15

Communicable Diseases of Children

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The communicable diseases of childhood are a source of significant disruption for the family and a particular challenge to the family physician. Although most of these illnesses are self-limited and without significant sequelae, the socioeconomic impact due to time lost from school (and work), costs of medical visits and remedies, and parental anxiety are enormous. Distressed parents must be treated with sensitivity, patience, and respect for their judgment, as they have often agonized for hours prior to calling the physician. They are usually greatly reassured when given a specific diagnosis and an explanation of the natural history of even the most minor syndrome.

It is essential to promptly differentiate serious from benign disorders (e.g., acute epiglottitis versus spasmodic croup), to recognize serious complications of common illnesses (e.g., varicella encephalitis), and to recognize febrile viral syndromes (e.g., herpangina), thereby avoiding antibiotic misuse. To this end, the problem-oriented format of this chapter places primary emphasis on differential diagnosis. A detailed discussion of childhood immunizations and of infections in the day-care setting is beyond the scope of this chapter but is available in the "Red Book."[1] Neonatal infections are covered elsewhere in this text.

Differential Diagnosis of the Common Cold in Children

An infant or child with "a cold," as described by the parent or caretaker, may have one of several viral-like respiratory syndromes.[2] A "bad cold" may represent simply that, or it may be a specific, sometimes serious disorder such as bronchiolitis. A common cold is defined as an acute viral disease consisting of nasal stuffiness, sneezing, coryza, throat irritation, and minimal to no fever. Routine laboratory studies are unnecessary, and required treatment is minimal. If signs and symptoms deviate from these criteria, the history, physical examination, and appropriate laboratory studies often define one of several other more specific respiratory syndromes as summarized in Table 15.1.[3–10]

Key points to recall are that significant pharyngitis is *not* present with most colds, that most colds are 3- to 7-day illnesses (except for lingering cough and coryza for up to 2 weeks), and that abrupt worsening of symptoms or development of high fever mandates prompt reevaluation. Tonsillopharyngitis (hemolytic streptococci, Epstein-Barr virus, adenovirus, *Corynebacterium*) usually involves a sore throat, fever, erythema of the tonsils and pharynx with swelling or edema, and often headache and cervical adenitis. In addition to the entities listed in Table 15.1, colds must also be differentiated from allergic rhinitis, asthma, nasal or respiratory tree foreign bodies, adenoiditis, otitis media, sinusitis, diphtheria, influenza, bronchitis, respiratory mycoplasmal and chlamydial infections, measles prodrome, and pneumonia.[2,5] These entities are discussed elsewhere in this text.

Specific viral diagnosis is generally unnecessary for the common cold but may be useful to confirm specific syndromes such as pharyngoconjunctival fever, or when used in the first few cases of an outbreak of similar illnesses, especially to confirm agents such as influenza A or B or respiratory syncytial virus. The *nasal wash procedure* is a useful method for nasopharyngeal viral culture[11]: Three to seven milliliters of buffered saline is drawn into a 1-ounce tapered rubber bulb syringe. The bulb is inserted until it occludes the nostril. With one complete squeeze and release, the nasal wash is collected in the bulb. The material is then placed in viral culture or transport medium.

Fever

The degree and duration of fever should not be used to differentiate bacterial from viral infections, as many viral syndromes

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result in high or prolonged fever. Successful treatment of fever does not mandate driving the temperature to normal; comfort of the child should be a main consideration. Mild fever may be beneficial with viral illnesses.[12] In a placebo-controlled study of volunteers challenged intranasally with rhinovirus, aspirin and acetaminophen were significantly associated with suppression of serum neutralizing antibody response and increased nasal symptoms and signs; they also produced a trend toward longer duration of viral shedding.[13]

Croup

Croup is a spectrum of viral respiratory syndromes characterized by varying degrees of inspiratory stridor, cough, and hoarseness due to laryngeal-region obstruction. The symptoms include laryngitis (older children and adults) and nonrecurrent and spasmodic croup (diseases of younger children caused predominantly by parainfluenza 1 viruses). Prompt diagnosis and assessment of the severity of croup-like illnesses are essential. Table 15.1 lists salient features of croup syndromes and the often similar life-threatening illnesses acute epiglottitis and bacterial tracheitis. In addition, laryngeal diphtheria must always be considered in conjunction with croup; it is differentiated by the patient's immunization history, relatively slow progression of the disease, a high degree of hoarseness, and the pharyngeal signs of diphtheria.[2,7,8]

Respiratory Syncytial Virus

Respiratory syncytial virus (RSV) is the major cause of bronchiolitis and pneumonia in young children, with outbreaks occurring yearly during the winter and early spring. When an outbreak occurs, RSV tends to dominate infant and small child illnesses in a community, seeming to brush aside the activity of other respiratory viruses. An abrupt increase in bronchiolitis and pediatric pneumonia in a community suggests RSV infection, which may be confirmed by performing viral culture or rapid diagnostic tests in initial cases. The virus is spread by close contact (large respiratory droplets) or by entry through the nose or eyes from porous and nonporous fomites, hands, or gloves. Nosocomial infection is common. Naturally acquired immunity to RSV is incomplete, and repeated infections are common, although the primary encounter generally results in the most severe illness.[10] Bronchiolitis must be differentiated from asthma, an often difficult task. Asthma is favored by the following factors: age over 1 year, positive family history, *repeated* attacks, sudden onset without prodrome, prompt response to β-agonists.

Acute Parotid or Cervical Swelling

Parotid gland enlargement results in visible swelling distributed fairly evenly above and below the angle of the jaw; the swelling of acute cervical adenitis remains below the jaw. Elevated serum amylase may differentiate parotitis from cervical adenitis, abscess, or other mass but is not specific for mumps. Exudation of pus is seen only with suppurative parotitis, which may be caused by *Staphylococcus* or *Hemophilus influenzae.* Other causes of acute parotitis include viruses [influenza, parainfluenza, coxsackie, ECHO, Epstein-Barr, cytomegalovirus (CMV), lymphocytic choriomeningitis] and certain drugs.[14] Subacute and chronic parotitis and adenitis, which may be caused by a variety of conditions, are discussed elsewhere (see Chapter 76).

Acute unilateral cervical adenitis[2,15] in children is generally caused by β-hemolytic streptococci or *Staphylococcus aureus,* and it generally is a result of spread from local infections of the head and neck. Usual findings are a large, tender, unilateral cervical mass, with or without overlying erythema, fever, and leukocytosis. The diagnosis may be made on clinical grounds, but needle aspiration of the inflamed node for Gram and acid-fast stains and cultures is often useful. A skin test for tuberculosis should be considered. Complications may include node suppuration or bacteremia. Initial empiric antibiotic therapy is oral dicloxacillin, cephalexin, or amoxicillin-clavulenic acid (Augmentin). Incision and drainage should generally be performed for fluctuant or pointing nodes. Close follow-up is mandatory.

Causes of acute bilateral cervical adenitis are infectious mononucleosis, tularemia, and diphtheria.

Mumps

Mumps virus infects only humans and causes a *systemic* illness that may serve as a prototype for other systemic viral illnesses. It is spread primarily by respiratory droplets. The usual incubation period is 16 to 18 days (range 12–25 days). The period of communicability ranges from 7 days before swelling to 9 days after. The disease is most common during late winter and spring in North America, where a few thousand cases still occur each year. The peak incidence has shifted from the 5- to 9-year-old age group to those 10 to 19 years owing to incremental vaccine introduction between 1967 and 1977.[1,14,16]

Clinical Presentations and Diagnosis

Approximately 30% of infections are subclinical.[1,14,16] Classic symptoms are bilateral or unilateral parotid gland swelling (present for up to 10 days) and fever up to 40.0° (104°F) lasting 1 to 6 days. Parotitis may be preceded by several days of nonspecific symptoms including malaise, fever, headache, myalgias, and anorexia. Swelling of other salivary glands may occur. The clinical diagnosis can be made with confidence after observation of typical signs and symptoms in patients with outbreak-associated disease or known mumps exposure. Mumps virus may be isolated from throat washings, urine, cerebrospinal fluid (CSF), and other bodily fluids. A variety of paired serologic tests are available.

Serious sequelae may occur without observable parotitis. Meningeal signs may be present in 15% of cases and meningoencephalitis in 0.5%, with a case fatality rate of 1.4%.[1] In a series of 20 naturally acquired mumps meningoencephalitis cases, the average age was 6 years; most cases were characterized by fever, vomiting, meningismus, headaches, and sometimes seizures; and the CSF showed lymphocytosis.[17] Most mumps meningoencephalitis patients experience full recovery,

Table 15.1. **Selected Respiratory Tract Syndromes in Children**

Syndrome	Usual etiologic agents	Typical clinical presentation	Diagnosis
Common cold	Rhinovirus, coronavirus, parainfluenza virus, respiratory syncytial virus (RSV). Occasional: adenovirus, enterovirus, influenza virus, coxsackie virus, reovirus (*Mycoplasma pneumoniae*).	*Children:* Throat irritation, nasal stuffiness; then sneezing, clear rhinorrhea; followed in 1–3 days by more purulent rhinorrhea, nasal obstruction, subjective sore throat. May have malaise, myalgias, chilly feelings, headache, anorexia, mild fever, cough. *Nasopharyngitis:* same as above plus objective evidence of pharyngitis. *Infants:* Coryza, often nasal congestion, irritability, restlessness. More often febrile than children. May have feeding/sleep disturbance, vomiting, diarrhea.	Clinical. Viral cultures by nasal wash/nasopharyngeal swab not routinely warranted. Rule out tonsillopharyngitis (see text).
Pharyngoconjunctival fever	Adenoviruses.	Abrupt onset of fever, pharyngitis, conjunctivitis (uncommon if unassociated with swimming). Often headache, anorexia, cervical adenopathy, adenoid hypertrophy, eye discomfort, flushed face. May have malaise, myalgias, gastrointestinal symptoms, migratory palpebral erythema.[6] May persist 2 weeks.	Clinical. For specific diagnosis: conjunctival/pharyngeal viral culture; paired serology; rapid serologic tests.
Herpangina	Coxsackie A and B, and echoviruses. ? Herpes simplex virus.	Sudden onset of fever up to 41.1°C (106°F) followed by 1–14 small papular to vesicular to ulcerative lesions on erythematous base on anterior tonsillar pillars, soft palate, uvula, tonsils, posterior buccal mucosa. May have anorexia, drooling, sore throat, choryza, headache, abdominal/back pain, vomiting, diarrhea.	Clinical. For specific etiology: viral culture of lesions, throat, or rectum.
Pertussis (whooping cough)	Toxicogenic strains of *Bordetella pertussis.* (Whooping cough syndrome can be caused by *B. parapertussis, Chlamydia trachomatis,* adenoviruses.)	*Catarrhal stage:* mild upper respiratory symptoms with cough. Can progress to *paroxysmal stage:* severe bursts of cough with inspiratory whoop followed by emesis. Mild or no fever. Whoop may be absent in older children or < 6 months old. Apnea common in those < 6 months old. Duration of uncomplicated cases: 6–10 weeks.	Nasopharyngeal culture on special media (notify laboratory in advance). Many false-negative cultures. Serology diagnostic but insensitive. New tests being researched.
Laryngotracheobronchitis (nonrecurrent croup)[7,8]	Parainfluenza virus types 1–3; influenza virus, adenovirus, RSV (*Mycoplasma pneumoniae*).	Most common ages 3 months to 3 years; male/female 1.6:1.0; prodrome (2–5 days) of mild fever, rhinorrhea, malaise, sore throat, cough; then onset of barking, seal-like (croupy) cough, gradually increasing inspiratory stridor, fever up to 40.6°C (105°F), hoarseness, mildly inflamed pharynx. Wheezing if laryngotracheo*bronchitis.* Duration: 2–7 days.	Clinical. Correct diagnosis essential. Funneling of tracheal lumen (steeple sign) on frontal x-ray film most consistent finding, but nonspecific.
Spasmodic croup	Same as nonrecurrent croup.	Same ages as nonrecurrent croup; male/female 6:1. Minimal to no prodrome. Sudden nocturnal onset of croupy cough, dyspnea, and inspiratory stridor. No fever. Often family history or history of prior attacks and/or allergy/asthma history. Duration: 2–4 hours.	Clinical. Correct diagnosis essential.
Acute epiglottitis[7,8]	*Hemophilus influenzae* type B.	Typically 3–8 years old (less often < 2 years or adult). Prodrome uncommon. Abrupt onset and rapid progression of fever, marked apprehension, variable degrees of respiratory distress. May have sore throat, dysphagia, drooling, delirium, stridor, choking sensation. Looks toxic, has cherry red epiglottis, is in a sitting forward position with protruding chin/tongue; leukocytosis with left shift. (If < 2 years may appear like croup at first.)	Clinical features plus either enlarged epiglottis (thumb sign) on slightly extended lateral neck x-ray film or experienced, controlled view of epiglottis with intubation standby. Epiglottic (70% positive) and blood (90%) cultures for retrospective confirmation. Epiglottic gram stains/latex agglutination tests helpful.

Complications	Management	Prevention
Secondary infections: otitis media, acute sinusitis, bacterial adenoiditis, pharyngitis, pneumonia.	Increased fluid intake, saltwater nosedrops/sprays and/or cool-mist vaporization; consider acetaminophen or ibuprofen if significant fever/aches; dextromethorphan or codeine cough suppressant in children if cough disturbs sleep. Warm saltwater gargles and/or judicious use of antiseptic/anesthetic throat sprays/lozenges if nasopharyngitis.	Good handwashing. Isolation of patients impractical. Crowd/contact avoidance if susceptible to complications.
Superficial keratitis; secondary infections: sinusitis, otitis media, bacterial conjunctivitis.	Same as nasopharyngitis. *Avoid* steroid-containing ophthalmic preparations.	Adequate pool chlorination. Exclude ill persons from pools ≥2 weeks after recovery. Good handwashing.
Rarely: myocarditis, meningitis, encephalitis.	Fluids, supportive care. Useful local treatment of oral lesions: mixture of equal parts of viscous lidocaine 2% (Xylocaine), diphenhydramine (Benadryl) elixir, kaolin/pectin (Kaopectate) suspension: 4 drops to lesions prior to feeding up to 5 times/day (dispense 4–6 ml).	General preventive measures not necessary. Avoid unnecessary exposure to known cases.
Severe disease in infants. Pneumonia, seizures, encephalopathy. Case-fatality rate 0.5% in < 6-month-olds in U.S.	Supportive care. Hospitalize infants and those with severe disease. Erythromycin 40–50 mg/kg/day PO in 4 divided doses (maximum 2 g/day) for 14 days may ameliorate disease if started in catarrhal stage; thereafter limits spread to others. (Trimethoprim/sulfa unproved alternative.)	Respiratory isolation: for 5 days after initiation of erythromycin (3 weeks after onset of paroxysms if untreated). Prophylaxis: same 14-day antibiotic regimen as for treatment for household, day care, other close contacts of all ages. Immunization: universal for all children < 7 years old, usually given as DPT vaccine.[1]
Severe airway obstruction requiring intubation (uncommon). Rare: respiratory failure, pulmonary edema, pneumothorax/ mediastinum. Laryngotracheopneumonitis. Bacterial tracheitis.	Individualize based on symptom severity. Humidified air (oxygen if needed based on blood gases/oximetry). If hospitalized: racemic epinephrine (0.25–0.50 ml of 2.25% solution in 2–3 ml normal saline), anticipate rebound in 2 hours; single admission dose of dexamethasone phosphate (Decadron, Hexadrol) 0.6 mg/kg IM. Monitor for deterioration.	No specific recommendations.
Nonspecific, similar to common cold.	Avoid overtreatment. Humidification. Mild sedative at bedtime to prevent further attacks if certain of diagnosis.	No specific recommendations.
Abrupt airway obstruction with risk of death. Extraepiglottic metastatic infection rare despite bacteremia.	Advanced planning/rapid diagnosis. Intensive care. Endotracheal/nasotracheal intubation. Cefotaxime (Claforan) or ceftriaxone (Rocephin) or ampicillin/chloramphenicol. (Racemic epinephrine or corticosteroids *not* effective.)	*H. influenzae* type b vaccine should reduce incidence. Rifampin prophylaxis for *all* household contacts with at least one contact < 4 years old. Day-care: give if children < 2 years with 25 hours contact/week or if ≥ two invasive cases over 60-day period. Dosage 20 mg/kg (maximum 600 mg) daily by mouth × 4 days.[1]

Table 15.1. (continued)

Syndrome	Usual etiologic agents	Typical clinical presentation	Diagnosis
Bacterial tracheitis[7,9]	*S. aureus,* α-hemolytic streptococci, *H. influenzae* type b.	Combined symptoms of croup and epiglottitis. Slow or rapid clinical progression following croup-like onset; high fever, toxicity, stridor, bandemia.	Direct laryngoscopy and bronchoscopy. Usually normal epiglottis but subglottic narrowing, purulent tracheal secretions. Positive tracheal cultures.
Bronchiolitis[10]	RSV (50–70%), parainfluenzavirus and adenoviruses.	Age ≤ 2 years (especially ≤ 6 months) prodrome of coryza, fever to 40°C (104°F), gradually deepening cough (may be paroxysmal and induce emesis, no whoop); then dyspnea, tachypnea, prolonged expiration, chest wall retractions, wheezing and/or rales/rhonchi (apnea possible in young infants). Chest film: hyperinflation, multiple interstitial infiltrates.	Clinical and epidemiologic grounds (see text). Nasal wash for viral culture and rapid antigen tests for RSV.

Source: References 1–5.

but persistent sequelae include paralysis, seizures, cranial nerve palsies, and hydrocephalus.[16] Acute cerebellar ataxia by direct mumps virus invasion may occur.[18] Hearing loss—transient or permanent and often unilateral—may result, often in the absence of encephalitis. Hearing should be tested following mumps.[19]

Mumps orchitis[20] is rarely seen in prepubertal boys but occurs in 14% to 35% of adolescents, is generally unilateral, and follows parotitis (if present) by 4 to 8 days (up to 6 weeks); the average duration is 7 to 10 days. Symptoms and signs include gradual testicular pain and swelling, and often fever. Epididymitis or reactive hydrocele may occur. Impaired fertility occurs in 7% to 13% of cases, but complete sterility is infrequent.

A self-limited monoarticular or migratory polyarthritis may occur 1 to 3 weeks after parotitis (if present). Fever is often present along with variable elevations of the erythrocyte sedimentation rate (ESR).[21,22] Other rare complications of mumps include thyroiditis, nephritis, pancreatitis, transient electrocardiographic changes, myocarditis, mastitis, and pelvic pain in females.[14,16]

Management and Prevention

Treatment of viral parotitis, including mumps, is supportive and may include analgesics/antipyretics, fluids, and rest. Bed rest and medications have not been shown to prevent mumps orchitis,[14,20] but rest, scrotal elevation, ice packs, and antiinflammatory medications may ease scrotal symptoms.

Mumps patients should have respiratory isolation and be excluded from school or day-care until all manifestations have cleared. Generally, this period lasts 9 days after the onset of parotid swelling.[1] Mumps immune globulin is of no value. Mumps vaccine[1,16] does not prevent infection following known exposure but may be given in this setting to protect against subsequent exposures. Live mumps vaccine is usually given in a two-step regimen with measles and rubella vaccines (MMR).

Kawasaki Disease

Kawasaki disease (mucocutaneous lymph node syndrome) is an acute vasculitis that primarily affects infants and young children. It must be included in the differential diagnosis of fever, cervical adenitis, acute exanthems, and other mucocutaneous diseases. The etiology is unknown, but infectious agents or environmental factors are suggested as primary causes or cofactors.[23–25] Cases are most prevalent during late winter and early spring and may be epidemic. There is little to suggest person-to-person spread. Most cases occur in children less than 5 years of age; it is rare in infants under 6 months. The male/female ratio is 1.5:1.0. It occurs worldwide and is most prevalent in persons of Oriental descent. The recurrence rate is 3%, and the rate of disease in siblings is 1% to 2%.

Clinical Presentation and Diagnosis

Kawasaki disease is diagnosed on the basis of 5 days or more of fever *and* at least four of the following principal clinical features: (1) changes in peripheral extremities (acute: erythema and edema of the hands and feet; convalescent: membranous desquamation of the fingertips); (2) polymorphous exanthem; (3) bilateral, painless, nonexudative conjunctival injection; (4) changes in lips and oral cavity, such as erythema and cracking of lips, strawberry tongue, diffuse injection of oral and pharyngeal mucosa; and (5) acute, nonpurulent cervical lymphadenopathy (≥ 1.5 cm in diameter), usually unilateral.[23–25] Patients with fever and fewer than four other principal symptoms are considered to have Kawasaki disease if coronary artery disease is detected by two-dimensional echocardiography or coronary angiography. Young children particularly may have the disease without fulfilling all criteria.

A typical case[23] usually begins with fever up to 40°C (104°F) for 4 to 5 days to 4 weeks, the onset of which is followed shortly by bilateral conjunctival injection and often uveitis. A generalized morbilliform or urticaria-like rash appears within 5 days of illness, characterized by perineal area desquamation and erythema of the lips, pharynx, tongue, and hands and feet (with induration). Finger and toe desquamation usually begins 10 to 20 days after onset of fever.

Other noncardiac signs and symptoms that may be present, by system, include the following.[23–25]

Complications	Management	Prevention
Respiratory arrest.	Rapid bronchoscopy/diagnosis. Intensive care. Endotracheal intubation or tracheostomy. Antibiotics.	For *H. influenza* type b, see above.
Apnea/respiratory failure most likely first 48–72 hours after onset of dyspnea. ? Asthma later in life. Association of RSV with SIDS, CNS disorders, myocarditis, heart block.	Supportive care, hydration, monitor respiratory status. Oxygen. If hospitalized consider ribavirin 20 mg/ml or water small droplet aerosol by hood/tent/mask 12–18 hours/day × 3–7 days *if:* significant underlying cardiopulmonary disease or immunosuppression *or* severe illness (or at risk for severe illness, e.g., < 6 weeks old) *or* prolonged illness that may be detrimental (e.g., neurologic or metabolic disease). Ribavirin most beneficial in ventilated patients but expertise required.	Contact isolation for young children/infants. Large droplet precautions, careful handwashing and eye/nose goggles in the hospital. Breast-feeding seems to lower incidence.

Gastrointestinal tract: vomiting, diarrhea, abdominal pain, gallbladder hydrops, ileus, mild jaundice, or transaminase level elevation

Blood: elevated ESR, positive C-reactive protein (CRP), leukocytosis with left shift, mild anemia, thrombocytosis (after 2–3 weeks)

Urinary tract: sterile pyuria, proteinuria

Skin: transverse furrows of fingernails during convalescence, peripheral gangrene

Respiratory tract: cough, rhinorrhea, pulmonary infiltrate

Musculoskeletal system: arthralgias, arthritis

Neurologic system: aseptic meningitis, irritability, facial palsy, hearing loss.

Cardiovascular Complications

Early in the disease, signs of myocarditis become apparent in 25% of patients, including tachycardia, gallop rhythm, pericardial effusion, and mitral or aortic valve insufficiency. The major feature affecting the otherwise excellent prognosis in Kawasaki disease is coronary artery involvement. Coronary dilatation may be noted as early as 6 days to 4 weeks after onset of fever. Frank coronary artery aneurysms occur in 15% to 20% of patients and may be identified by skilled echocardiography in at least 95% of cases. Fortunately, more than 50% of aneurysms resolve within 1 year.[23]

Management

Goals of management are control of the acute inflammatory process and prevention of coronary artery involvement. Aspirin 100 mg/kg/day in four divided doses should be initiated once the diagnosis is tentatively made. After resolution of fever and other inflammatory signs, aspirin is continued at a single daily dose of 3 to 5 mg/kg/day for its antiplatelet effect. This regimen may be discontinued in 6 to 8 weeks if no coronary involvement is present. Aspirin is continued at 3 to 5 mg/kg/day for at least 1 year if coronary involvement is present (indefinitely if aneurysms persist). Dipyridamole (Persantine), 3 to 6 mg/kg/ day in three divided doses, is used if aspirin is not tolerated.

A single dose of intravenous immune globulin 2 mg/kg over 10 hours should be administered.

An echocardiogram should be obtained as soon as the diagnosis of Kawasaki disease is suspected and repeated at 4 weeks and at 2 months after onset. All patients with significant coronary artery disease should be followed initially and then long term in conjunction with a cardiovascular team experienced in treatment of Kawasaki disease complications. Guidelines for such follow-up are established.[23] The overall prognosis is good. The case-fatality rate approximates 0.4%. Those with large aneurysms are the most at risk.

Viral Exanthems

Numerous viruses are capable of producing exanthems in children. The major entities are summarized in Table 15.2.[26–31] Like mumps (discussed above), each of these illnesses may be subclinical, and each occasionally produces arthritis, encephalitis, and other significant sequelae. The diagnosis is largely clinical, but some overlap does occur (e.g., mild forms of measles resembling other exanthems and roseola-like illnesses caused by enteroviruses and other agents). Laboratory tests that may be helpful or diagnostic[1,27] include the following: immunoglobulin M (IgM) or serial IgG antibody titers for measles and rubella; viral cultures for rubella; IgM titers for parvovirus B19; Tzanck smear or culture of vesicles for varicella-zoster/herpes virus (herpes virus 6 serology being tested).

Other childhood viral exanthems, with and without fever, include the maculopapular, petechial, or vesicular exanthems of enterovirus and adenovirus infections. They include hand-foot-mouth disease (coxsackievirus), which presents as macules and vesicles of the hands and feet following a prodrome and preceding herpangina lesions (see above). A hepatitis B subtype may produce symmetric, flat-topped erythematous papules of the face, buttocks, and extremities (Gianotti-Crosti syndrome) in young children.[27] Zoster, characterized by vesicles and bullae on an erythematous base in dermatomal distribution, may occur in children of all ages following acquisition of varicella-zoster virus. Viral exanthems must be differentiated from (1) bacterial (e.g., scarlet fever), rickettsial, and parasitic infections; (2) the

Table 15.2. **Major Exanthem-Producing Viral Diseases During Childhood**

Disease	Agent	Incubation period	Transmission	Period of contagion	Description of rash	Distribution	Duratio
Measles (rubeola)	Measles virus (paramyxovirus)	8–12 Days until symptoms; avg. 14 days until rash	Direct contact with infectious droplets; airborne spread	3–5 Days before rash to 4 days after	Purplish-red, maculopapular, blotchy	Generalized after distal spread from hairline, includes palms/soles	4–7 Days
Rubella (German measles)	Rubella virus (togavirus)	14–23 Days	Direct or droplet nasopharyngeal secretion contact	7 Days before rash to 14 days after (up to 1 year if congenital)	Diffuse, discrete, reddish-pink, macular	Starts on face, spreads distally to trunk and extremities	3–4 Days
Roseola (exanthem subitum)	Human herpesvirus 6	5–15 Days	Unknown; 95% of patients are 6 months to 3 years old	Unknown (probably maximal during febrile phase)	Rose-pink macules and papules	Starts on trunk, spreads to head and extremities	Few hours to 2 days
Erythema infectiosum (fifth disease)[28]	Parvovirus B19	4–14 Days	Presumed respiratory secretions and blood; school/day care outbreaks occur (attack rates up to 50% in households, 60% in schools)	Before rash appears (≥ 7 days after illness if aplastic crisis)	Intense erythema of cheeks (perhaps ears) with circumoral sparing ("slapped cheeks") often followed in 1 day by symmetric, reticulated erythematous maculopapular rash	Body rash starts on extremities (not palm/soles), spreading centrally or caudally (may be atypical/ rubelliform)	Cheeks: 1–4 days Body rash: fluctuation with environmer changes for weeks
Chickenpox	Varicella-zoster virus (a herpesvirus)	11–22 Days	Direct contact with lesions or airborne droplets	1–2 Days before rash to 5 days after (and for duration of vesicles)	Scattered erythematous macules that vesiculate, rupture, and crust (often all types of lesions seen at presentation)	Progressive, diffuse, includes scalp	Several days

Source: References 1, 26, 27.

Clinical presentation		Course	Complications	Management	Congenital infection	Prevention
Fever	Other symptoms					
Moderate to high, 5 days or more	Conjunctivitis, coryza, cough, Koplik's spots (white spots on red oral mucosa opposite first/second molars)	Fever, cough, conjunctivitis starts first; rash appears on days 3–7; mild, modified course seen if partial passive immunity; atypical if older killed vaccine used	Otitis media, bronchopneumonia, encephalitis, subacute sclerosing panencephalitis	Supportive	Preterm labor, miscarriage, low birth weight	Live attenuated measles vaccine usually given as MMR. Recommendations for special situations available.[1] For exposed susceptibles: measles vaccine; consider immune globulin (IG) 0.25 ml/kg IM for normal hosts (within 6 days)
Mild	Headache, malaise, coryza; postauricular, suboccipital, posterior cervical adenopathy; polyarthralgias (arthritis); palatal petechiae (in some)	Starts with adenopathy and malaise; fever and rash begin on day 3	Encephalitis, thrombocytopenia	Supportive	Risk of fetal infection and congenital rubella syndrome (fetal death, prematurity, congenital defects)	Live rubella vaccine usually given as MMR (see mumps, above)
High, continuous up to 40.6°C (105.3°F)	Coryza, red tonsils, otitis media, lymphadenopathy; WBC count normal or low	Abrupt fever and irritability for 3–5 days; rash starts with resolution of fever (rash may be absent)	Febrile seizures, encephalitis	Supportive, control of fever	None reported	None
Mild or none	Arthralgias/arthritis; may have mild respiratory symptoms without rash; may be asymptomatic	Rash and other symptoms jointly may follow prodrome of fever, headache, malaise, myalgias	Chronic anemia in immunodeficient patients; aplastic crisis if chronic hemolytic anemia (often fever, malaise, myalgias without rash); hemophagocytic syndrome[29]	Supportive; consider IV immunoglobulins if chronic infection in immunodeficient patients	Fetal hydrops and death: risk if proved maternal infection (< 20 weeks' gestation) < 10%; risk from occupational exposure < 1%. Anomalies not associated.	Good hygiene and handwashing. Routine occupational exclusion of pregnant women not recommended (avoid aplastic cases)
Mild or none	Moderate to intense pruritus, mucous membrane lesions, arthritis, thrombocytopenia, hepatitis	May have mild constitutional prodrome; rash, fever, other symptoms appear concurrently	Bacterial superinfection, otitis media, Reye syndrome, meningitis, encephalitis, glomerulonephritis, cerebellar ataxia, transverse myelitis, pneumonia	Supportive. Avoid salicylates. Symptomatic relief for pruritus (antihistamines, calamine, colloidal oatmeal baths). IV acyclovir if immunocompromised; oral acyclovir 20 mg/kg (max. 800 mg) 4 times daily for 5 days reduces fever, lesions, constitutional symptoms if started within 24 hours of rash[30], but may be best reserved for > 14 years or high risk.[31] Local treatment for oral lesions as for herpangina (Table 15.1)	Maternal infection < 20–28 weeks' gestation may yield congenital anomalies. Maternal infection within 5 days of delivery may yield severe neonatal disease.	Varicella-zoster immune globulin (VZIG) for all susceptible exposed persons at risk for progressive disease—follow guidelines[1]

rash of mononucleosis following amoxicillin treatment; (3) drug- and toxin-mediated eruptions; and (4) other dermatologic disorders.

Viral Gastroenteritis

Viral gastroenteritis is the most common cause of acute childhood diarrhea in the United States, with rotavirus the most frequent agent in young children.[32] Annual rotavirus epidemics begin in Mexico during late fall and progress systematically across the continent, reaching the Northeast by April.[33] Nosocomial and day-care center outbreaks are common. Although rotavirus infections occur most often in children 6 months to 2 years of age, all ages are affected, including one-third of the parents of affected children. The virus destroys enterocytes of the upper jejunal villi, which usually induces a secondary lactose intolerance of up to 3 weeks' duration. As with most viral causes of gastroenteritis, the resulting diarrhea is usually watery and nonbloody; and fever, if present, is mild. Three days of vomiting usually precedes or accompanies 3 to 10 days of diarrhea, which may result in considerable dehydration. Fever, abdominal pain, and respiratory symptoms may also occur.[1,32]

Norwalk-like viruses cause community and common source outbreaks in school-age and older children and adults. Symptoms last 12 to 60 hours, with nausea (predominant symptom), vomiting, and diarrhea followed in up to half of the children by fever, chills, headache, myalgias, and malaise. Adenovirus type 40 and type 41 infections peak in children less than 2 years of age and cause a 1-week rotavirus-like illness in which diarrhea predominates and vomiting is profound.[1,32] Respiratory symptoms occur in approximately 70%, but this proportion is similar to that seen with other viral gastroenteritides.[34] Other viruses cause a few cases of gastroenteritis.

Diagnosis

When infants and children are seen in the office with mild symptoms and historical and physical findings consistent with one of the above viral syndromes, no special diagnostic tests are indicated.[35] Certain historical factors, such as the travel and exposure history, may suggest other nonviral etiologies. Bloody diarrhea suggests invasive enteric bacteria, pseudomembranous colitis, *Entamoeba,* or even Crohn's disease in an adolescent.[35,36] The absence of fetal leukocytes on the stool white blood cell (WBC) stain[37] supports the diagnosis of viral gastroenteritis but is also seen with enterotoxigenic bacterial and parasitic infections. Stool cultures should be reserved for patients with historical evidence of bacterial infection, high fever, blood or mucus in the stool, or a positive stool WBC stain.[35] Ova and parasite examinations are done if historical evidence or prolonged diarrhea is present. Specific viral diagnoses may be best supported by commercial antigen detection kits for rotavirus and adenovirus, and by acute and convalescent serum titers for Norwalk viruses. Viral tests are probably most useful for confirming the etiology early in outbreaks, for severe cases, and for suspected disease at unusual ages or seasons.

Aside from diagnostic clues, the physical examination should focus on estimation of the hydration and hemodynamic state (mucous membrane moisture, tears, skin turgor, and fontanel, orthostatic, or mental status changes). Significantly dehydrated children, especially infants, should be hospitalized, along with those with severe illness or electrolyte abnormalities.

Management

Oral rehydration is the mainstay of outpatient treatment of viral diarrhea, and vomiting is not a contraindication (use small, frequent administrations). Commercially available preparations (e.g., Pedialyte) are acceptable,[36] and rice-syrup solids based preparations (e.g., Ricelyte) appear to decrease stool volume and promote greater fluid and electrolyte absorption compared to glucose-based ones.[38,39] Breast-fed infants may be managed by continuing breast-feeding and supplementing with oral rehydration solutions. Rehydration with clear liquids such as soft drinks, gelatin water, and apple juice is not recommended owing to the high carbohydrate, high osmolality, and low electrolyte composition of these fluids. Diluting such items into commercial solutions, however, may be required so older infants and children accept the rehydration solution. Caffeine, plain water, and excessive use of soups (high sodium content) must be avoided.[36] Early refeeding of solids (within 24 hours), particularly formula, to non-breast-fed infants appears to be safe and beneficial. Many experts choose lactose-free formula for the first 2 to 3 weeks, whereas others advocate this practice only if reintroduction of milk or regular formula exacerbates diarrhea.[32,36,40] A BRAT diet (bananas, rice, applesauce, and toast) may be useful as the first solids fed after the onset of childhood diarrhea. Specific antidiarrheal agents are generally not indicated.

Most agents of viral gastroenteritis are spread by the fecal-oral (and possibly the respiratory) route. Contaminated water and fomites may be important. Prevention involves good hygiene, sound advice for travel (care in the use of water and food), boiling of shellfish, and meticulous handwashing.

Pinworms

Humans are the only natural host of the pinworm, *Enterobius vermicularis,* a 1-cm white, thread-like helminth that is responsible for some 42 million current infections in the United States. It is most common in school-age children, regardless of socioeconomic status, and is most prevalent among family members, in institutions, and in areas of crowding. Oral ingestion of eggs begins a 15- to 43-day life cycle that involves temporary attachment of larvae in the duodenum, habitation and copulation of adult worms in the cecum and adjacent gut, and migration of gravid females out of the anus at night to deposit thousands of eggs in the perianal and perineal regions. Although larvae may reenter through the anus, reinfection of the patient and infection of others generally occurs via the fecal-oral route, most commonly under the fingernails of the patient. Linens and furry animals may act as fomites.[37,41,42]

Clinical Presentation and Diagnosis

A large proportion of patients are asymptomatic, and the classic symptoms of perianal itching, restless sleep, and irritability may be no more common among infected than uninfected children. Occasionally, the parasite migrates and causes vulvovaginitis-urethritis (most common), salpingitis, prostitis, and bowel, liver, and other organ system disease. Anorexia, weight loss, and personality changes (due to the misconceived stigmata associated with having pinworms) are sometimes seen. Unless peritoneal invasion occurs, eosinophilia is not present.

Parents may observe worms on an infected child by nocturnal flashlight examination of the anal verge, but the parasites may be confused with white thread. The *cellophane tape test* detects 50% of infections on the first examination and 99% if five examinations are done. A clear piece of cellophane tape is placed against the areas of the perianal region early in the morning, placed face down on a clean glass slide, and brought to the physician's office for microscopic examination.[37,41,42]

Management

Because pinworm infection spreads within families and may be asymptomatic, initial treatment of all family members is best. Medication kills only adult worms, so retreatment of symptomatic individuals 2 weeks after the initial therapy may improve cure rates. Mebendazole 100 mg (Vermox chewable tablets) in a single dose for adults and children produces the fewest side effects (rarely, abdominal pain/diarrhea) but should not be used during pregnancy. Pyrantel pamoate (Antiminth) suspension, 11 mg/kg in a single dose (maximum 1 g) is an effective alternative but may cause significant gastrointestinal side effects.[42] Preventive measures such as frequent hand and fingernail washing, avoidance of digit sucking, and decontamination of clothing, sleeping quarters, and toilet seats may decrease reinfection but may not justify the associated increased psychological trauma and stigmata associated with pinworms.[41]

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