Identifying Infectious Rashes in Children

With the typical child contracting many rashes, it is important for the family physician to know which rashes are infectious and which are not.

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Rash is the most common condition with which children present, alongside a parent, to their physician’s office. There are multiple types of rash in childhood including infectious, allergic, contact-reactions, autoimmune and neoplastic. This article will focus on those rashes that occur in children outside the neonatal period, secondary to an infectious etiology.

Infectious rashes can be subdivided into those caused by viruses, bacteria and other miscellaneous infectious agents. As is true of most infectious conditions in children, the most common etiology for infectious rash is a viral infection.

To diagnose the etiology of a rash, a complete history and physical examination is required. There is a “story” to every rash. The history of the rash should focus on rash progression (Table 1), associated symptoms (Table 2) and exposures (Table 3).

Viral Exanthems

Maculopapular Rashes
Rubeola/measles. Measles used to be an epidemic disease. Fortunately, with immunization, it is now seen infrequently. The incubation period for rubeola after contact is eight to 12 days to the onset of prodromal symptoms.
Infectious Rashes

and 14 days to onset of rash. The prodrome is three to five days in duration, and includes enanthem (Koplik spots), fever, cough, coryza and conjunctivitis.

The rash of measles begins as macular and progresses to maculopapular (with or without petechiae). In severe cases, a confluent rash can occur, which includes the palms and soles. There is slight pruritis associated with the rash. The rash begins at neck, hairline, behind ears and the posterior cheek. The characteristic spread is:

- Day 1: Face, neck, upper arms, upper chest;
- Day 2: Back, abdomen, entire arms, and thighs;
- Day 3: Feet and begins to fade on face;
- Days 4 and 5: Symptoms subside; desquamation and discoloration occur after the rash fades.

Rubella. This has an incubation period of 14 to 23 days after contact. The prodrome is of mild catarrhal symptoms for a few days and lymphadenopathy at least 24 hours prior to rash. The rash is maculopapular, with areas of flushing. The rash has rapid evolution and rapid fading (it classically clears by the third day). Spread of the rash is from the face to the trunk. There may or may not be associated mild pruritis.

Parvovirus B19. The incubation period for parvovirus is four to 14 days (and could be as long as 21 days). There is a brief non-specific illness with fever, malaise, myalgias and headache as a prodrome. The facial rash is intensely red with a “slapped cheek” appearance, with or without circumoral pallor. There also is a symmetric maculopapular lacy rash on the trunk. The rash then moves peripherally to the arms, thighs and buttocks. The rash often is pruritic.

Roseola/HHV-6. The incubation period for HHV-6 is nine to 10 days. The prodrome is a high-grade fever for three to seven days. The rash is erythematous and maculopapular, lasting hours to days.

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Adenoviruses. These cause a variety of different manifestations including: respiratory tract infections, gastroenteritis, conjunctivitis and cystitis. These viruses also can cause a maculopapular rash in patients.

Epstein-Barr Virus and Cytomegalovirus. These can cause mononucleosis-like syndromes and lead to a maculopapular rash after treatment with antibiotics (especially with amoxicillin).

Vesicular Rashes
Herpes Simplex Virus (I and II). These viruses lead to a characteristically painful vesicular lesion that can be oral-labial (herpes simplex virus type I [HSV-I] majority) or genital (HSV-II). The lesions can be predicted by the sensation of tingling prior to a breakout. HSV also can cause gingivostomatitis, eczema herpeticum, herpetic whitlow, conjunctivitis and keratitis. There are primary infections with subsequent secondary reactivations from the nerve root, which tend to be less severe than the primary outbreak.

Varicella zoster (chicken pox and shingles). Primary infection caused by the varicella zoster virus causes chicken pox. The incubation period is 14 to 16 days (from 10 to 21 days after contact). The rash of chicken pox is generalized, pruritic and goes through different stages. The rash is initially maculopapular, then goes to a vesicular stage and then crusts over. The patient with chicken pox often will have different stages of the rash presenting at once. Hemorrhagic chicken pox occurs more commonly in the immunocompromised host. Secondary bacterial infections of the chicken pox lesions (e.g., cellulitis) are the most common complication. Necrotizing fasciitis, an infection secondary to Group A Streptococcus, although rare, is highly associated with chicken pox. The varicella zoster virus becomes latent in the dorsal root ganglion. Reactivation of the virus leads to herpes zoster or shingles. The rash appears along the dermatome supplied by the dorsal root that was reactivated. The rash is vesicular and is painful. The pain can persist beyond one month (post-herpetic neuralgia).

Enteroviruses (Coxsackie A16 and enterovirus 71 [hand, foot and mouth disease]). These summer viruses present as vesicular lesions with a predilection for the
hand, feet and mouths of affected patients. The illness is self-limited, with total recovery. Lesions can be painful enough to prevent normal eating and drinking in the affected patient.

**Other Rashes**

*Molluscum contagiosum.* The poxvirus causes molluscum contagiosum. The lesions are small, discrete and flesh-colored to translucent dome-shaped papules, which often have central umbilication. The lesions occur most commonly on the trunk, face and extremities, but can be generalized.

*Warts (human papilloma virus).* The human papilloma viruses lead to epithelial tumors (warts) of skin and mucous membranes. The lesions appear as dome-shaped, with conical projections that give a rough appearance. The lesions tend to be asymptomatic and self-limited.

**Bacterial Infections**

*Staphylococcal Infections*

*Staphylococcus aureus* is responsible for causing rashes in children. The conditions that staphylococcus can cause include:

- **Impetigo.** This lesion begins as a papule, which rapidly evolves into a vesicle surrounded by an area of erythema. Vesicular lesions give rise to pustules that gradually enlarge and then break down over four to six days to form characteristically thick crusts (golden). They occur at sites of non-intact skin. *Staphylococcus* can cause bullous and non-bullous forms of impetigo.

- **Cellulitis.** This is a painful, erythematous and indurated infection of the skin and soft tissues. It often presents after an initial injury to the patient’s skin, and is associated with lymphangitis and lymphadenopathy.

- **Staphylococcal scalded skin syndrome.** This syndrome is caused by toxins (exfo-
liative toxin A and B) released by certain *S. aureus* strains. Its initial presentation is of fever, generalized erythema and skin tenderness. Within 24 hours, affected skin gathers into folds, forming superficial blisters, and when the blisters rupture, the skin peels off in sheets, leaving a moist, painful, red base.

- **Staphylococcal toxic shock syndrome.** This is an acute, multisystem disease with high fever, hypotension, vomiting, abdominal pain, diarrhea, myalgias, non-focal neurologic abnormalities and an erythematous rash. The rash is diffuse, red and macular (sunburn-like) and appears within 24 hours. There may be associated hyperemia of the mucous membranes. There may be petechiae on days three and four of the illness. After resolution, there is associated desquamation, especially of the palms and soles.

**Group A Streptococcal (GAS) Infections**

GAS is responsible for the following conditions in children:

- **Impetigo.** GAS causes non-bullous impetigo associated with honey-crusted lesions on skin, particularly around the nares.

- **Scarlet fever.** This is caused by a strain of GAS that produces an erythrogenic toxin. The clinical manifestation includes fever, headache with or without vomiting, and facial flushing with marked circumoral pallor. The rash lasts four to 10 days, starts on the second clinical day of illness on the trunk and spreads to the extremities. The palms, soles and face are spared, and the rash has a sandpaper-like texture. The skin in the cutaneous folds of the neck and anterior groin may develop increased prominence and show as dark red lines (Pastia’s lines).

  Strawberry tongue may be present. One to two weeks after the onset of illness, the skin will begin to peel. This may last several weeks.

- **Cellulitis.** The description is similar to the description for *S. aureus*.

- **Erysipelas.** This is a deep infection of the skin, usually on the face, with marked lymphoid involvement. The patient...
usually febrile and the skin is doughy to the touch.

- **Rheumatic fever.** Erythema marginatum occurs very infrequently. Early in the disease it presents as pink macules over the trunk. Later, there is blanching in the middle of the lesions, with fusing of the borders and leads to a serpiginous-looking lesion.

- **Streptococcal toxic shock syndrome (STSS).** Patients develop rapidly progressive hypotension and multisystem organ failure.

- **Necrotizing fasciitis.** This is a deep-seated infection of the subcutaneous tissues resulting in progressive destruction of fascia and fat. It may occur as a component of STSS or among patients with no signs of shock/organ failure. It may or may not have muscle involvement (myositis).

**Meningococcal infections—meningococcemia/purpura fulminans**

*Neisseria meningitidis* causes a life-threatening condition known as meningococcemia, which is a blood-stream infection with the pathogen. The rash of meningococcemia is one that no one should ignore. The rash, however, can initially begin non-specifically as maculopapular or urticarial in nature. The classic non-blanching petechiae also can be the presenting rash. These can coalesce to develop into purpura (Figure 1.) The onset of this infection is abrupt, with fever, chills, malaise and rash. The misdiagnosis of this rash will lead to patient mortality and morbidity.

**Other Infections**

**Lyme disease (erythema chronicum migrans)**

Lyme disease is secondary to infection by the spirochete *Borrelia burgdorferi*. The spirochete is transmitted to humans by a tick bite. The skin is the initial target of infection and inflammation, which leads to the characteristic rash: annular rash occurs seven to 14 days after bite (from three to 32 days). The initial lesion is usually at the site of the bite, and may be uniformly erythematous or a target lesion with central clearing or central vesicular/necrotic areas (average diameter 15 cm).
In the secondary stage, skin lesions can appear, which are smaller and secondary to the migration of the spirochete.

**Syphilis**
This infection is sexually transmitted, secondary to the spirochete *Treponema pallidum*. In primary syphilis, a painless chancre occurs on the genitals with regional lymphadenopathy. In secondary syphilis, a generalized skin eruption that is maculopapular (scaling) occurs on the head, neck, palms and soles.

**Rocky Mountain Spotted Fever**
The spirochete *Rickettsia rickettsii* causes Rocky Mountain Spotted Fever. A tick, the natural host and reservoir, introduces the spirochete to humans by a bite. Initially, patients complain of headache, fever, anorexia and restlessness. The third day after a bite, a skin rash appears which, initially, is maculopapular on the extremities and then spreads over the entire body. The rash subsequently becomes more petechial or hemorrhagic in nature.

**Scabies**
This is a skin infestation by a mite. The rash is an erythematous papular eruption and can be vesicular in children under two years of age. The rash is intensely pruritic, especially at night. The skin manifestation is secondary to a hypersensitivity reaction to the proteins of the mite. Transmission of the mite is skin to skin. If scabies is suspected, one must look for the mite burrows. Bacteria may secondarily infect the bites.

**Kawasaki Disease (Atypical mucocutaneous lymph node syndrome)**
Kawasaki disease is of unknown etiology but infectious and post-infectious etiologies are postulated. Typical Kawasaki disease requires a five-day history of fever with four of the following five criteria presenting:

- Rash of any type (non-vesicular);
- Cervical lymphadenopathy more than 1.5 cm;
- Mucositis: dry, red cracked lips and strawberry tongue;
- Swollen hands and feet (especially tips), which progresses to peeling; and
- Bilateral non-purulent conjunctivitis.

Atypical disease may have fewer criteria present. Treatment for Kawasaki disease is to prevent or reverse coronary aneurysm formation.

Suggested Readings