You have recently encountered 4 children with a very distinct, well-demarcated, flat erythroderma of the facial area. You must determine whether this rash is due to erysipelas, which presents with an erythematous flat, painful, cellulitis-like rash most commonly on the face or lower extremities. The differential diagnosis in each of these cases also includes allergic manifestations such as a drug hypersensitivity, angioedema, or contact dermatitis; local reactions such as sunburn or other thermal burn; viral infections, such as fifth disease, etc; and bacterial infections such as cellulitis, early staphylococcal or streptococcal scalded skin, and early necrotizing fasciitis. You will also likely be tested by some children who will fail your initial “prudent” antibiotic choice, as these cases will illustrate.

CASE SCENARIOS

Case 1
A previously healthy white, female 6-month-old presents to your office with fussiness, a 3-day-long fever of up to 103°F, decreased appetite, rhinorrhea, excessive drooling, and pulling at her ears. The mother has also noted occasional vomiting without diarrhea, poor sleeping, and tremendous clinginess. The child is fully immunized; she is receiving no medications except for ibuprofen every 8 hours.

In the office, her temperature is 101°F, pulse rate 134, respiratory rate 24, and oxygen saturation 99%. When you examine her she is fairly distractible and cooperative for her age. Her dermatologic examination reveals the rash seen in Figure 1. Her tympanic membranes are normal, and because she has now become much less cooperative after you removed the cerumen from her ear canals, a cursory look at her posterior pharynx shows it is fairly reddened. The remainder of her examination is normal, including the lymph nodes, lungs, heart, abdomen, vaginal-rectal area, and range of motion of the extremities. Because she was so cranky and irritable, you obtain a complete blood count (CBC). While you are waiting on those results, something else bothers you about her examination.

Figure 1. A 6-month-old white female presents with 3 days of fever, crankiness, and a unilateral flat, non-tender, well-demarcated rash of the face only.

Case 2
A previously healthy white, female 8-month-old presents to your office on a September morning with abrupt 12-hour onset of irritability, fever to 102°F, no appetite, and no sleep the night before. She attends daycare, has had no gastrointestinal complaints, no tick bites, and has received no other medications except for an ibuprofen dose that...
Healthy Baby

morning. She is fully immunized and no other family members have been ill.

In the office her temperature is 101°F, pulse rate 144, respiratory rate 30, and oxygen saturation 97%. The nurse had an extremely difficult time obtaining her weight and her vital signs due to her crying. Her physical examination reveals the well-demarcated rash on the face (see Figure 2). She is very difficult to examine and you can find no way to distract her from crying. She looks ill but does not appear toxic. The remainder of her physical examination is normal, including the pharynx, tympanic membranes, supple neck, cervical nodes, heart, lungs, abdomen, and extremities. The rash localized solely on her face is surprisingly warm, blanches readily, and may even be tender and indurated. Despite her low-grade fever and being nontoxic, she is very irritable, and your clinical index of suspicion urges you to consider this child as having an entirely different level of illness. Something vague really bothers you about her clinical examination.

Case 3

In August, a previously healthy, fully immunized, white, male 15-month-old who recently has been at the swimming pool presents with an acute 24-hour onset of a discreet flat erythroderma that is mildly tender and fairly well demarcated along the lower face, arms, and entire frontal trunk area. The mother says she had the baby’s face covered with a towel while he was sitting in the stroller at the swimming pool. She is not certain whether she used sunscreen. He has been regularly receiving his amoxicillin during the past 3 days for a previously diagnosed episode of acute otitis media (AOM) in your office. On this day, he has developed some crankiness and fever to 101°F, but she denies that he has had any vomiting, diarrhea, sore throat, tick bites, or exposure to Streptococcus pyogenes or Staphylococcus aureus from any family members.

On physical examination, he has developed the rash seen in Figures 3A and 3B. It is non-tender but very warm. His temperature is 100.5°F, pulse rate 110, and respiratory rate 20. His tympanic membranes are still reddened with a partial purulent air fluid level. His pharynx and neck are normal, as are his heart, lungs, abdomen, genitalia, extremities, and affect.

His CBC shows a leukocyte count of 13,200 cells/mm³ with normal hemoglobin and platelets. Your primary considerations are whether this is a photosensitivity reaction, drug reaction, or simple sunburn. Yet, something that caught your eye earlier really bothers you about his clinical examination.

Case 4

A previously healthy, fully immunized (with PCV7), white, female 7-year-old presents to your office with a 3-day-long, well-demarcated, tender facial erythroderma (see Figure 4, page 189). The rash is unrelated to any trauma, bites, or scratches. She has had low-grade fevers up to 102°F for the entire episode. Two days earlier, her initial leukocyte count was 13,300 cells/mm³; she was started on oral clindamycin to
cover the most common pathogens of nontraumatic facial and periorbital cellulitis: *S. aureus* and *Streptococcus pyogenes.* However, on this day, the rash has spread somewhat and is more tender; she appears more ill, has a right AOM, and a fever of 102.1°F. Her leukocyte count has increased to 17,540 cells/mm³. You are perplexed at the worsening of her cellulitis and wonder what therapeutic options you have now.

**CASE DISCUSSIONS**

**Case 1**

When you initially examined the baby, you noticed a red spot or blister in the posterior pharynx. You ask the mother if she would allow you to obtain a more thorough glance into the posterior pharynx. You position the baby face up on her mother’s lap, while you hold the infant’s head between your knees to obtain the least traumatic, most optimal evaluation. With a tongue blade firmly pressing down the tongue, you are able to see the multiple small, round, red-based, yellow punctuate lesions on the posterior palate.

You have confirmed that this child has herpangina, most likely caused by one of the enteroviruses, such as the common Coxsackie A16. You have not before seen this type of flat red rash associated with herpangina, as you are much more familiar with the more typical small vesicles, blisters, macule-papules, or even petechiae among the many dermatologic variations of the hand, foot, mouth disease. The child’s leukocyte count was reassuringly 4,700 cells/mm³, and the child appeared fairly happy and interactive when left alone. Subsequently, you reassured the mother, and asked her to force clear fluids and to use ibuprofen as needed for the fever and fussiness.

**Case 2**

Although the facial rash in this child is remarkably similar to the rash in Case 1, some clinical manifestations are diametrically opposite. Merely watching this infant in the office for 15 minutes, you note that the child never calms down, cries constantly, and cannot even be distracted. Relying on your clinical instinct, you decide to tell the mother that you think her child is actually very ill and that she needs to have a CBC, blood culture, and hospitalization for intravenous (IV) antibiotics until you can sort out the clinical syndrome. You fear that she may have something like a very early toxic shock-like syndrome, even though her blood pressure was normal. Somewhat confirming your suspicions, her white blood count is 21,500 cells/mm³ with 90% segmented neutrophils and normal hemoglobin and platelets.

You immediately send her to the nearest children’s hospital (which, if you are a rural practitioner, may be more than 30 miles away), and ask the hospitalist to consider this child foremost as having either erysipelas or very early toxic shock syndrome/scalded skin syndrome caused by either Group A *Streptococcus* (GAS) or *S. aureus*.

“The superficial cellulitis, erysipelas, referred to as *St. Anthony’s fire* in the Middle Ages, is characterized by the appearance of a bright erythematous plaque with a distinct, elevated border that sharply demarcates affected from unaffected skin. The lesion most often involves the face or lower extremity, although extensive involvement of the trunk has been noted. The involved skin is warm and tender and may have a peau d’orange appearance. Large tension bullae may be seen in the erythematosus zone. The patient generally appears toxic and is highly febrile, and rapid extension of the affected skin may occur over the course of hours.”

“Toxic shock syndrome” is defined by the 2012 AAP Red Book as: isolation of GAS from a sterile or non-sterile site, plus at least two of the following: renal impairment, coagulopathy, hepatitis, ARDS, erythroderma, or soft tissue necrosis. You obtain a blood culture, but you have observed that a pathogen is very rarely obtained by aspirating the leading edge of any cellulitis. You request that the infant immediately be started on both IV clindamycin to cover for methicillin-resistant *Staphylococcus aureus* (MRSA) and as a protein synthesis inhibitor to shut down the gram-positive exotoxin production, and IV ceftriaxone for more broad-spectrum coverage and more bactericidal cell wall inhibitor activity.

The child’s first 24 hours in the hospital are somewhat stormy when she develops an even higher leukocyte count, high fevers, and decreased urine output, requiring a few IV fluid challenges. Her blood culture grows GAS on day 2, but then her fever breaks, and the rash slowly begins to diminish. The child never develops the devastating “flesh eating” *Streptococcus* syndrome that you were quite concerned about. She subsequently does well, receiving a full 7 days of IV clindamycin for her GAS bacteremia, probable erysipelas, and possible very early toxic shock-like erythroderma.

Figure 4. A 7-year-old white female with a red flat and tender, well-demarcated rash on her right cheek for 3 days, who has been receiving oral clindamycin for the past 2 days.
**Case 3**

This toddler was not ill looking, so at first you presumed this was either an accidental sunburn, drug reaction, or virus-induced photosensitivity. However, you noted the few red spots on the lateral neck (see Figure 3A, page 188), thus you ask the mother to maneuver the child’s face upward so that you can obtain a better look at the neck, as seen in Figure 5A. You now know your diagnosis: this is likely a case of spreading intertrigo and folliculitis due to *S. aureus*. Although intertrigo of the neck is often caused by *Candida* or a mixture of bacteria, sometimes *S. aureus* can infect the area. This child’s rash is more akin to a staphylococcal “surgical scarlet fever” exotoxin reaction or erythroderma, rather than the typical sand-papery streptococcal scarlet fever rash that would have responded to amoxicillin. Also, you can barely perceive the fine papular rash on the chest upon closer re-inspection. Because the child looks nontoxic and is interactive with you, you doubt that this rash is an early staphylococcal scalded skin syndrome.

Thus, after obtaining a skin culture of the intertrigo, you elect to empirically treat the infant with oral clindamycin to cover for MRSA strain of *S. aureus* that is so highly prevalent in most communities. Within 48 hours the infant’s surgical scarlet fever and intertrigo has nearly resolved (see Figure 5B), and the culture grows MRSA.

**Case 4**

The condition of this school-age child worried you due to her persistent fever, more ill appearance, and her spreading facial cellulitis that was unresponsive to oral clindamycin. You are most likely dealing with either a different pathogen, such as clindamycin-resistant pneumococcus or a clindamycin-resistant MRSA strain. You discuss the immediate empirical therapeutic options with the mother — either broad-spectrum IV antibiotics in the hospital, or an initial outpatient trial for 24 hours with intramuscular (IM) ceftriaxone 50 mg/kg to measure the child’s response. The mother opts for an outpatient IM ceftriaxone trial, much to the dismay and tears of the youngster. When the child returns in 24 hours, she feels much better, her cellulitis is markedly improved, and her fever has resolved. Thus you presume that she probably had a buccal cellulitis most likely caused by a strain of resistant pneumococcus, such as serotype 19A or 6A, which is not included in her earlier PCV7 vaccine. Buccal cellulitis caused by *Haemophilus influenzae* type B was common in the 1970s, but is virtually unheard of at her age and in 2013. She receives 1 more dose of IM ceftriaxone with no further recurrence of the rash or fever.

**REFERENCES**