Rubella (German Measles)

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Essentials of diagnosis & typical features

- History of rubella vaccination usually absent.
- Prodromal nonspecific respiratory symptoms and adenopathy (postauricular and occipital).
- Maculopapular rash beginning on face, rapidly spreading to the entire body, and disappearing by fourth day.
- Few systemic symptoms.
- Congenital Infection.
- Retarded growth, development.
- Cataracts, retinopathy.
- Purpuric "blueberry muffin" rash at birth, jaundice, thrombocytopenia.
- Deafness.
- Congenital heart defect.

General Considerations

If it were not teratogenic, rubella would be of little clinical importance. Clinical diagnosis is difficult in some cases because of its variable expression. In one study, over 80% of infections were subclinical. Because of inadequate vaccination, outbreaks now occur in adolescents or adults. Rubella is transmitted by aerosolized respiratory secretions. Patients are infectious 5 days before until 5 days after the rash.

Congenital rubella, in infants both of unimmunized women and of women who have apparently been reinfected in pregnancy, is now rare.

Clinical Findings:

The incubation period is 14–21 days. The nondistinctive signs may make exposure history unreliable. A history of immunization makes rubella unlikely but still possible. Congenital rubella usually follows maternal infection in the first trimester.

A. Symptoms and signs:

1. Infection in children—Young children may only have rash. Older patients often have a nonspecific prodrome of low-grade fever, ocular pain, sore throat, and myalgia. Postauricular and suboccipital adenopathy (sometimes generalized) is characteristic. This often precedes the rash or may occur without rash. The rash consists of erythematous discrete maculopapules beginning on the face. A "slapped-cheek" appearance or pruritus may occur. Scarlatiniform or morbilliform rash variants may occur. The rash spreads quickly to the trunk and extremities after it fades from the face; it is gone by the fourth day. Enanthem is usually absent.

2. Congenital infection—More than 80% of women infected in the first 4 months of gestation are delivered of affected infants; congenital disease occurs in less than 5% of women infected later in pregnancy. Later infections can result in isolated defects, such as deafness. The main manifestations are as follows:

a. **Growth retardation**. Between 50% and 85% of infants are small at birth and remain so.

b. **Cardiac anomalies**. Pulmonary artery stenosis, patent ductus arteriosus, ventricular septal defect.

c. **Ocular anomalies**. Cataracts, microphthalmia, glaucoma, retinitis.

d. **Deafness.**

e. **Cerebral disorders**. Chronic encephalitis.

f. **Hematologic disorders**. Thrombocytopenia, dermal nests of extramedullary hematopoiesis or purpura ("blueberry muffin" rash), lymphopenia.

g. **Others**. Hepatitis, osteomyelitis, immune disorders, malabsorption, diabetes.

B. Laboratory findings:

Leukopenia is common, and platelet counts may be low. Congenital infection is associated with low platelet counts, abnormal liver function tests, hemolytic anemia, pleocytosis, and very high rubella IgM antibody titers. Total serum IgM is elevated, and IgA and IgG levels may be depressed.

C. Imaging:

Pneumonitis and bone metaphysial longitudinal lucencies may be present in x-rays of children with congenital infection.

Diagnosis & Differential Diagnosis:

Virus may be isolated from throat or urine from 1 week before to 2 weeks after onset of rash. Children with congenital infection are infectious for months. The virus laboratory must be notified that rubella is suspected. Serologic diagnosis is best made by demonstrating a fourfold rise in antibody titer between specimens drawn 1–2 weeks apart. The first should be drawn promptly, because titers increase rapidly after onset of rash. Both specimens must be tested simultaneously by a single laboratory. Specific IgM antibody can be measured by immunoassay. Because the decision to terminate a pregnancy is usually based on serologic results, testing must be done carefully.

Rubella may resemble infections due to enterovirus, adenovirus, measles, EBV, roseola, parvovirus, Toxoplasma gondii, and Mycoplasma. Drug reactions may also mimic rubella. Because public health implications are great, sporadic suspected cases should be confirmed serologically or virologically.

Congenital rubella must be differentiated from congenital CMV infection, toxoplasmosis, and syphilis.

Complications & Sequelae

A. Arthralgia and arthritis:

Both occur more often in adult women. Polyarticular involvement (fingers, knees, wrists), lasting a few days to weeks, is typical. Frank arthritis occurs in a small percentage of patients. It may resemble acute rheumatoid arthritis.

B. Encephalitis:

With an incidence of about 1:6000, this is a nonspecific parainfectious encephalitis associated with a low mortality rate. A syndrome resembling subacute sclerosing panencephalitis has also been described in congenital rubella.

C. Rubella in pregnancy:

Infection in the mother is self-limited and not severe.

Prevention:

Rubella is one of the infections that potentially could be eradicated. Standard prenatal care should include rubella antibody testing. Seropositive mothers are at no risk; seronegative mothers are vaccinated after delivery.

A pregnant woman possibly exposed to rubella should be tested immediately; if seropositive, she is immune and need not worry. If she is seronegative, a second specimen should be drawn in 4 weeks, and both specimens should be tested simultaneously. Seroconversion in the first trimester suggests high fetal risk; such women require counseling regarding therapeutic abortion.

When pregnancy termination is not an option, some experts recommend intramuscular administration of 20 mL of immune globulin within 72 hours after exposure in an attempt to prevent infection. (This negates the value of subsequent antibody testing.) The efficacy of this practice is unknown.

Treatment & Prognosis:

Symptomatic therapy is sufficient. Arthritis may improve with administration of anti-inflammatory agents. The prognosis is excellent in all children and adults but poor in congenitally infected infants, in whom most defects are irreversible or progressive. The severe cognitive defects seem to correlate closely in these infants with the degree of growth failure.