14 days previously.
- **Prodrome** of fever, cough, conjunctivitis, and coryza.
- **Koplik spots** (few to many small white papules on a diffusely red base on the buccal mucosa) 1–2 days prior to and after onset of rash.
- **Maculopapular rash** spreading down from the face and hairline to the trunk over 3 days and later becoming confluent.
- **Leukopenia**.

General Considerations:

This childhood exanthem is "rarely" seen in the developed countries because of vaccination.

The attack rate in susceptible individuals is extremely high; spread is respiratory.

Morbidity and mortality rates in the developing world are substantial because of underlying malnutrition and secondary infections.

Because humans are the sole reservoir of measles, there is the potential to eliminate this disease worldwide.

Clinical Findings:

A history of contact with a suspected case may be absent because airborne spread is efficient and patients are contagious during the prodrome. Contact with an imported case may not be recognized.

In temperate climates, epidemic measles is a winter-spring disease. Many suspected cases are misdiagnoses of other viral infections.

Symptoms and signs:
High fever and lethargy are prominent.

Sneezing, eyelid edema, tearing, copious coryza, photophobia, and harsh cough ensue and worsen.

Koplik spots are white macular lesions on the buccal mucosa, typically opposite the lower molars. These are almost *pathognomonic* for measles, although they may be absent.

A discrete maculopapular rash begins when the respiratory symptoms are maximal and spreads quickly over the face and trunk, coalescing to a bright red. As it involves the extremities, it fades from the face and is completely gone within 6 days; fine desquamation may occur.

Fever peaks when the rash appears and usually falls 2–3 days thereafter.

Laboratory findings:

Lymphopenia is characteristic. Total leukocyte counts may fall to 1500/μL.

An experienced cytologist may see multinucleated giant cells in oral mucosal scrapings and in nasal secretions, but the diagnosis is usually made by detection of measles IgM antibody in serum drawn at least 3 days after the onset of rash or later by detection of a significant rise in antibody. Direct detection of measles antigen by fluorescent antibody staining of nasopharyngeal cells is a useful rapid method.

Imaging:
Chest x-rays often show hyperinflation, perihilar infiltrates, or parenchymal patchy, fluffy densities. Secondary consolidation or effusion may be visible.

Complications & Sequelae:

A. Respiratory complications
These occur in up to 15% of patients. Bacterial superinfections of lung, middle ear, sinus, and cervical nodes are most common. Fever that persists after the third or fourth day of rash suggests such a complication, as does leukocytosis. Bronchospasm, severe croup, and progressive viral pneumonia or bronchiolitis (in infants) also occur. Immunosuppressed patients are at much greater risk for fatal pneumonia than are immunocompetent patients.

B. Cerebral complications
Encephalitis occurs in 1 in 2000 cases. Onset is usually within a week after appearance of rash. Symptoms include combativeness, ataxia, vomiting, seizures, and coma. Lymphocytic pleocytosis and a mildly elevated protein concentration are usual CSF findings, but the fluid may be normal. Forty percent of patients so affected die or have severe neurologic sequelae.

"Subacute sclerosing panencephalitis" is a slow measles virus infection of the brain that becomes symptomatic years later in about 1 in 100,000 previously infected children. This progressive cerebral deterioration is associated with myoclonic jerks and a typical EEG pattern. It is fatal in 6–12 months. It rarely occurs following administration of vaccine, with an estimated incidence of less than 1 per 1,000,000 vaccinated individuals. High titers of measles antibody are present in serum and CSF.

C. Other complications:
These include **hemorrhagic measles** (severe disease with multiorgan bleeding, fever, cerebral symptoms), **thrombocytopenia**, **appendicitis**, **keratitis**, **myocarditis**, and **premature delivery** or **stillbirth**. Mild **liver** function test elevation has been detected in up to 50% of cases in young adults; frank jaundice may also occur. Measles causes transient **immunosuppression**; thus, reactivation or progression of tuberculosis (including transient cutaneous anergy) occurs in untreated children.

Treatment, Prognosis, & Prevention:

Recovery generally occurs 7–10 days after onset of symptoms.

Therapy is **supportive**: eye care, cough relief (avoid opioid suppressants in infants), and fever reduction (acetaminophen, lukewarm baths; avoid salicylates).

Secondary bacterial infections should be treated promptly; antimicrobial prophylaxis is not indicated.

Ribavirin is active in vitro and may be useful in infected immunocompromised children.

In malnourished children, **vitamin A** supplementation should be given to attenuate the illness.

The current two-dose active vaccination strategy is successful. **Vaccine** should not be withheld for concurrent mild acute illness, tuberculosis or positive tuberculin skin test, breast-feeding, or exposure to an immunodeficient contact. The vaccine is recommended for HIV-infected children without severe HIV complications and adequate CD4 cells (³ 15%).

Vaccination prevents the disease in susceptible exposed individuals if given within 72 hours.

Immune globulin (0.25 mL/kg intramuscularly; 0.5 mL/kg if immunocompromised) will prevent or modify measles if given within 6 days.

Suspected cases should be diagnosed promptly and reported to the local health department