Chapter 14

PEDIATRIC CONDITIONS

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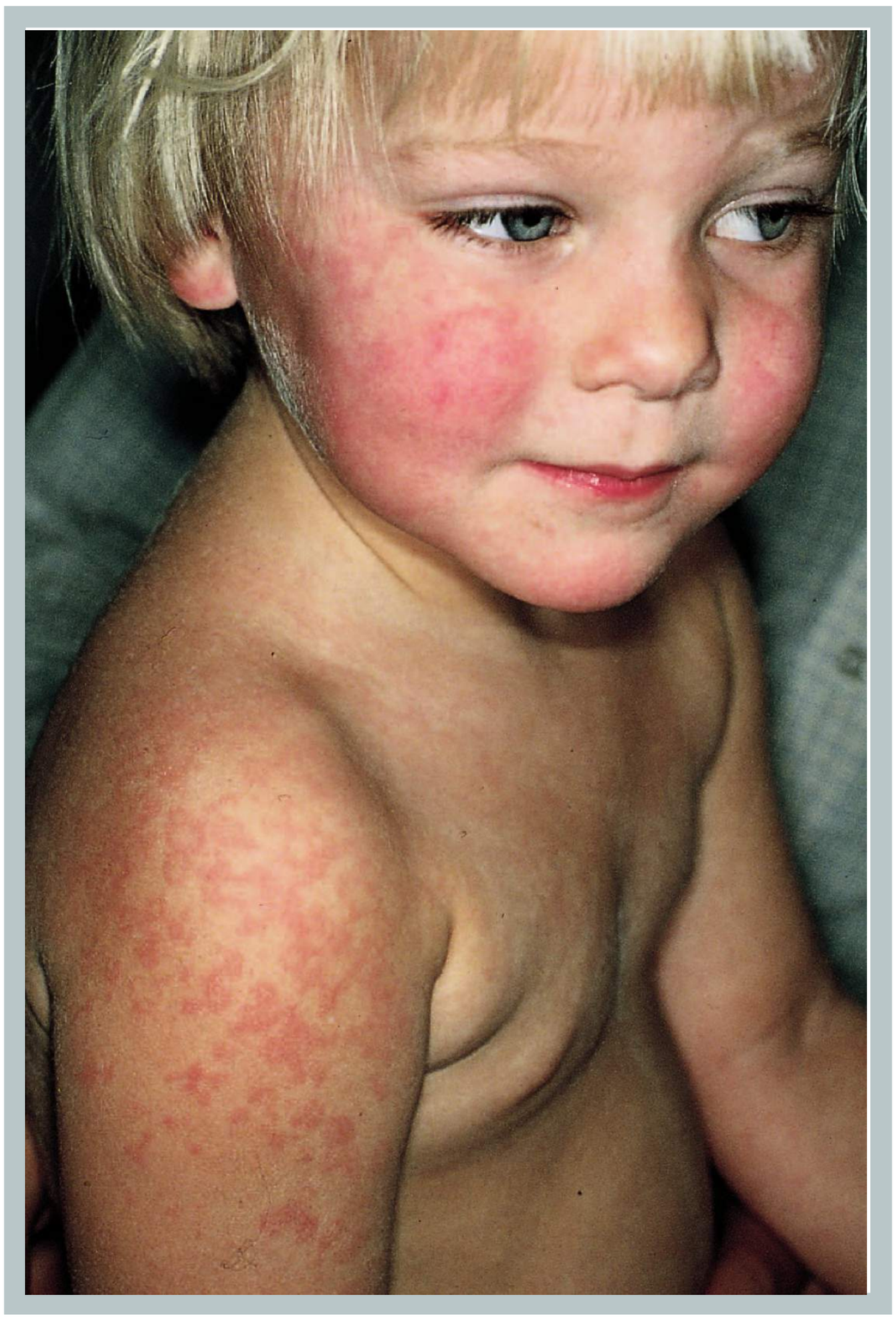


Fifth Disease. Toddler with the classic slapped-cheek appearance of fifth disease caused by parvovirus B19. Note the lacy reticular macular rash on the shoulder and upper extremity. (Photo contributor: Anne W. Lucky, MD.)

ERYTHEMA TOXICUM NEONATORUM

Clinical Summary

Erythema toxicum neonatorum is a benign, self-limited vesicopustular lesion of unknown etiology that occurs in up to 70% of term newborns. It is characterized by discrete, small, erythematous macules or patches up to 2 to 3 cm in diameter with 1 to 3 mm firm pale yellow or white papules or pustules in the center. The trunk and proximal extremities are predominantly involved. This rash usually appears within the first 24 to 72 hours of life but may be present at birth. The distinctive feature of erythema toxicum is its evanescence, with each individual lesion usually disappearing within 5 to 7 days. New lesions may occur in a waxing and waning fashion. The diagnosis is usually made based on the clinical appearance of the rash in an otherwise well-appearing neonate without any systemic signs of illness. Wright-stained slide preparations of a scraping from the center of the lesion demonstrate numerous eosinophils. Cultures from these lesions will be negative. The differential diagnosis includes neonatal acne, transient neonatal pustular melanosis, newborn milia, miliaria, infantile acropustulosis, neonatal herpes simplex, bacterial folliculitis, candidiasis, and impetigo of the newborn.

Management and Disposition

No specific therapy is indicated in the setting of a well-appearing newborn with normal activity and appetite. Parents should be educated and reassured about the evanescence of the rash. In cases where impetigo, Candida, or herpes infections are suspected, a smear from the center of the lesion and bacterial and viral cultures may be necessary to make a final diagnosis.

Pearls

1. Erythema toxicum is the most common newborn rash.
2. The lesions may present anywhere on the body but tend to spare the palms and soles.
3. Laboratory evaluation is unnecessary.

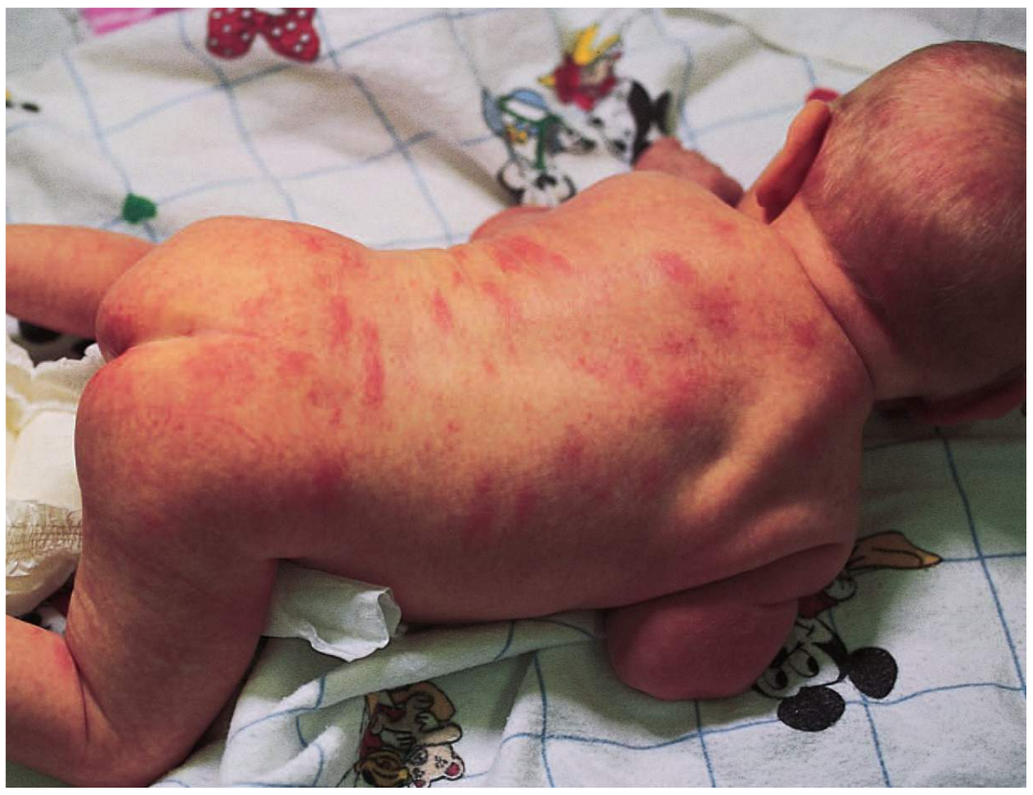


FIGURE 14.1 ■ **Erythema Toxicum.** Newborn infant with diffuse macular rash of erythema toxicum. (Photo contributor: Kevin J. Knoop, MD, MS.)

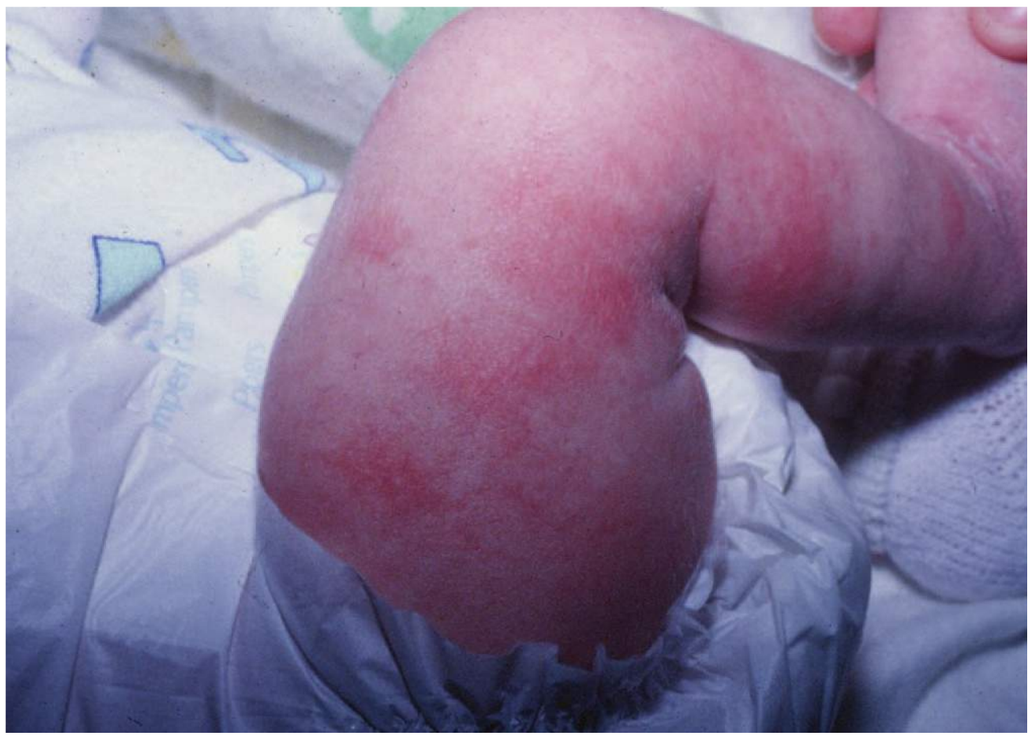


FIGURE 14.2 ■ **Erythema Toxicum.** Close-up of lower extremity of a neonate with erythema toxicum. (Photo contributor: Robert W. Hickey, MD.)

Clinical Summary

Nevus simplex (salmon patch) is the most common vascular lesion in infancy, present in about 40% to 60% of newborns. It appears as a blanching, slightly pink-red macule or patch most commonly on the nape of the neck, the glabella, mid-forehead, or upper eyelids. Lesions generally fade over the first 2 years of life but may become more prominent with crying or straining.

Management and Disposition

Parental education and reassurance can be helpful, but no immediate treatment is indicated. Pulsed dye laser may be considered for persistent lesions that are cosmetically undesirable.

Pearls

1. Salmon patches are composed of ectatic dermal capillaries.
2. Salmon patches appear symmetrically and cross the midline in contrast to the unilateral distribution of a port-wine stain.
3. When seen on the nape of the neck, this lesion is referred to as a stork bite or as an angel's kiss when appearing on the forehead.
4. About 5% of those appearing at the nape of the neck will remain permanently or recur.
5. Obtain imaging to evaluate for spinal dysraphism in patients with a lumbosacral nevus simplex and another lumbosacral abnormality (dermal sinus or pit, patch of hypertrichosis, or deviated gluteal cleft).

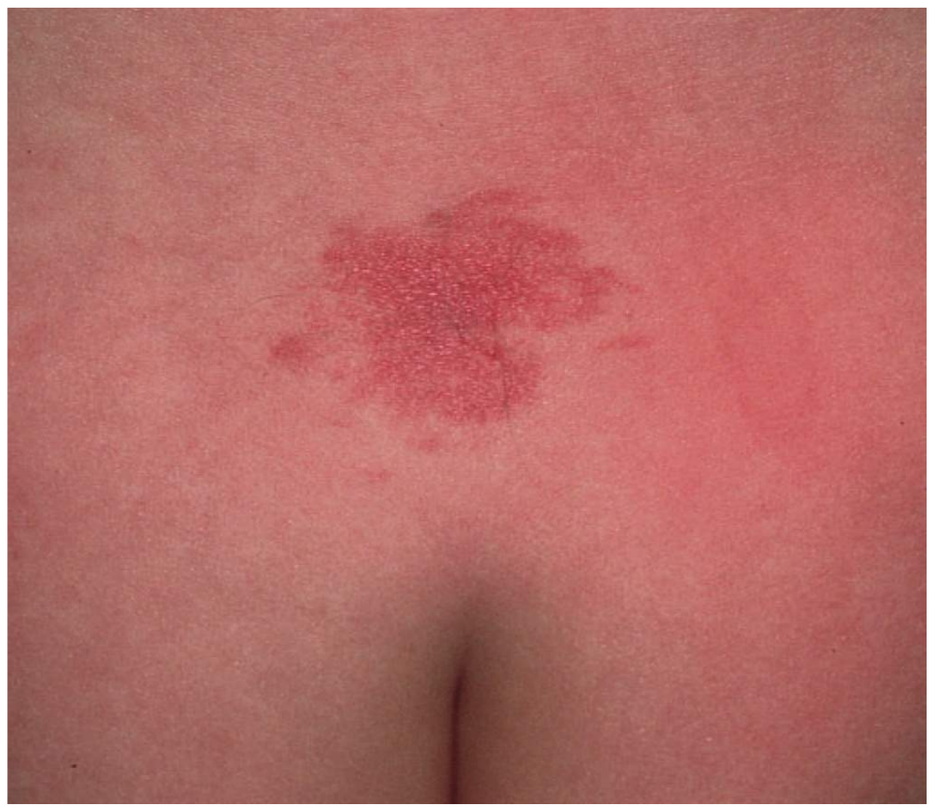


FIGURE 14.4 ■ **Salmon Patches.** Child with patch over lower back consistent with salmon patches. (Photo contributor: Anne W. Lucky, MD.)

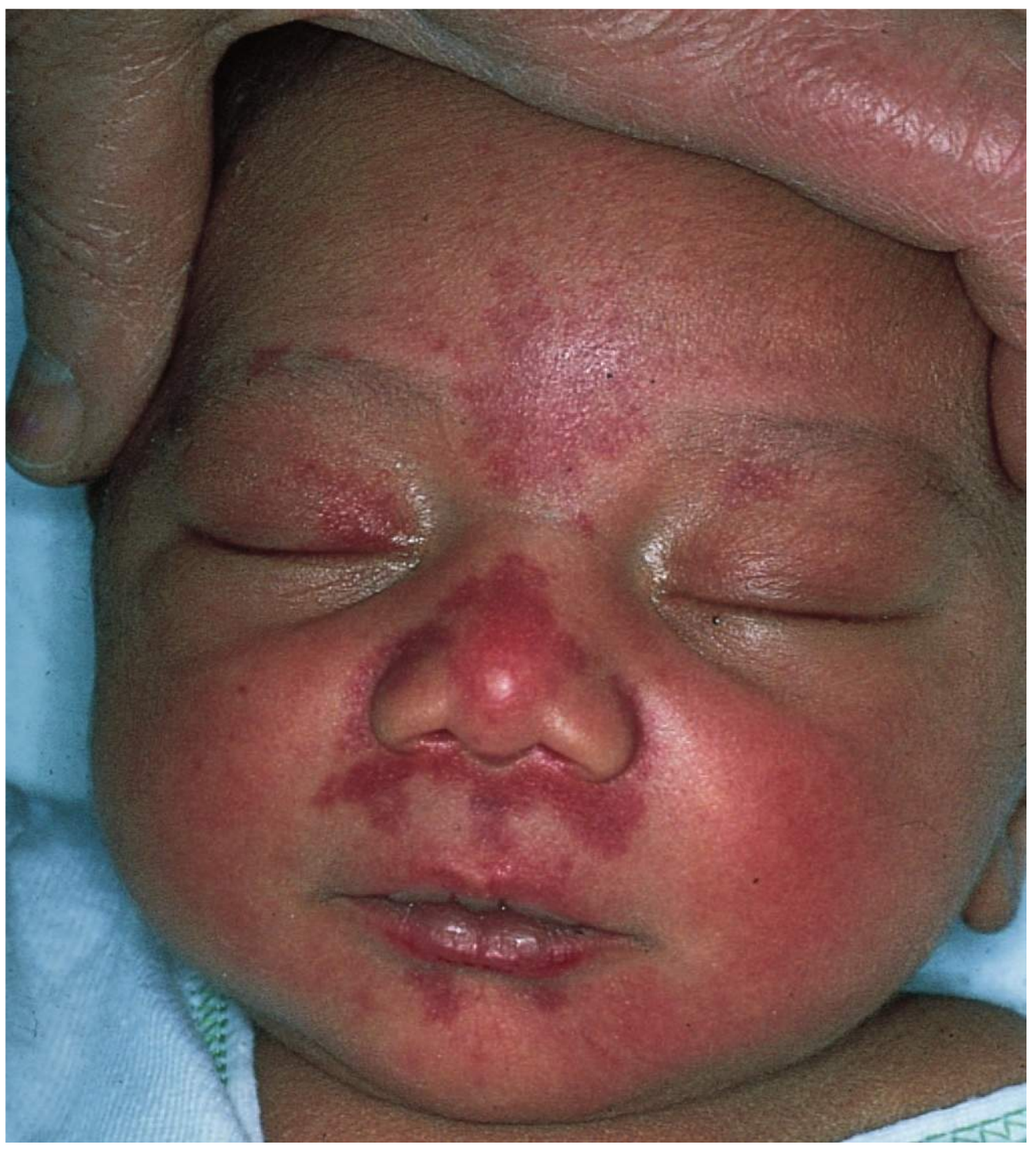


FIGURE 14.3 ■ **Salmon Patches.** Newborn with characteristic salmon patches over his face. (Photo contributor: Anne W. Lucky, MD.)

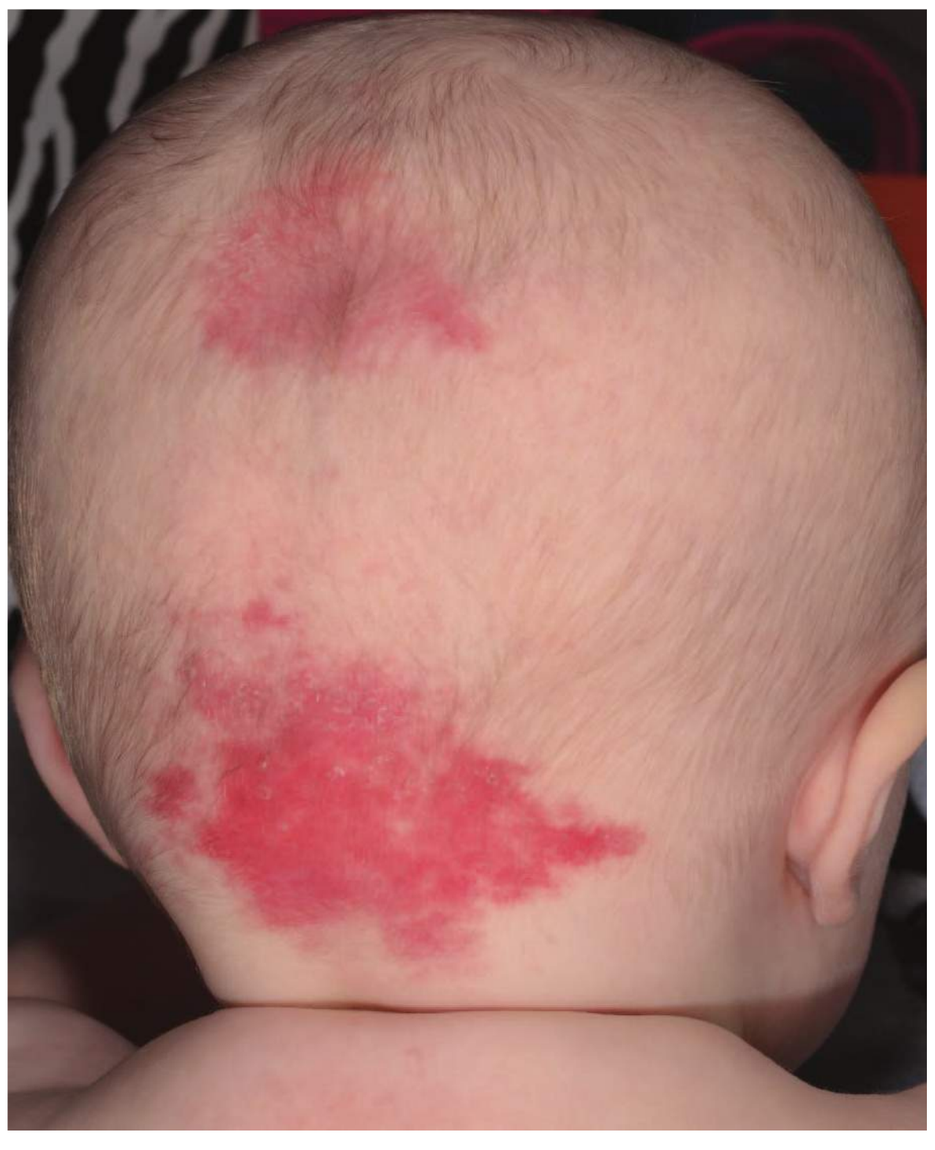


FIGURE 14.5 ■ **Salmon Patches.** Characteristic lesion on the nape of the neck is commonly called a stork bite. (Photo contributors: Katharine Hanlon and Kara Shah, MD, PhD.)

NEONATAL JAUNDICE

Clinical Summary

Neonatal jaundice is the yellowing of the skin, sclerae, and/or mucous membranes caused by bilirubin deposition. It occurs when total serum bilirubin is in excess of 5 mg/dL and progresses in a head-to-toe fashion as levels increase. Most cases of physiologic (<12 mg/dL) jaundice are self-limited without sequelae, and appear on the second or third day of life peaking between the third and fifth day. Preterm infants may peak later.

The increased bilirubin production in newborns is a result of turnover of fetal red blood cells, a temporary decrease in conjugation and clearance by the immature newborn liver, and increased enterohepatic circulation. Risk factors for indirect hyperbilirubinemia include maternal diabetes, prematurity, drugs, polycythemia, traumatic delivery with cutaneous bruising or hematoma, breastfeeding, and ABO (O mother and A/B infant) or Rh(D) incompatibility (Rh(D) negative mother and Rh(D) positive infant). Most infants with jaundice have no "disease" per se, but a careful history and organized approach is necessary to identify potentially pathologic causes. Kernicterus manifests in a multitude of irreversible neurologic abnormalities and is the long-term result of bilirubin-induced neurologic dysfunction secondary to extreme unconjugated hyperbilirubinemia which leads to neuronal death and pigment deposition in the basal ganglia and cerebellum.

Management and Disposition

Initial laboratory workup should include blood type, Coombs test, complete blood count (CBC) with smear for red cell morphology, reticulocyte count, and indirect and direct bilirubin levels. Although transcutaneous bilirubin measurement devices are widespread, they can underestimate total bilirubin at levels >15 mg/dL; therefore, a serum measurement is recommended for severely jaundiced newborns. Initial management should ensure adequate hydration and treatment of the underlying condition. The level of serum bilirubin at which to start phototherapy can be obtained from a standardized nomogram and is dependent upon the infant's postnatal age in hours, gestational age, and an assessment of risk factors. In general, the goal of phototherapy is to maintain the bilirubin level below 20 mg/dL. Exchange transfusion is considered if the serum level remains elevated (22-25 mg/dL) despite appropriate phototherapy.

Pearls

1. Onset of clinical jaundice in the first 24 hours of life strongly suggests the presence of a pathologic process.
2. Direct serum bilirubin concentration exceeding 10% of total serum bilirubin or 2 mg/dL suggests hepatobiliary disease or a metabolic disorder.
3. Bilirubin levels at which to initiate phototherapy or exchange transfusion should be modified for prematurity, sepsis, low birth weight, and other risk factors.

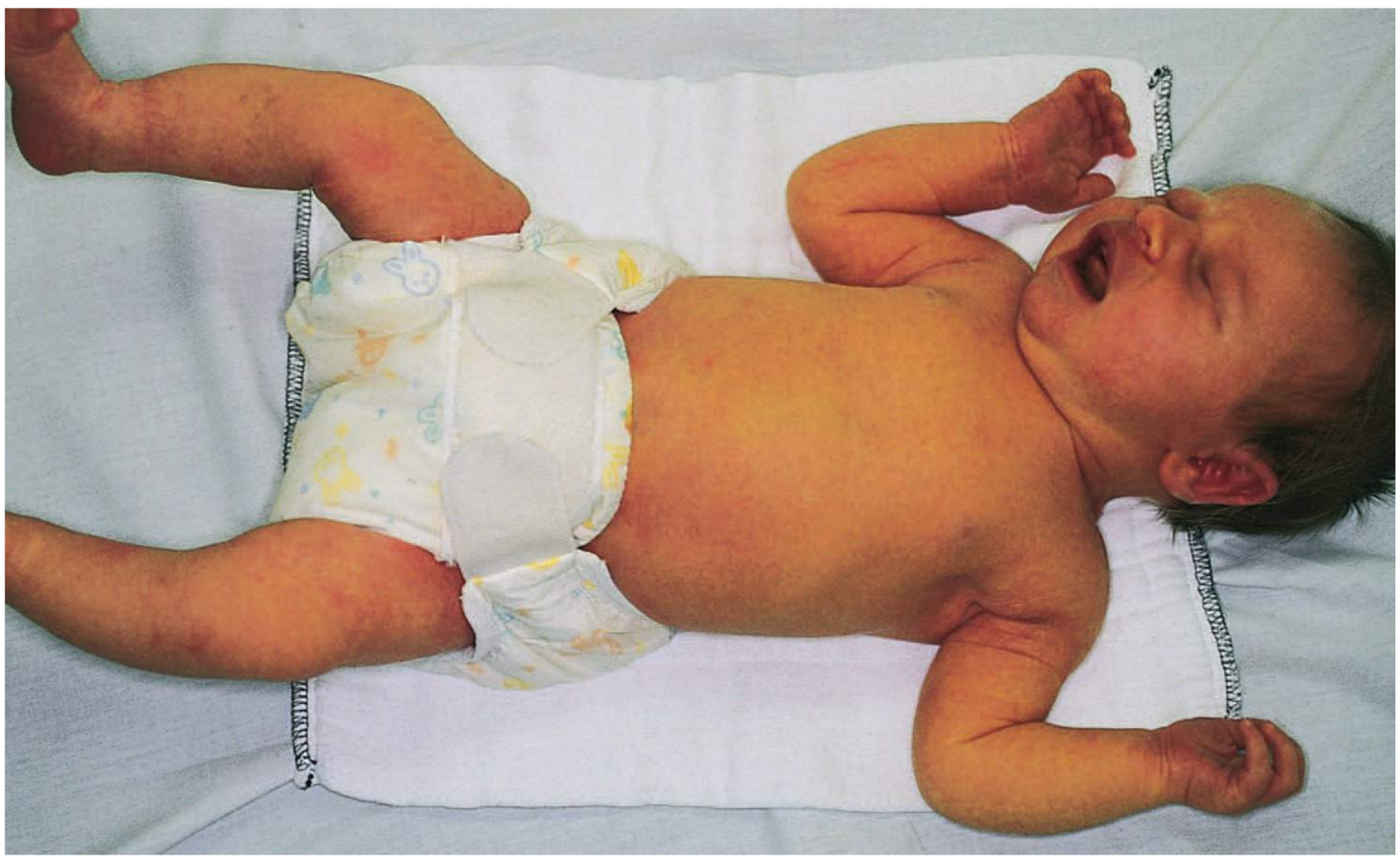


FIGURE 14.6 ■ **Neonatal Jaundice.** Newborn with yellowish hue to skin consistent with jaundice. (Photo contributor: Kevin J. Knoop, MD, MS.)

NEONATAL MILK PRODUCTION (WITCH'S MILK)

Clinical Summary

Neonatal galactorrhea occurs in up to 6% of term newborns and is usually secondary to transplacental transfer of maternal estrogen. These hormonal effects (maternal estrogens and endogenous prolactin) lead to palpable breast buds in approximately one-third of all term newborns. Males and females are equally affected. In most cases, the breast enlargement and galactorrhea begin to subside after the second week of life in males and 2 to 6 months in females. Infants with neonatal breast hypertrophy may be predisposed to infections (mastitis or abscess) possibly incited by repetitive manipulation of the enlarged breast bud by a caregiver. The differential diagnosis includes early mastitis with purulent nipple discharge.

Management and Disposition

Treatment is not necessary unless infection is suspected. Parents can be reassured that this is a normal finding, and follow-up to resolution should occur at routine well child care visits.

Pearls

1. Classical presentation includes the presence of clear colostrum-like secretions in newborns with hypertrophied mammary tissue without erythema or tenderness. Persistence of enlarged breast buds beyond 6 months of age should prompt follow-up with a pediatric endocrinologist.
2. In an older child, galactorrhea may be the presenting sign of hypothyroidism or pathologically elevated prolactin levels.

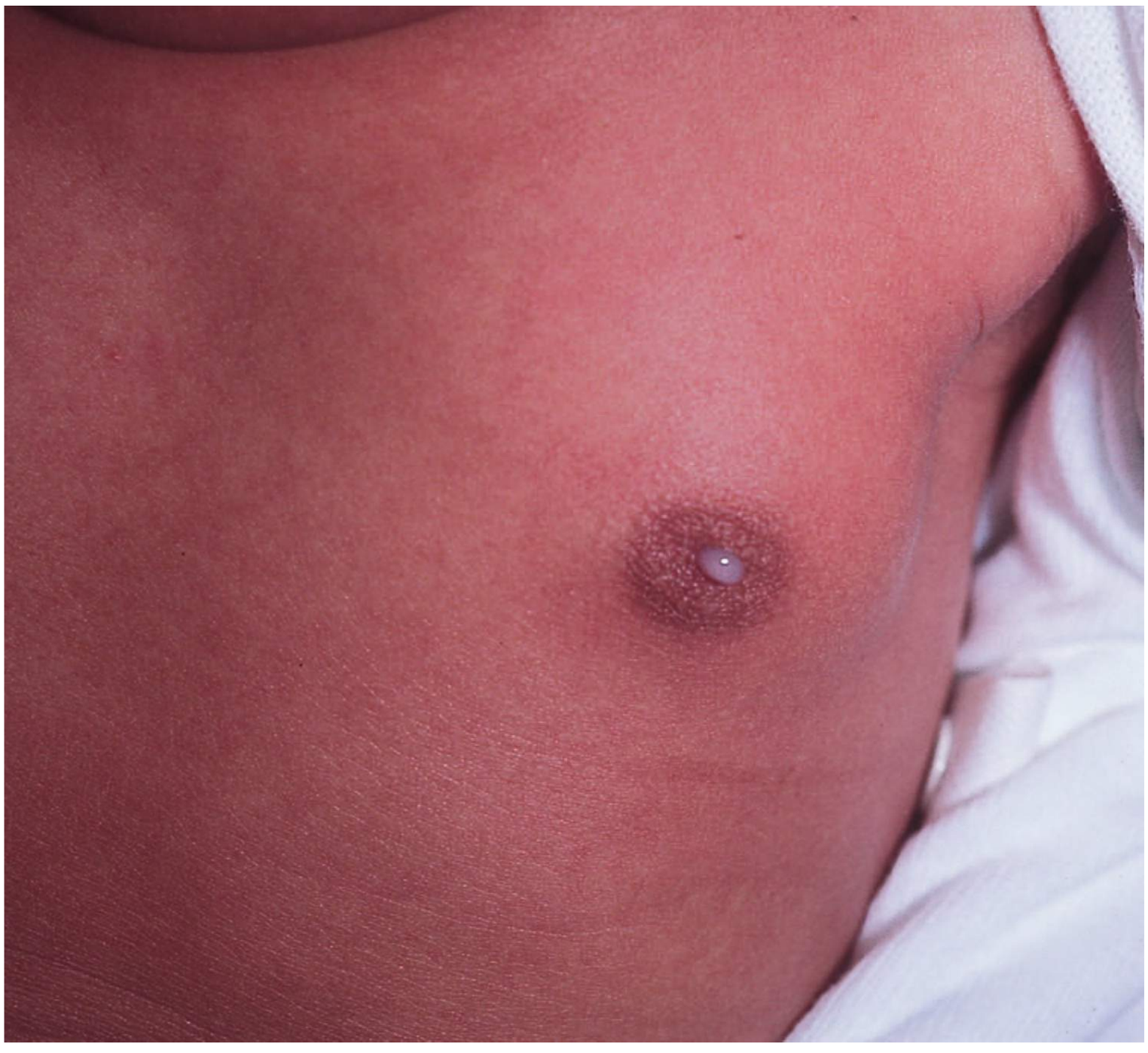


FIGURE 14.7 ■ **Witch's Milk.** Milky fluid draining from the nipple in a newborn. (Photo contributor: Michael J. Nowicki, MD.)

NEONATAL MASTITIS

Clinical Summary

Neonatal mastitis is an infection of the breast tissue that occurs in full-term neonates with a peak incidence in the third week of life. Females are affected more often than males in a 2:1 distribution. Clinically, it manifests as swelling, induration, erythema, warmth, and tenderness of the affected breast. The ipsilateral axillary lymph nodes may be swollen. Approximately two-thirds have palpable fluctuance. In some cases, purulent discharge may be expressed from the nipple. Fever may be present in 25% of affected patients. Other systemic symptoms (irritability, decreased appetite, and vomiting) are less common but indicate a more severe infection if present. Bacteremia is rare. Staphylococcus aureus is the most common pathogen, causing 75% to 85% of cases. Rarely, gram-negative organisms or group B or D Streptococcus are the cause. If treatment is delayed, mastitis may progress rapidly with involvement of subcutaneous tissues and subsequent toxicity. In the initial stages, neonatal mastitis may mimic mammary tissue hypertrophy owing to maternal passive hormonal stimulation. Minor trauma, cutaneous infections, and duct blockage may precede this infection.

Management and Disposition

Immediate treatment is important to avoid cellulitic spread and breast tissue damage. In cases of mild cellulitis with no associated fluctuance in an otherwise well-appearing, afebrile neonate, culture of nipple discharge (if present) and oral antibiotics (antistaphylococcal penicillin or first-generation cephalosporin) with close outpatient follow-up are sufficient. Adjustment of coverage can be made once results of cultures or Gram stain are available, especially in the presence of gram-negative bacilli. In cases involving systemic signs of infection, rapid subcutaneous spread, or toxic appearance, a complete sepsis workup should be performed followed by hospitalization. If no organism is seen on Gram stain, a parenteral antistaphylococcal penicillin plus an aminoglycoside or cefotaxime alone should be used. In cases of palpable fluctuance, prompt surgical consultation should be obtained to assess the need for needle aspiration or incision and drainage. Conservative treatment with intravenous (IV) antibiotics often results in resolution of the fluctuance without surgical intervention. Recovery is usually within 5 to 7 days.

Pearls

1. Antibiotic choice should include coverage for S aureus.
2. Maintain a low threshold for initiating a sepsis workup and IV antibiotics.
3. Delays in the diagnosis and treatment may lead to distortion of the nipple, impairment of the secretory capacity of the breast, and reduction in the size of the adult breast.

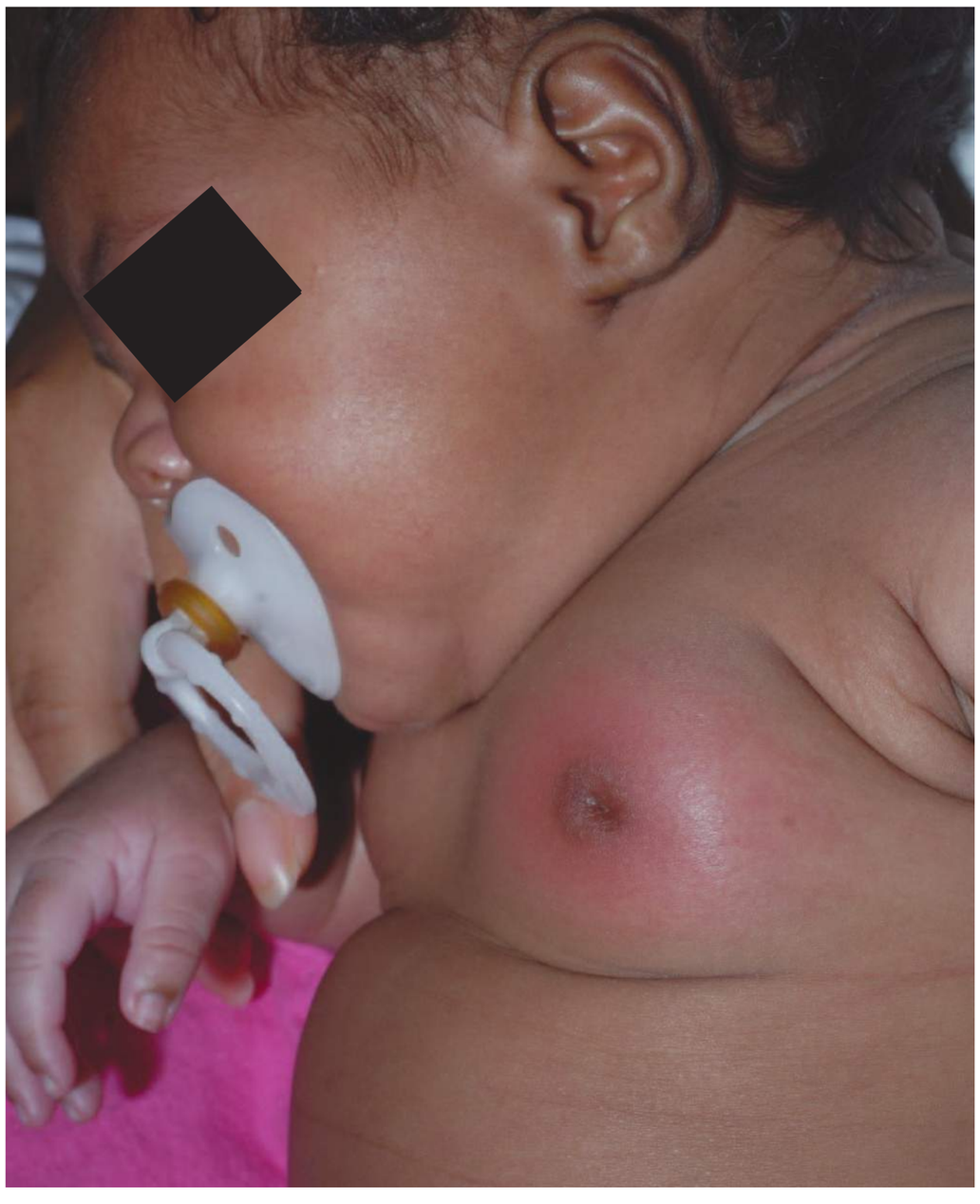


FIGURE 14.8 ■ **Neonatal Mastitis.** Neonate with left-sided breast swelling, erythema, and purulent discharge. (Photo contributor: Lawrence B. Stack, MD.)

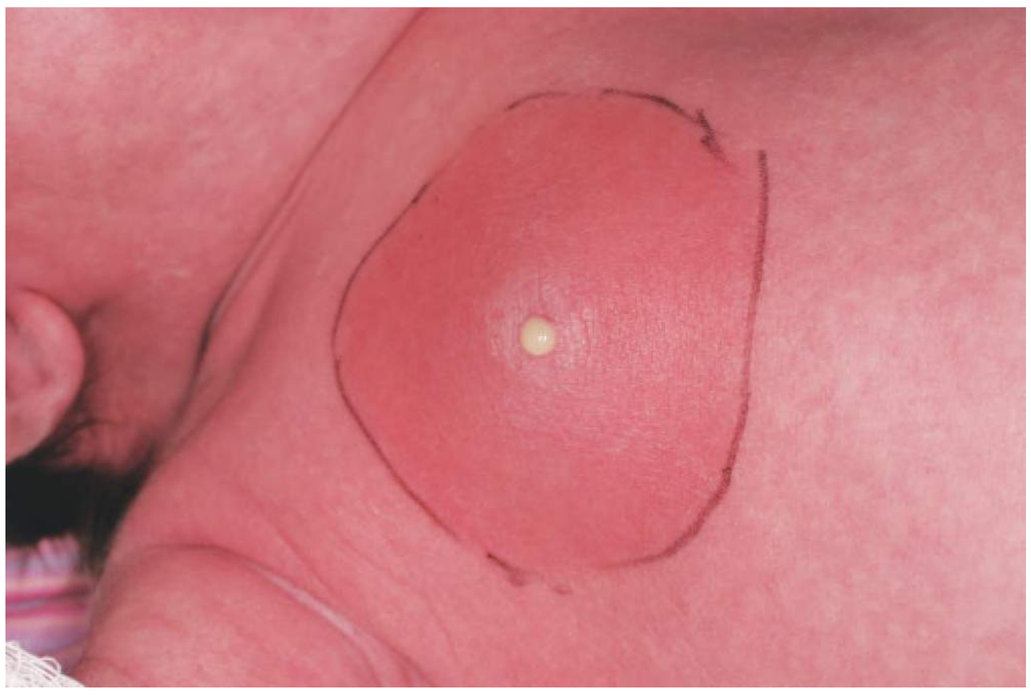


FIGURE 14.9 ■ **Neonatal Mastitis.** Neonate with marked swelling, erythema, and purulent discharge. (Photo contributor: Emergency Medicine Department, Naval Medical Center, Portsmouth, VA.)

Clinical Summary

An umbilical granuloma is granulation tissue with incomplete epithelialization that persists following cord separation. It is the most common cause of an umbilical mass in neonates. Parents will describe a persistent discharge from the umbilicus after the cord has dried and separated. It appears soft, pink, wet, and friable. Infants with an umbilical granuloma do not have localized swelling, redness, warmth, tenderness, or fever. An umbilical polyp is a rare anomaly resulting from the persistence of the omphalomesenteric duct or the urachus and may have a similar appearance. A polyp is usually firm with a mucoid secretion. The differential diagnosis also includes omphalitis, an infection of the umbilicus and surrounding structures, which should be considered in ill-appearing neonates.

Management and Disposition

Advise parents to keep the granuloma dry and exposed to the air as often as possible. Cleaning and drying of the umbilical cord base with alcohol is not necessary and may irritate the skin and delay healing. Cauterization of the granuloma by application of topical silver nitrate is the treatment of choice. It is important to protect the surrounding skin (apply petroleum jelly or antibiotic ointment) and remove excess silver nitrate to avoid chemical burns and skin staining. The cauterization may need to be repeated at 3-day intervals if drainage persists.

Pearls

1. An umbilical granuloma is the most common umbilical mass in neonates.
2. The only sign of granuloma formation may be the presence of nonpurulent discharge noted in the diaper area that is in contact with the umbilicus.
3. Omphalitis presents with redness of the periumbilical area typically tracking upward in the midline and often with a purulent discharge from the umbilicus. It can progress to abdominal wall cellulitis or peritonitis and requires a complete sepsis workup and hospital admission for treatment with broad-spectrum parenteral antibiotics.

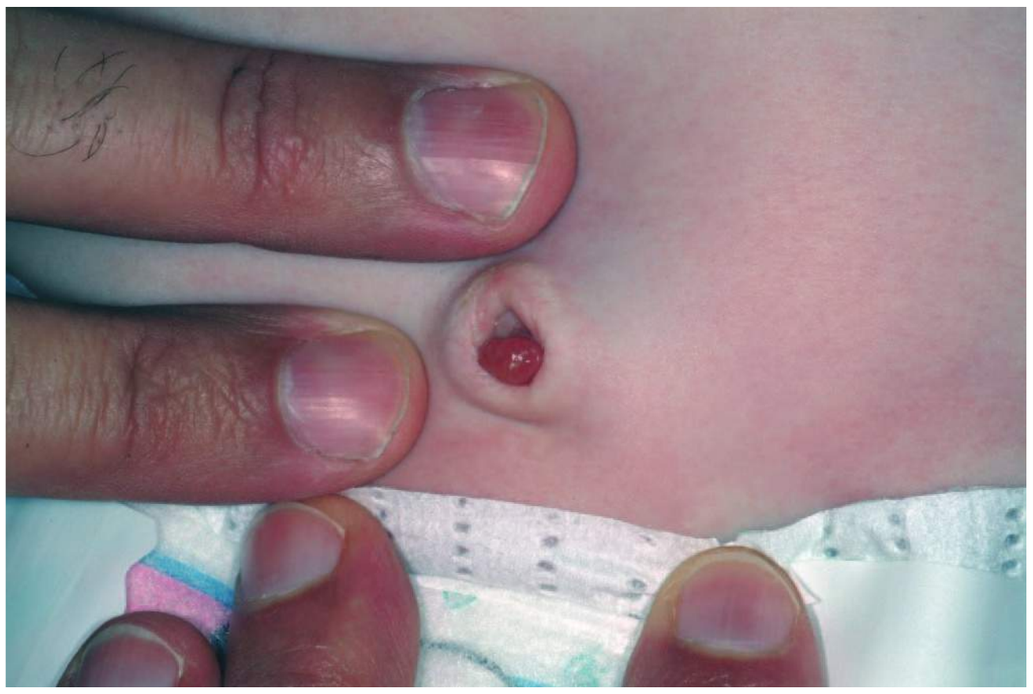


FIGURE 14.10 ■ **Umbilical Granuloma.** Newborn infant with umbilical granuloma visible in umbilicus. (Photo contributor: Anne W. Lucky, MD.)

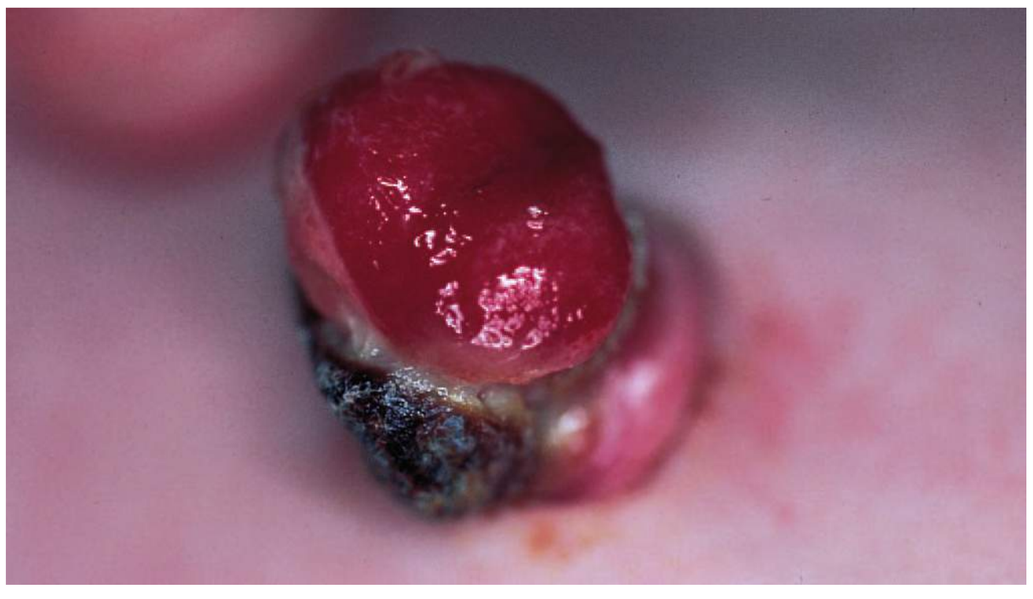


FIGURE 14.11 ■ **Omphalomesenteric Duct.** This red mass resembling a granuloma was found to be an omphalomesenteric duct. (Photo contributor: Kevin J. Knoop, MD, MS.)

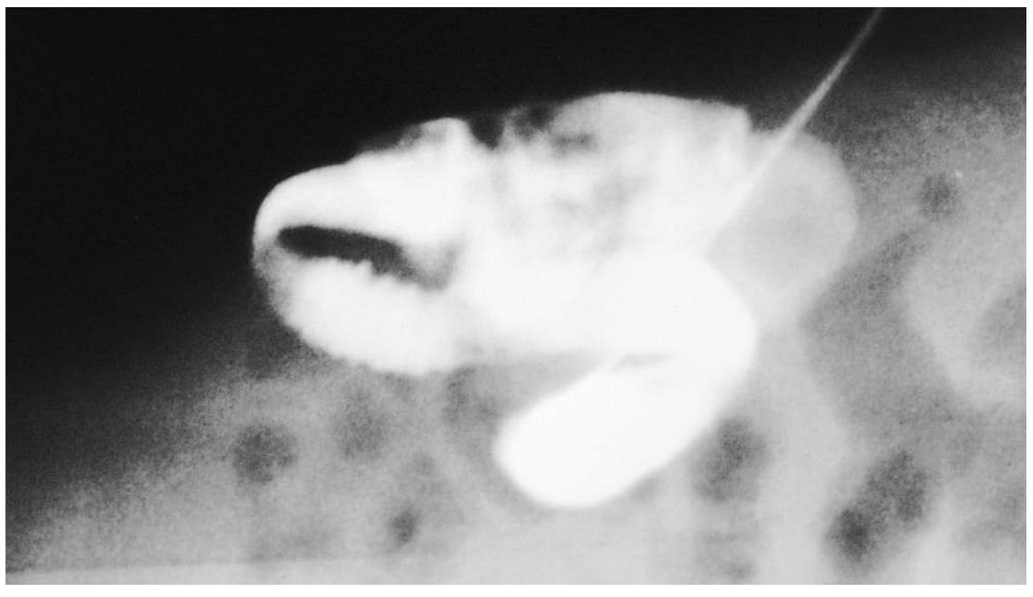


FIGURE 14.12 ■ **Omphalomesenteric Duct.** A fistulogram confirms continuity between the duct and intestine. (Photo contributor: Kevin J. Knoop, MD, MS.)

HYPERTROPHIC PYLORIC STENOSIS

Clinical Summary

Hypertrophic pyloric stenosis (HPS) is characterized by progressive postprandial, nonbilious vomiting that steadily increases in frequency and amount due to hypertrophy of the pyloric musculature and edema of the pyloric canal, producing gastric outlet obstruction. It is usually diagnosed in infants from birth to 5 months, most commonly at 2 to 8 weeks of life. The vomiting may become forceful and is then described as projectile (although this pattern is not always present). There is a familial tendency, and white males (especially firstborn) are more frequently affected. During the physical examination, peristaltic waves may be observed traveling from the left upper to right upper quadrants. The hypertrophy of the antral and pyloric musculature produces the "olive" to palpation (best palpated in the epigastrium or right upper quadrant after emesis or emptying the stomach with a nasogastric tube). As a result of persistent vomiting, hypochloremic, hypokalemic metabolic alkalosis with varying degrees of dehydration, and failure to thrive may occur when the diagnosis is not made early in the course.

The finding of a pyloric olive on palpation of the abdomen is pathognomonic. Ultrasound is a useful tool to confirm the diagnosis when the olive is not evident on examination or early in the course. The differential diagnosis includes intestinal obstruction or atresia, malrotation with volvulus, hiatal hernia, gastroenteritis, adrenogenital syndrome, increased intracranial pressure, esophagitis, sepsis, gastroesophageal reflux, and poor feeding technique.

Management and Disposition

Treatment includes correction of electrolyte imbalances and dehydration, as well as emergent surgical consultation for curative Ramstedt pyloromyotomy. Failure to correct metabolic alkalosis prior to surgery can increase the risk of postoperative apnea. Patients may benefit from a nasogastric tube on low intermittent suction.

Pearls

1. HPS is the most common cause of metabolic alkalosis in infancy.
2. Serial examinations and observation of the child after oral fluid challenges for persistent projectile vomiting may aid in making the diagnosis.
3. Clinical manifestations of pyloric stenosis begin at a mean age of 3 weeks after birth.
4. Pyloric ultrasonography is the diagnostic study of choice.
5. Less than 2% of infants with HPS have bilious vomiting.

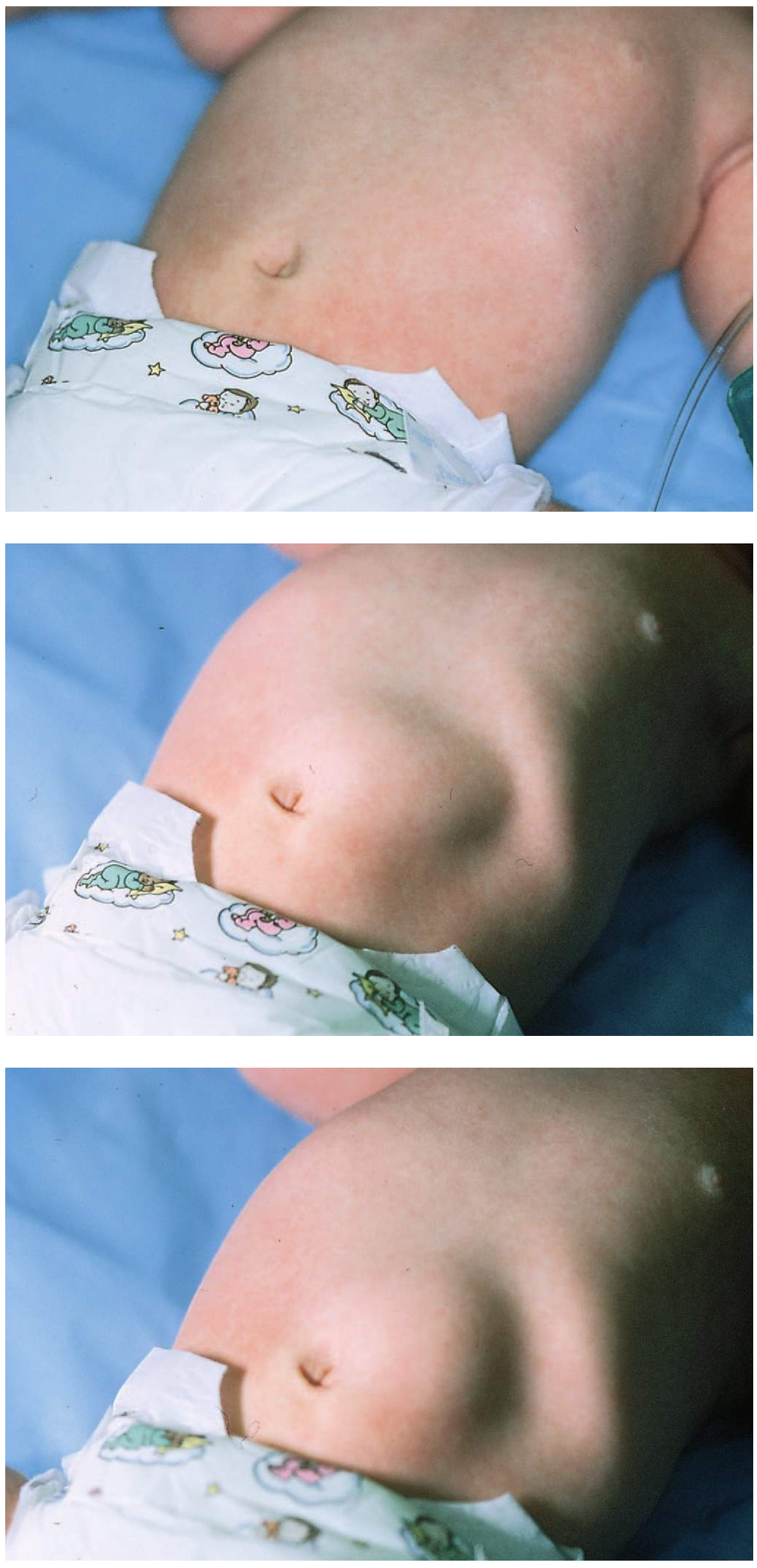


FIGURE 14.13 ■ **Gastric Wave of Hypertrophic Pyloric Stenosis.** A gastric wave can be seen traversing the abdomen in this series of photographs of a patient with HPS. (Photo contributor: Kevin J. Knoop, MD, MS.)

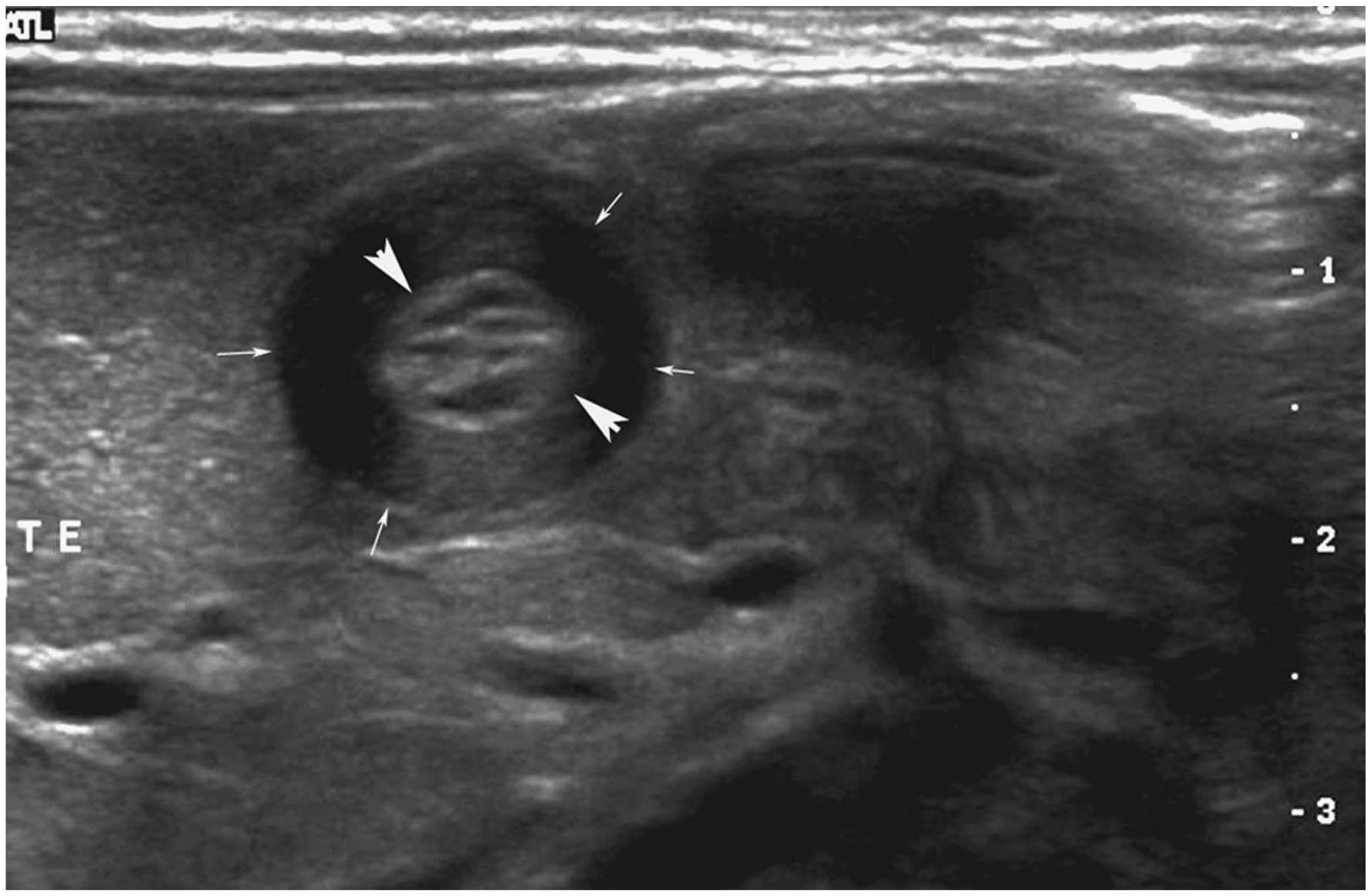


FIGURE 14.14 ■ **Ultrasound Target Sign in HPS.** This transverse ultrasound shows redundant, infolded mucosa (arrowheads) between muscular components (arrows), referred to as the "target sign." (Photo contributor: Lawrence B. Stack, MD.)

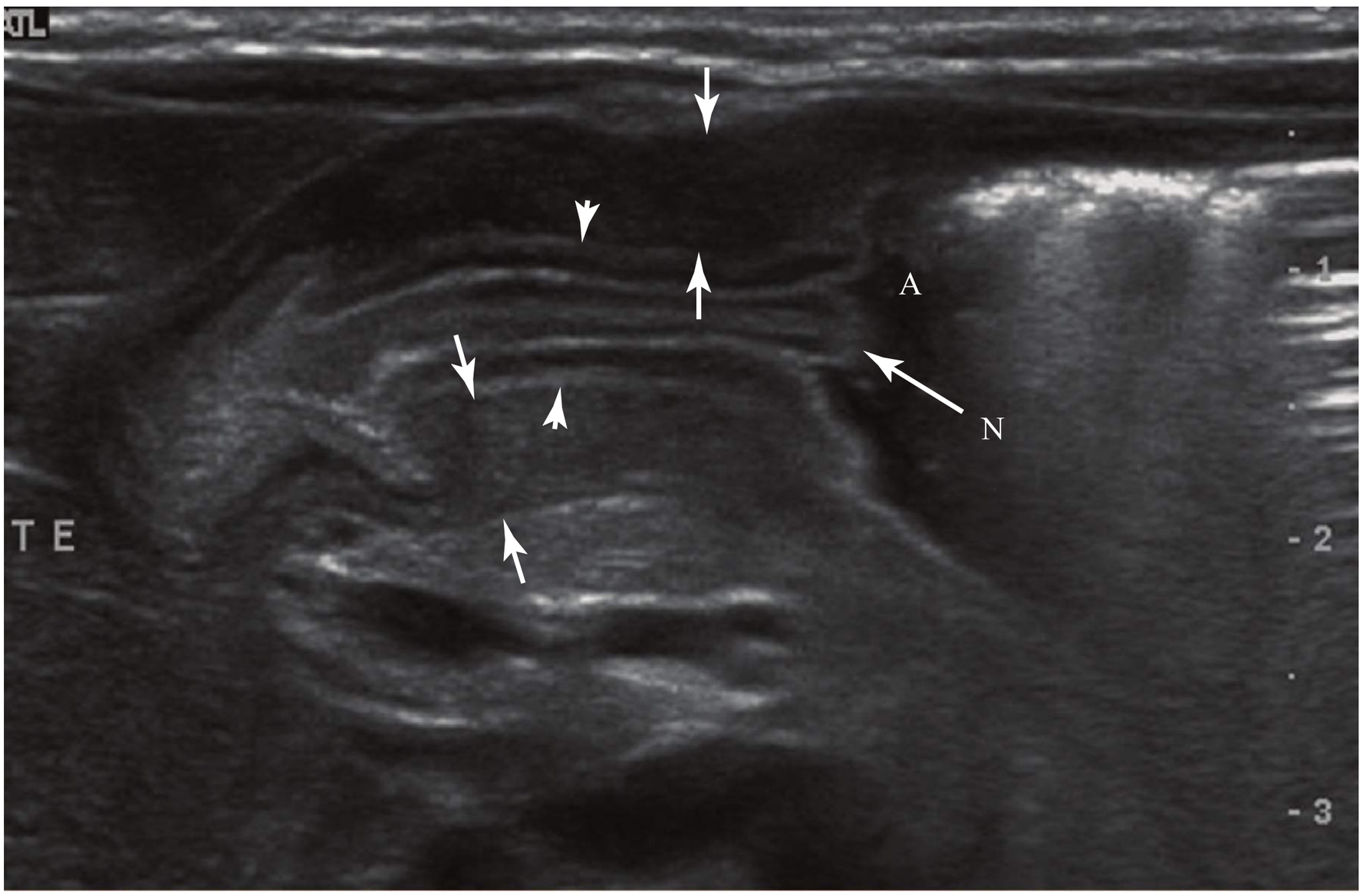


FIGURE 14.15 ■ **Antral Nipple Sign in HPS.** This longitudinal sonogram shows two-layered, thickened mucosa (arrowheads) surrounded by muscular components (arrows). Redundant pyloric mucosa protrudes into the fluid in the gastric antrum (A), forming the "antral nipple sign" (N) in HPS. (Photo contributor: Lawrence B. Stack, MD.)

INTESTINAL MALROTATION WITH VOLVULUS

Clinical Summary

Intestinal malrotation is primarily a condition of infancy but can be seen in older children and adults. During the fourth week of embryonic development, the primary intestinal loop bulges into the yolk sac, rotates 270 degrees counterclockwise, and returns to the enlarged abdominal cavity between the eight and tenth week where it is fixed via mesentery that extends from the ligament of Treitz to the ileocecal valve in the right lower quadrant. In malrotation, the duodenum, jejunum, and cecum are partially rotated with the bowel anchored by an abnormally thin band of mesentery. The bowel can twist on this thin mesentery, which contains the superior mesenteric artery, leading to acute intestinal obstruction and midgut vascular compromise known as volvulus. Also, the abnormally positioned cecum now rests in the upper abdomen fixed to the right lateral abdominal wall by bands of peritoneum (Ladd bands) that cross and can obstruct the duodenum.

Presentation is with vomiting in 50% of cases. The vomiting may not be bilious, but it is classically described as such. Patients are often irritable with significant abdominal tenderness. Third space fluid losses increase as gut ischemia progresses. Differential diagnosis includes pyloric stenosis, intestinal atresias, appendicitis, necrotizing enterocolitis, and intussusception.

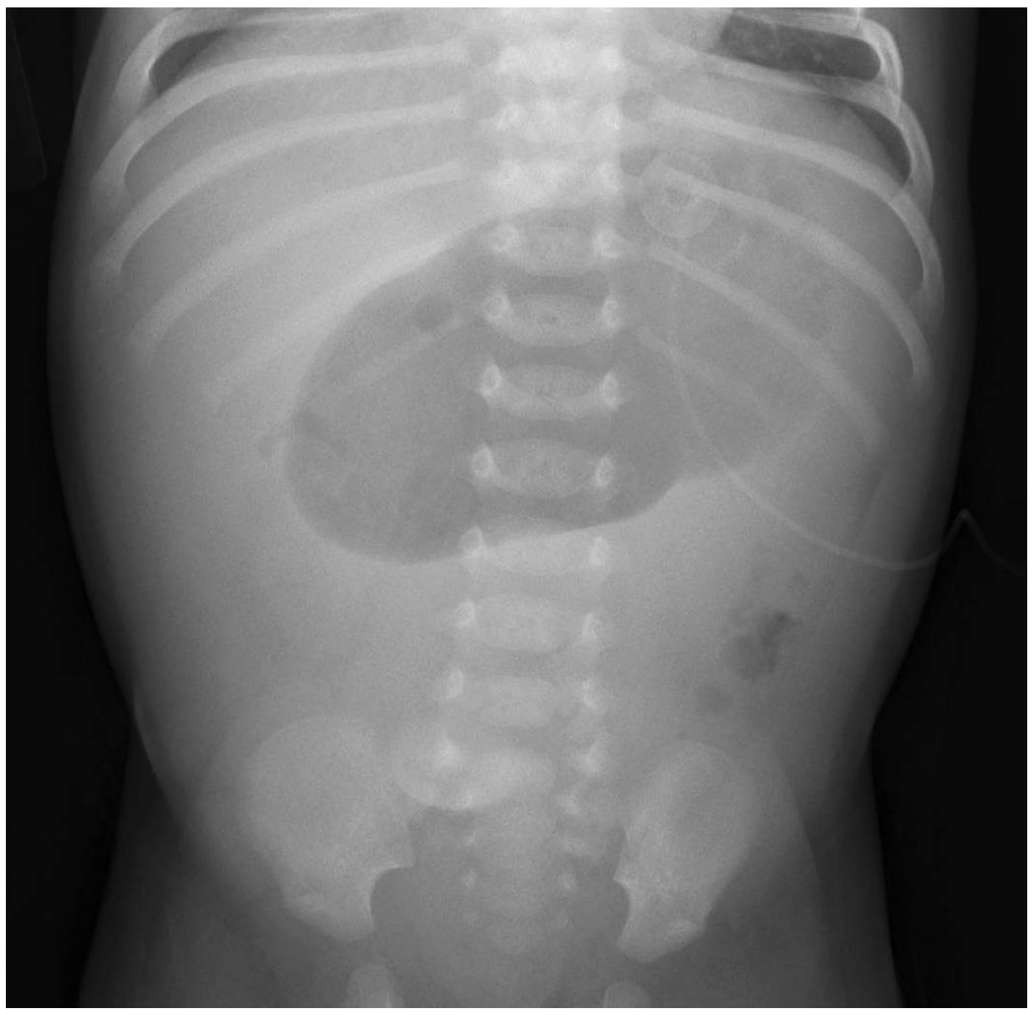


FIGURE 14.16 ■ **Intestinal Malrotation with Volvulus.** Antero-posterior x-ray of the abdomen in a newborn with bilious emesis. Dilated, air-filled stomach with a relative paucity of gas in the distal bowel. (Photo contributor: Alexander Towbin, MD.)

Management and Disposition

Emergent surgical consultation is indicated. Plain x-rays are rarely diagnostic, but may show signs of small bowel obstruction. An upper gastrointestinal (GI) series is the most helpful study, though one-quarter of cases may have an equivocal result. Preoperative management consists of resuscitation with IV fluids, placing a gastric tube for decompression, and administration of broad-spectrum antibiotics.

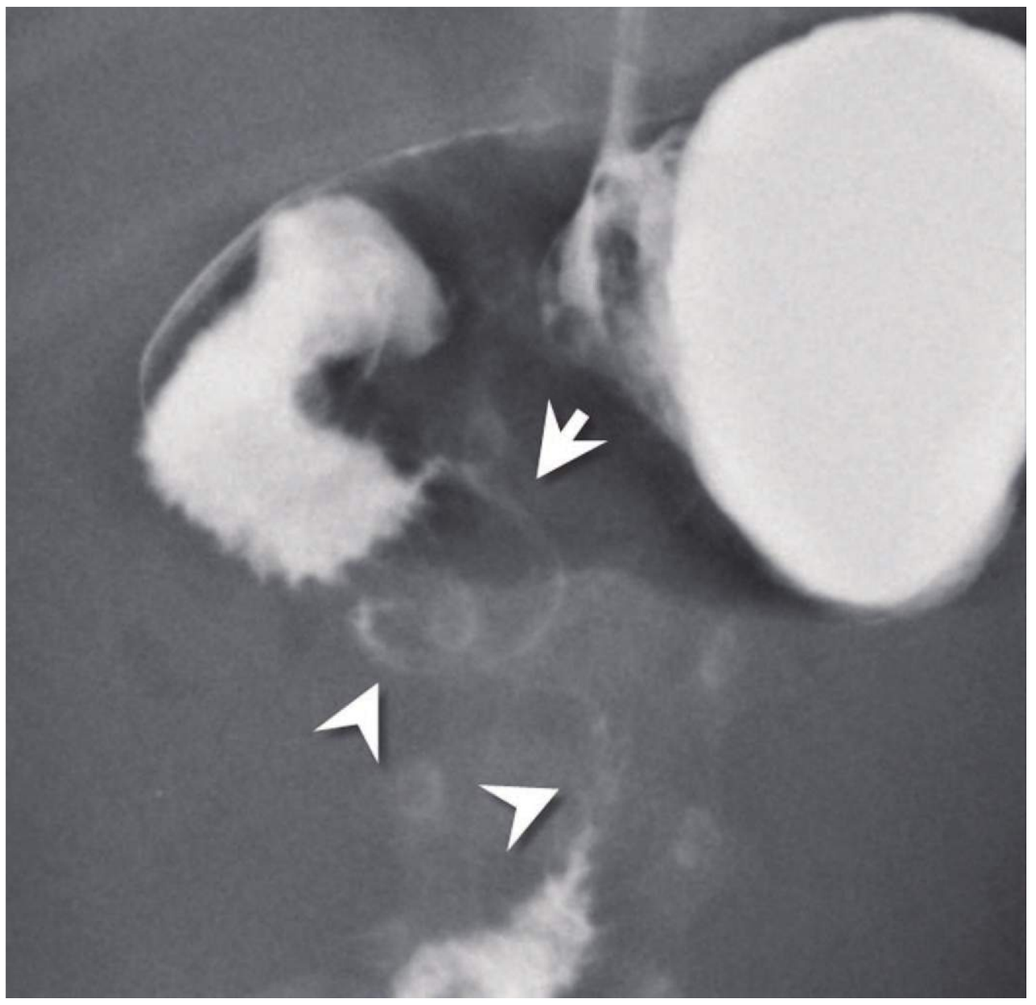


FIGURE 14.17 ■ **Intestinal Malrotation with Volvulus.** Antero-posterior radiograph from an upper GI series shows malrotation with midgut volvulus. The duodenum (arrowheads) does not cross midline and does not extend superiorly to the level of the pylorus. The spiraling downward appearance is consistent with volvulus. (Photo contributor: Alexander Towbin, MD.)

Pearls

1. Fifty percent of patients with malrotation present with volvulus in the first month of life.
2. Over 30% of cases of malrotation with volvulus present beyond early childhood usually with an insidious onset with abdominal pain as the most common symptom.
3. The imaging test of choice is the upper GI series, but computed tomography (CT) should be considered in ill-appearing patients in which administration of enteral contrast may be difficult.

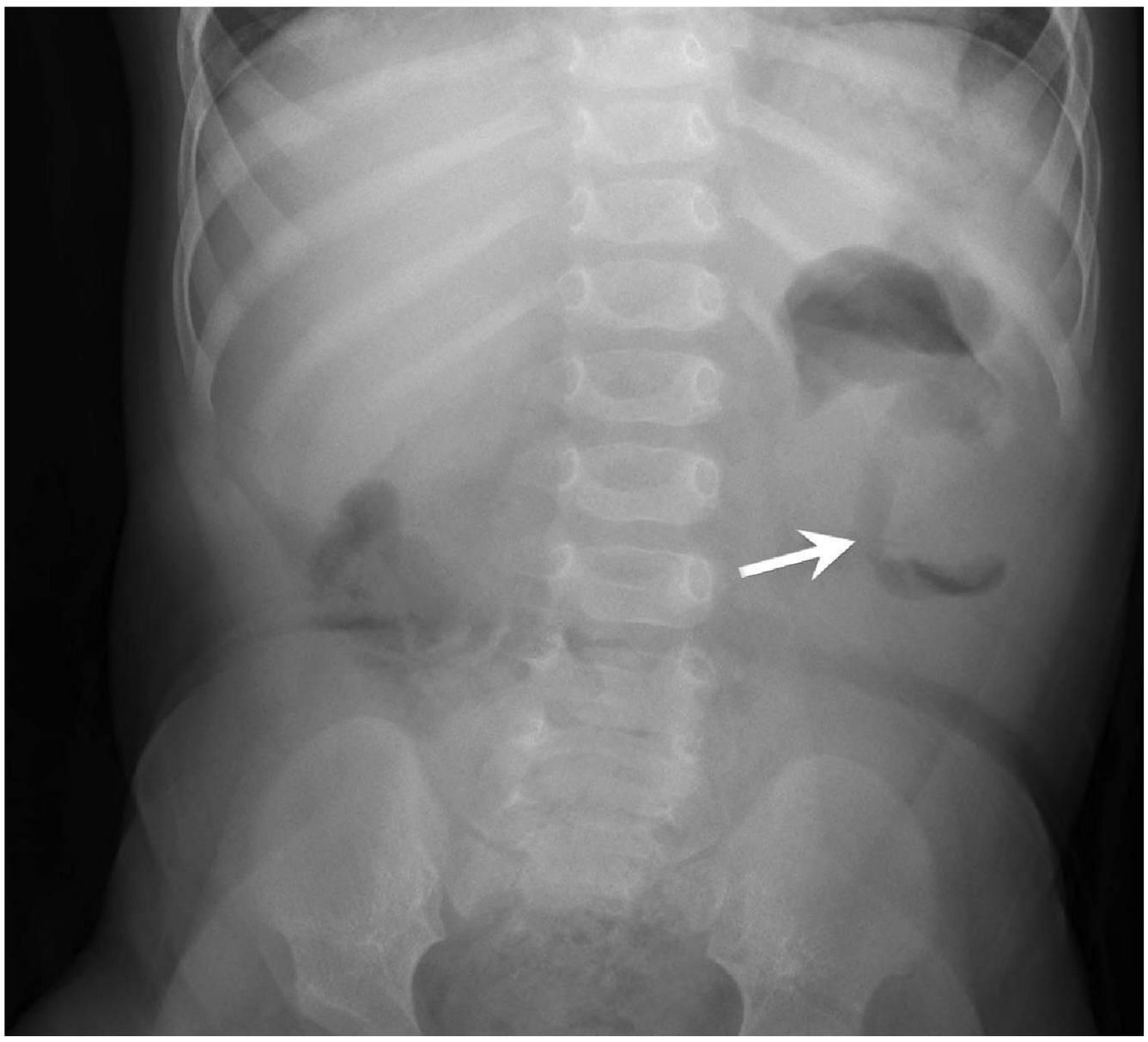


FIGURE 14.18 ■ **Intestinal Malrotation with Volvulus.** Soft tissue mass (arrow) surrounded by air within the left upper quadrant near the hepatic flexure. (Photo contributor: Alexander Towbin, MD.)

ERYTHEMA INFECTIOSUM (FIFTH DISEASE)

Clinical Summary

Erythema infectiosum is a viral infection caused by parvovirus B19, presenting most commonly between 4 and 10 years of age. It begins with nonspecific prodromal symptoms including malaise, coryza, headache, fever, nausea, and diarrhea. Two to 5 days into the illness, the classic rash appears characterized initially by the "slapped cheeks" appearance of a bright red malar macular rash that spares nasal ridge and perioral areas. A reticulated, lacy erythematous maculopapular eruption with central clearing then appears on the extensor surfaces of extremities. The differential diagnosis includes other morbilliform eruptions such as measles, rubella, roseola, and infectious mononucleosis. Bacterial infections (eg, scarlet fever), drug reactions, and other skin conditions such as guttate psoriasis, papular urticaria, atopic dermatitis, and erythema multiforme are also included in the differential.

Management and Disposition

Treatment is aimed at symptomatic relief. Parents can be reassured that this exanthem is benign and self-limited. Once the rash appears, the patient is no longer contagious. It is important to educate the patient and family about the possible risk of parvovirus B19 as a cause of hydrops fetalis or fetal deaths early in pregnancy. It can also cause an aplastic crisis in patients with hematologic conditions such as sickle cell disease, hereditary spherocytosis, and various hemolytic anemias, or in the immunocompromised.

Pearls

1. Recrudescence of the lacy, reticular rash may occur with exercise, overheating, emotional upset, or sun exposure as a result of cutaneous vasodilatation.
2. Parvovirus B19 is the most common cause of hydrops fetalis. Pregnant mothers of children diagnosed with erythema infectiosum should have their serologic status determined.
3. In young adults, parvovirus B19 can cause papular purpuric gloves and socks syndrome.

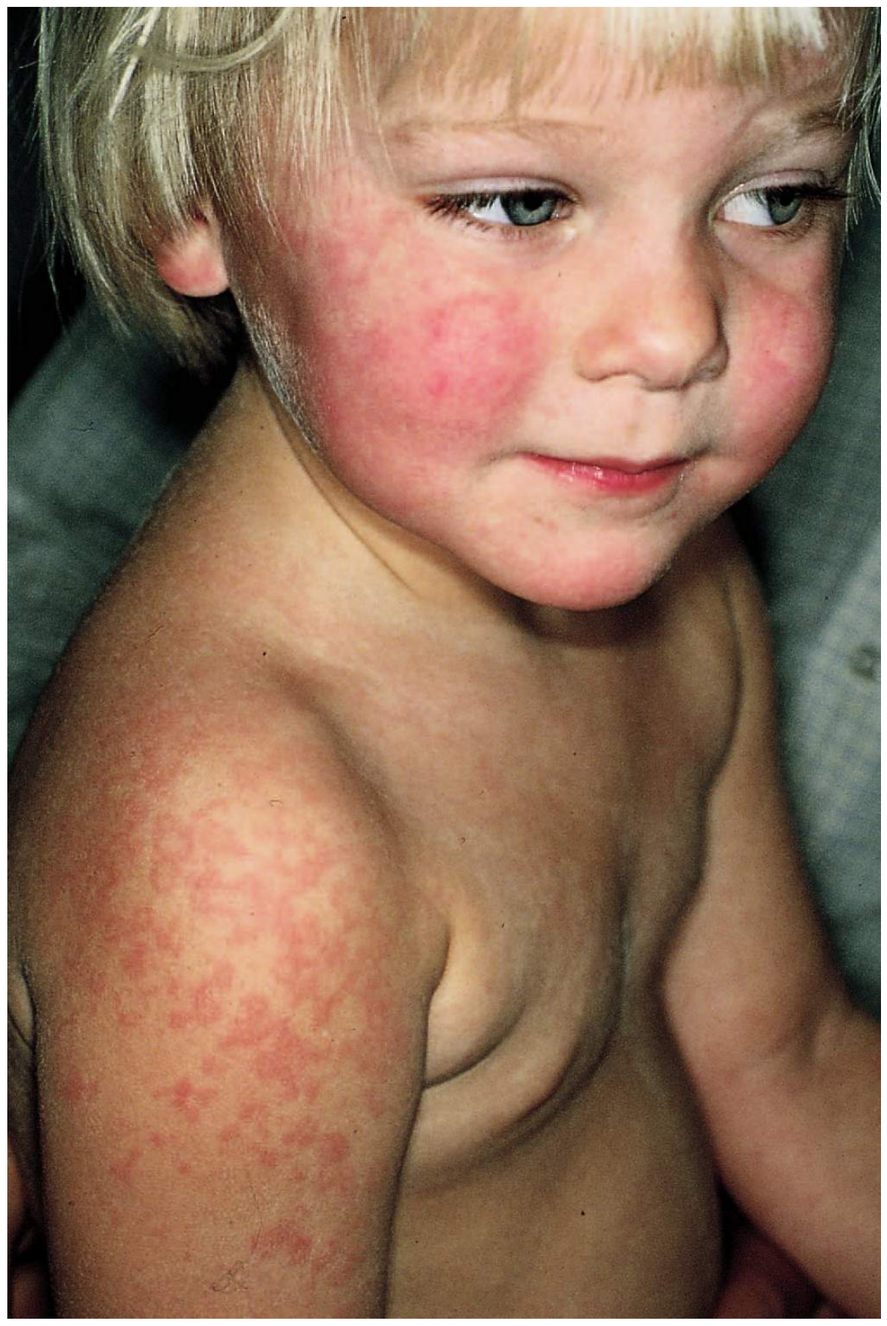


FIGURE 14.19 ■ **Fifth Disease.** Toddler with the classic slapped-cheek appearance of fifth disease caused by parvovirus B19. Note the lacy reticular macular rash on the shoulder and upper extremity. (Photo contributor: Anne W. Lucky, MD.)

ROSEOLA INFANTUM (EXTHANTHEM SUBITUM)

Clinical Summary

The typical presentation of roseola infantum involves a prodrome characterized by a 3- to 5-day history of high fever often exceeding 40°C (104°F) in a child 6 months to 3 years of age. The child may be fussy, but is often otherwise well-appearing. After the child's fever abates, the typical exanthem appears characterized by blanching erythematous macules and papules on the trunk, neck, proximal extremities, and occasionally the face. The rash fades within 2 to 4 days, but may only last several hours. The causative agent in 90% of cases is human herpesvirus 6 (HHV-6). The differential diagnosis includes viruses such as measles, rubella, parvovirus B19, or infectious mononucleosis. Bacterial infections (eg, scarlet fever), drug reactions, and other skin conditions such as guttate psoriasis, papular urticaria, and erythema multiforme are also included in the differential.

Management and Disposition

As with most viral infections, only supportive therapy is necessary. Special attention should be paid in maintaining fluid intake, fever control for the patient's comfort, and parental education about the benign, self-limited nature of this illness.

Pearls

1. With defervescence and appearance of the rash, the patient is no longer contagious.
2. The most frequent complication of roseola is febrile seizures.

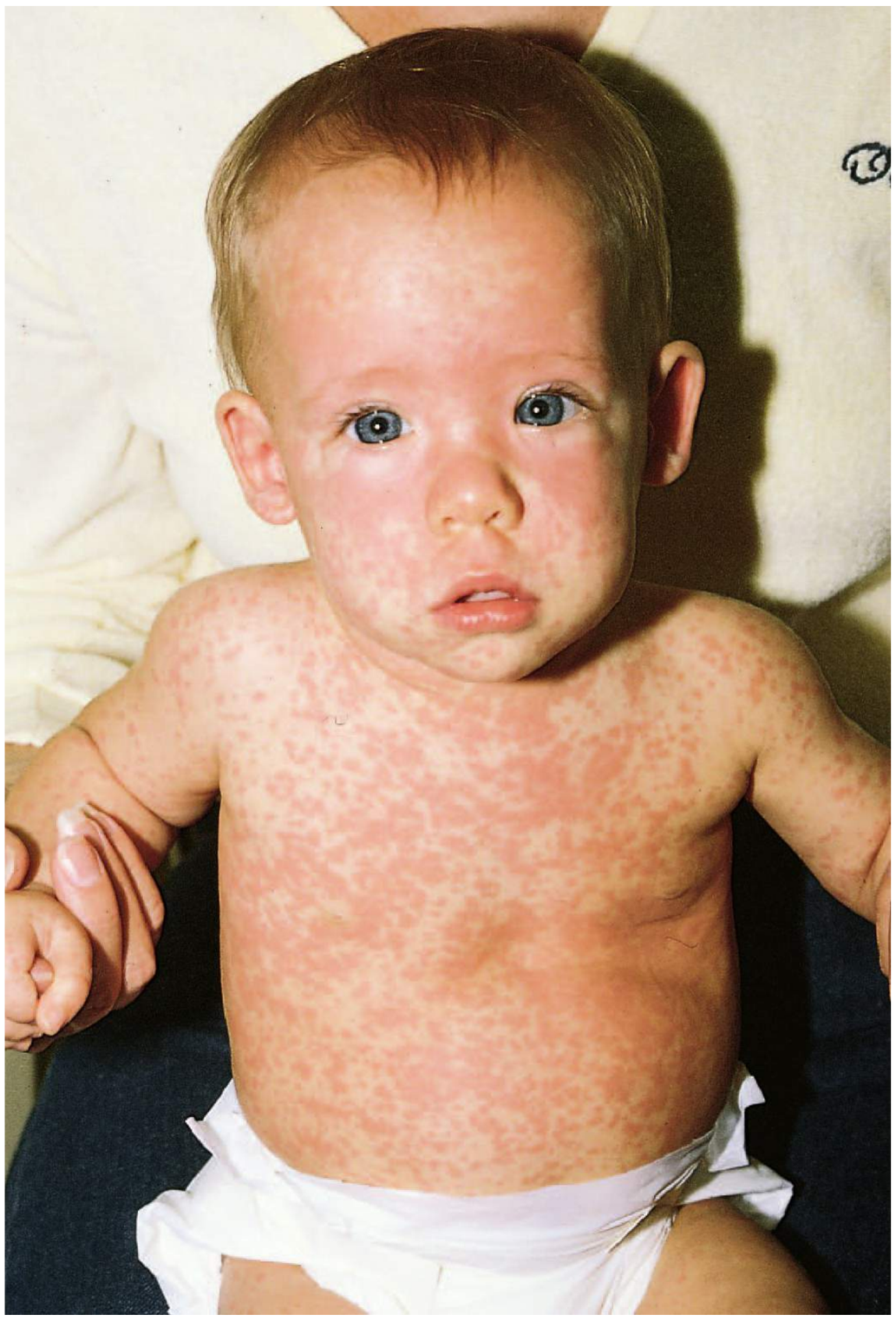


FIGURE 14.20 ■ **Roseola Infantum (Exanthem Subitum).** Toddler with maculopapular eruption of roseola. (Photo contributor: Raymond C. Baker, MD.)

IMPETIGO

Clinical Summary

Impetigo is a contagious bacterial infection of the superficial skin that is caused by Streptococcus pyogenes (group A β-hemolytic Streptococcus) and S aureus. It most commonly affects children 2 to 5 years of age and usually involves the face and extremities. It begins as small vesicles or pustules with very thin roofs that rupture easily with the release of a cloudy fluid and subsequent formation of honey-colored crusts. Variants include bullous impetigo and the ulcerative form. The lesions may spread rapidly by autoinoculation secondary to scratching and coalesce to form larger areas of infection. The differential diagnosis includes second-degree burns, varicella, herpes simplex infections, nummular dermatitis, superinfected eczema, and scabies.

Management and Disposition

These lesions are highly contagious and spread by direct contact. Hand washing and personal hygiene should be emphasized to the patient and family. Application of topical antibacterials, such as mupirocin ointment, has proven to be as effective as oral antibiotics if there are a limited number of lesions and no bullae. Over-the-counter triple antibiotic ointments (bacitracin-neomycin-polymyxin B) may not be as effective. If lesions are extensive and/or bullous, oral antibiotic coverage is indicated.

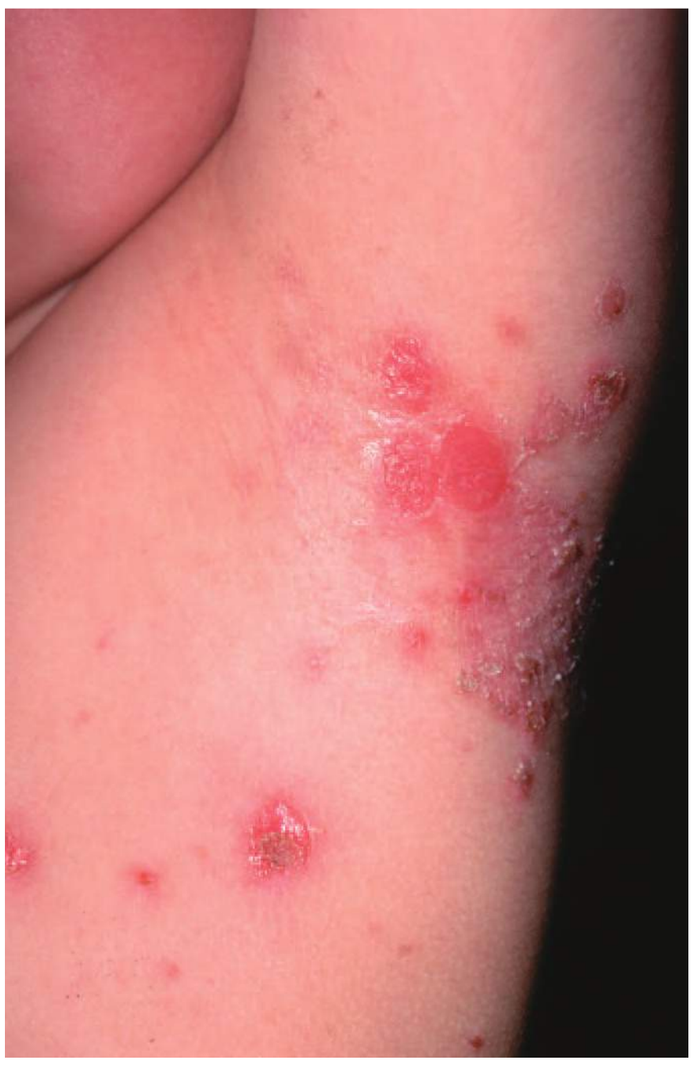


FIGURE 14.22 ■ **Impetigo.** Chest wall and axillary erosions oozing discharge forming honey-colored crusts. (Photo contributor: Lawrence B. Stack, MD.)

Effective oral agents include first-generation cephalosporins, dicloxacillin, amoxicillin with clavulanic acid, or clindamycin.

Pearls

1. Inflicted cigarette burns may resemble the lesions of impetigo.
2. Poststreptococcal glomerulonephritis and rheumatic fever can be complications of impetigo caused by group A β-hemolytic Streptococcus.

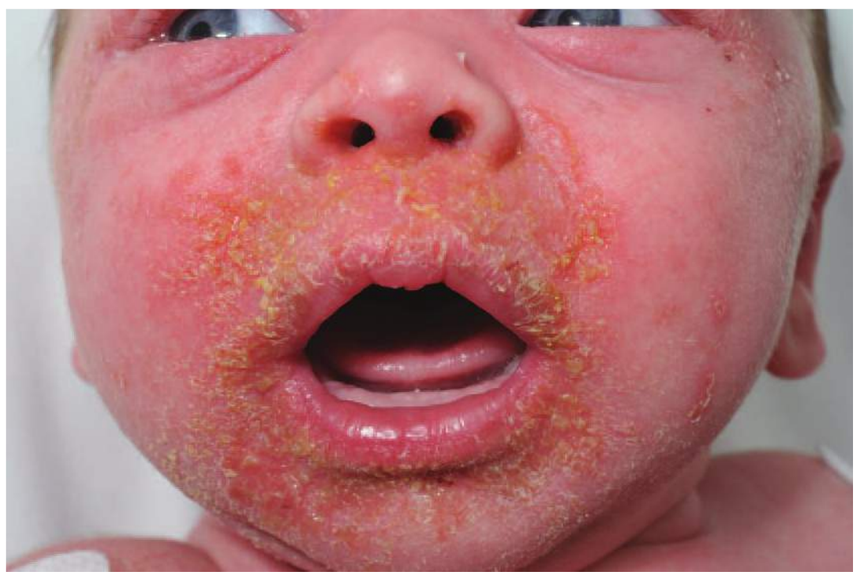


FIGURE 14.21 ■ **Impetigo.** Infant with perioral erosions oozing discharge forming honey-colored crusts. (Photo contributor: Lawrence B. Stack, MD.)

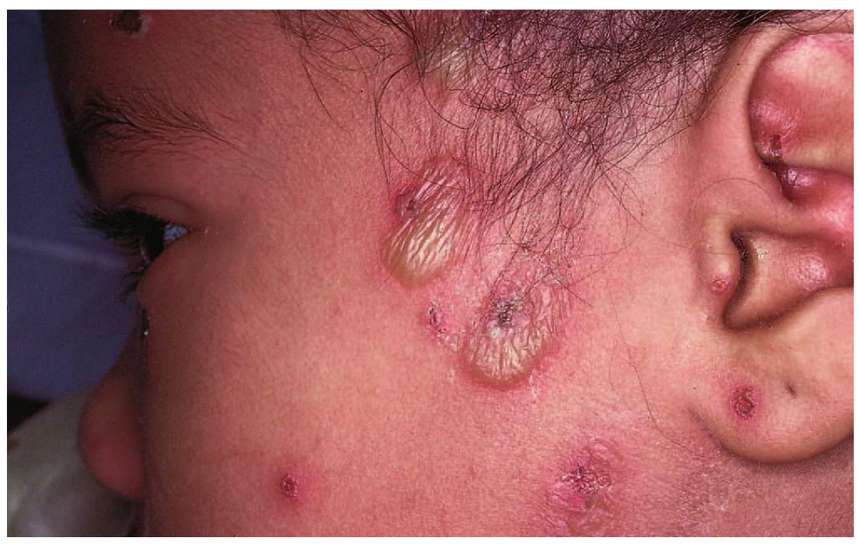


FIGURE 14.23 ■ **Bullous Impetigo.** A child with impetiginous lesions on the face. Note the formation of bullae. (Photo contributor Anne W. Lucky, MD.)

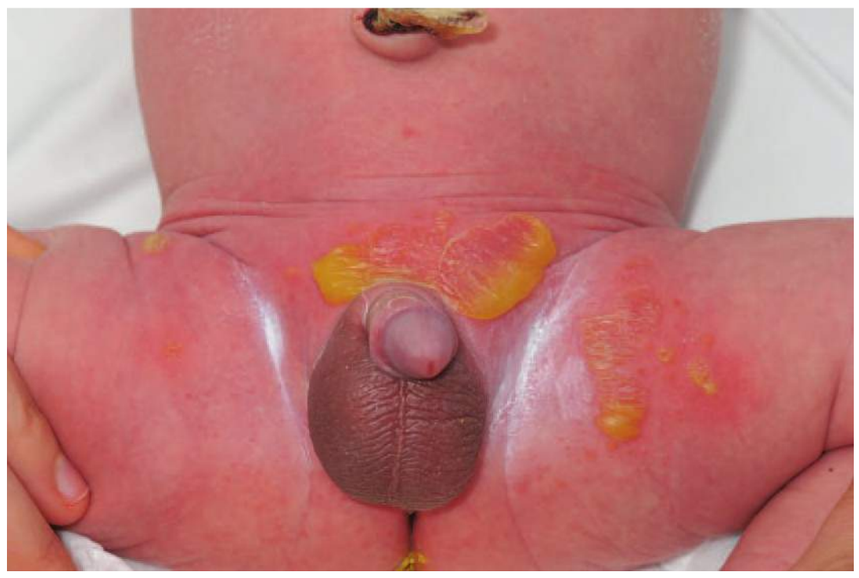


FIGURE 14.24 ■ **Bullous Impetigo.** Bullae with honey-colored exudate in a neonate with fever. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Measles presents as an acute febrile illness with a 3- to 4-day prodromal period characterized initially by fever, malaise, and anorexia, followed closely by conjunctivitis, coryza, and cough. Koplik spots, the pathognomonic enanthem of measles, appear as 1- to 3-mm red papules with gray-white centers on the buccal mucosa. They usually present approximately 48 hours before the development of the characteristic erythematous blanching maculopapular rash. This rash appears during the third or fourth day of the illness as dark red to purple macules and papules on the forehead, around the hairline, behind the earlobes, subsequently spreading in a cephalocaudad progression. Individual lesions often become confluent. The lesions tend to fade in the same order that they appear.

Most cases recover without complications; others may develop otitis media, croup, pneumonia, encephalitis, myocarditis/pericarditis, keratitis, and rarely subacute sclerosing panencephalitis (SSPE), a very late complication. The differential diagnosis of the characteristic rash is vast and includes exanthem subitum, rubella, infections caused by echovirus, coxsackie and adenoviruses, toxoplasmosis, infectious mononucleosis, scarlet fever, Kawasaki disease, drug reactions, Rocky Mountain spotted fever, and meningococcemia.

Management and Disposition

Supportive therapy includes bed rest, antipyretics, and adequate fluid intake. Complications should be treated accordingly. Currently available antivirals are not effective. Postexposure prophylaxis includes administration of the measles-mumps-rubella (MMR) vaccine within 72 hours of exposure to a patient with active measles. Passive immunization with intravenous immune globulin (IVIG) is effective for prevention and attenuation of measles if given within 6 days of the initial exposure especially in pregnant women and the immunocompromised. During outbreaks, MMR vaccine can be given to infants younger than 12 months. However, such infants require an additional two doses of MMR at the recommended ages after their first birthday.

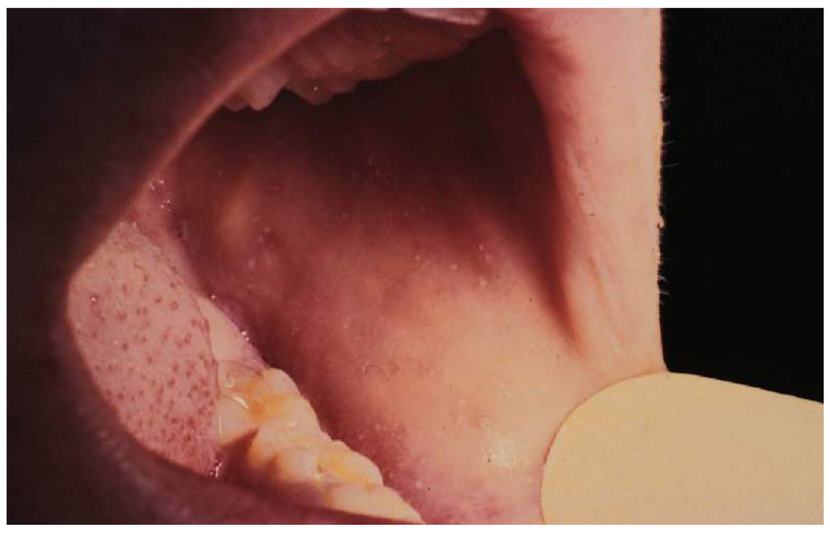


FIGURE 14.25 ■ **Koplik Spots.** Tiny white dots (Koplik spots) are seen on the buccal mucosa. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. Measles is now rare in the United States but remains endemic in other parts of the world.
2. MMR vaccine is preferable to IVIG as postexposure prophylaxis.
3. Morbilliform means measles-like.

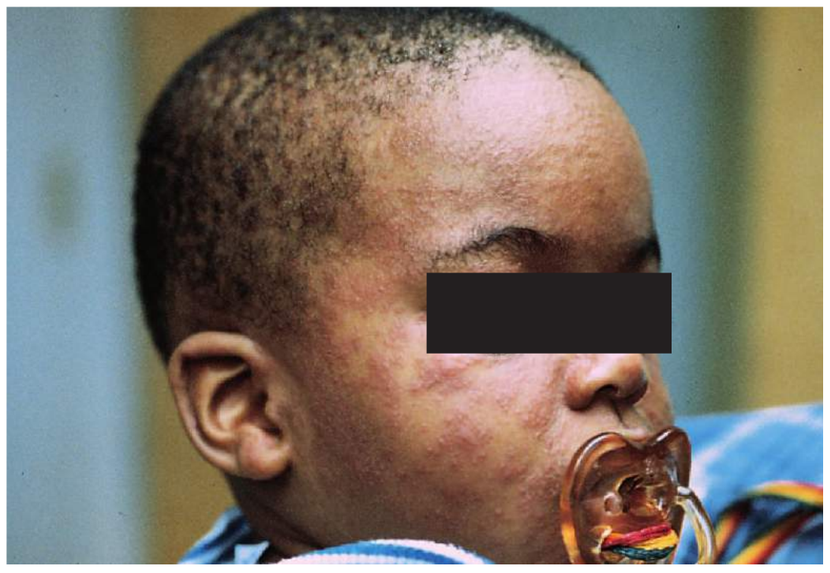


FIGURE 14.26 ■ **Measles.** School-aged child with a morbilliform rash on his face consistent with measles. (Photo contributor: Javier A. Gonzalez del Rey, MD.)

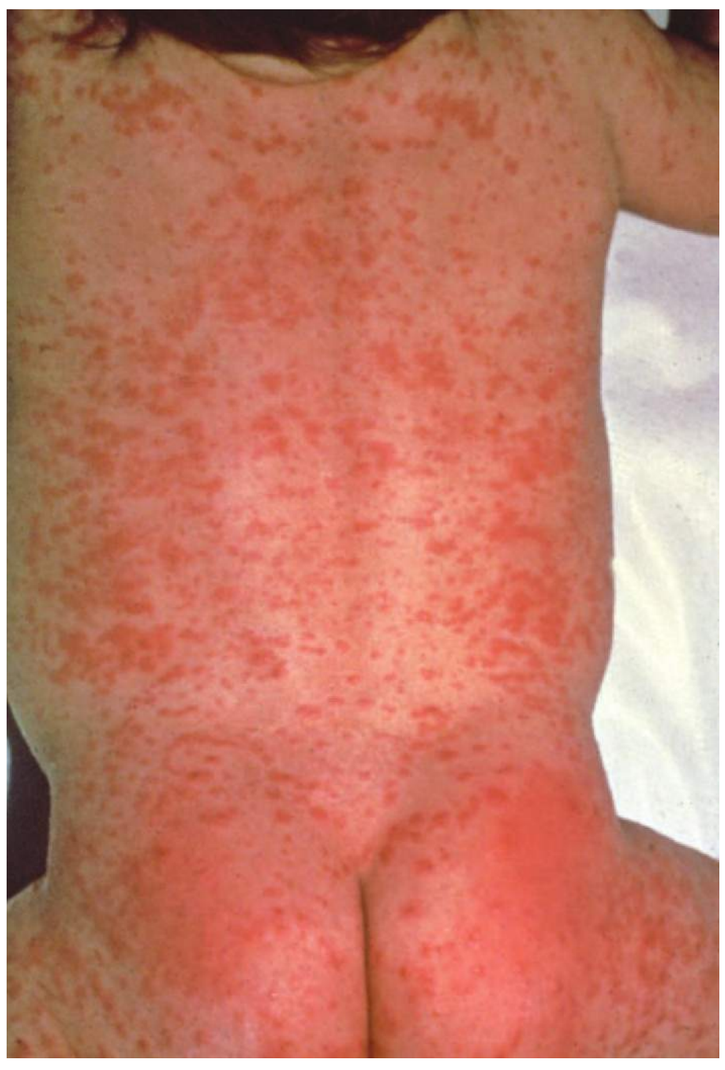


FIGURE 14.27 ■ **Measles.** School-aged child with a morbilliform rash on his face consistent with measles. (Photo contributor: David Effron, MD.)

VARICELLA (CHICKENPOX)

Clinical Summary

Chickenpox results from primary infection with varicella zoster virus (VZV) and is characterized by a generalized pruritic vesicular rash, fever, and mild systemic symptoms. Fifteen days after exposure and following a prodrome of fever, malaise, pharyngitis, and/or loss of appetite, the characteristic generalized pruritic vesicular rash develops. The lesions usually develop 24 hours after the onset of illness, appear in crops, start on the trunk and spread peripherally, and evolve from erythematous, pruritic macules to papules and vesicles (rarely bullae) that finally crust over within 48 hours. The classic lesions are teardrop vesicles surrounded by an erythematous ring ("dewdrop on a rose petal"). The most common complication of varicella is secondary bacterial skin infection, usually with S pyogenes or S aureus. Other complications from varicella include encephalitis, glomerulonephritis, hepatitis, pneumonia, arthritis, and meningitis. Cerebellitis (manifested clinically as ataxia) may develop and is usually self-limited. Other viral infections that may manifest with vesicular rashes include herpes simplex, zoster, coxsackie, influenza, echovirus, and vaccinia. On occasion, varicella can be confused with papular urticaria.

Management and Disposition

Suspected varicella infection should lead to strict isolation early in the emergency department or office encounter. Most children have a self-limited illness and do not develop any complications. Treatment should be supportive and directed to pruritus and fever control while avoiding salicylates because of their association with Reye syndrome. Oral acyclovir initiated within 24 hours of the onset of the rash may result in a modest decrease in the duration of symptoms and in the number and duration of skin lesions. Acyclovir is not recommended routinely for treatment of uncomplicated varicella in an otherwise healthy child less than 12 years of age. In the immunocompromised host, varicella zoster immunoglobulin (VZIG), IV acyclovir, and hospital admission are indicated.

Pearls

1. Skin lesions in varicella present in successive crops so that macules, papules, vesicles, and crusted lesions may all be present at the same time.
2. Healthy children are no longer contagious when all lesions have crusted over (usually 4-5 days from the development of the initial lesions).
3. Consider oral acyclovir for those at risk for more severe infection, including anyone older than 12 years, with chronic diseases, and taking chronic aspirin or corticosteroid therapy. Postexposure prophylaxis includes VZIG within 96 hours of exposure in high-risk patients (immunocompromised patients or pregnant women) or immunization with varicella vaccine within 72 hours of exposure in non–high-risk patients.

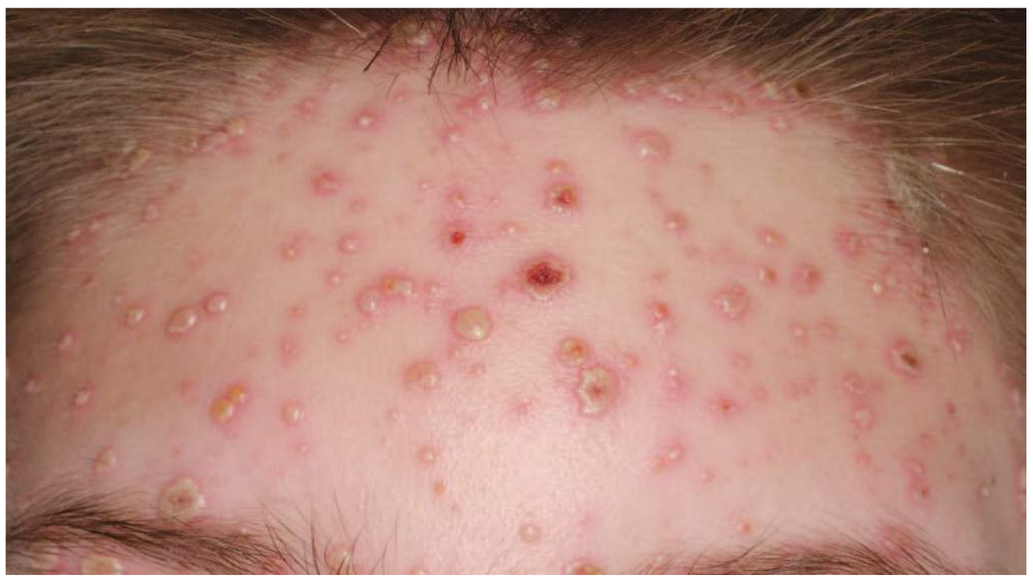


FIGURE 14.28 ■ **Varicella (Chickenpox).** Multiple cloudy vesicles of varicella. (Photo contributors: Katharine Hanlon and Kara Shah, MD.)

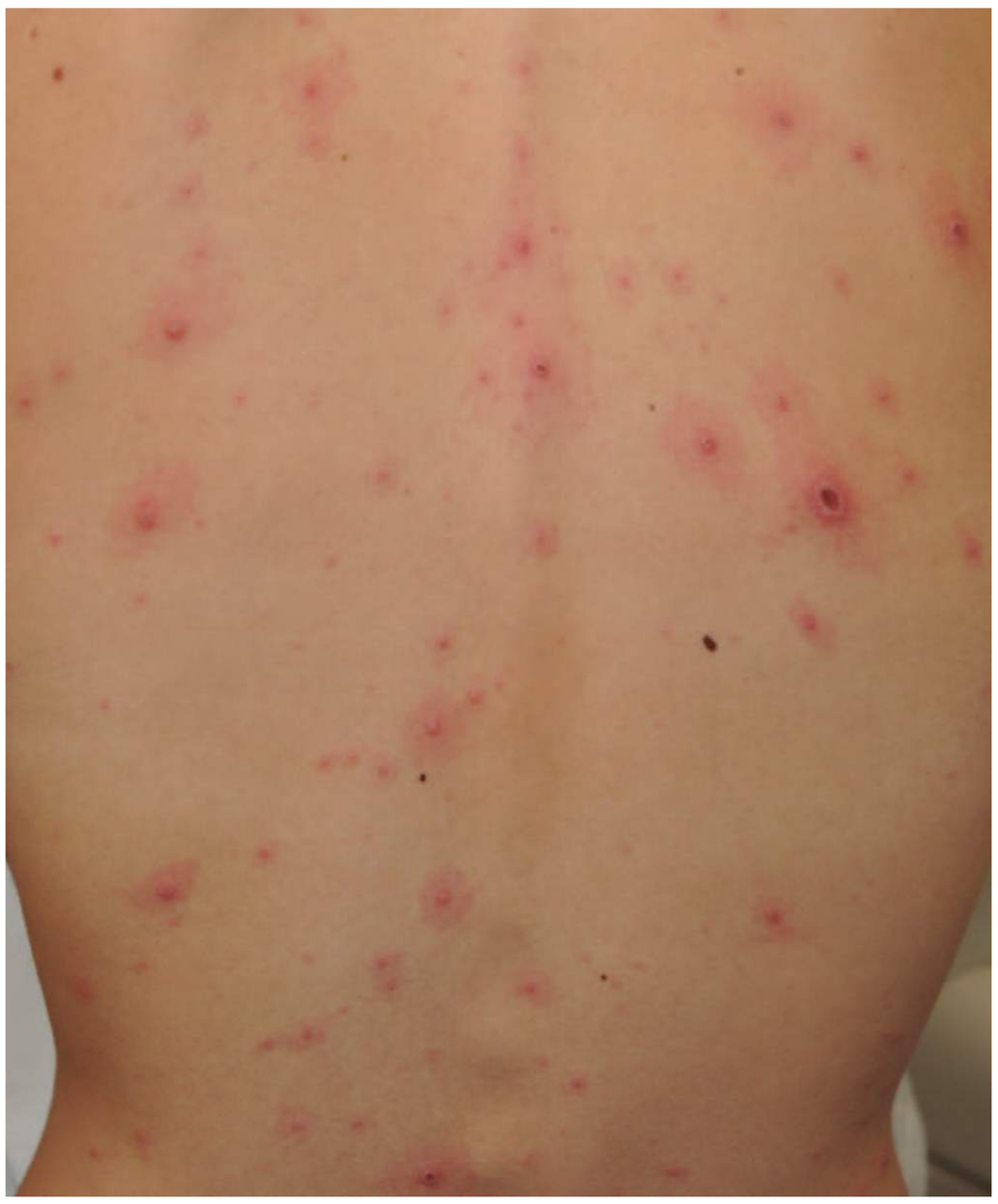


FIGURE 14.29 ■ **Varicella (Chickenpox).** Vesicles in different stages of maturation. Note vesicles surrounded by an erythematous ring ("dewdrop on a rose petal"). (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Zoster (shingles) represents a reactivation of latent VZV and has been noted as early as the first week of life in infants born to mothers who contracted varicella during pregnancy. The lesions present as clustered vesicles or bullae in a dermatomal distribution. The pain of acute neuritis occurs in 75% of patients with herpes zoster. Prodromal pain can be constant or intermittent, burning or stabbing and can precede the lesions by days to weeks. Sometimes pain can persist beyond 1 month after the lesions have disappeared (known as postherpetic neuralgia).

The diagnosis is usually made clinically; however, tissue cultures, direct fluorescent antibodies, and Tzanck smears can be done from vesicle scrapings for confirmation. Impetigo and cutaneous burns may mimic the appearance of herpetic vesicles. Varicella (chickenpox) is more diffusely spread, although a small crop of lesions may mimic zoster. Zoster also may be confused with herpes simplex virus (HSV) infection, although a close examination should reveal a dermatomal distribution in zoster.

Management and Disposition

Currently, acyclovir (800 mg orally five times per day for 7-10 days initiated within 48 hours of the onset of the rash in children ≥12 years) is the treatment of choice for zoster infections in immunocompetent children. Pain relief and prevention of secondary infection are also important. IV antiviral therapy is recommended for immunocompromised patients. There is no pediatric formulation of famciclovir or valacyclovir, and there are insufficient data on their use in children.

Pearls

1. Zoster can occur in children of all ages.
2. The most common sites for the development of zoster lesions are those supplied by the trigeminal nerve and the thoracic ganglia.
3. A patient with zoster can transmit chickenpox (varicella) to a nonimmune child or adult.
4. Herpes zoster ophthalmicus is a sight-threatening condition that presents with hyperesthesia in the effected eye. Vesicular lesions on the nose (Hutchinson sign) are associated with an increased risk of eye involvement.

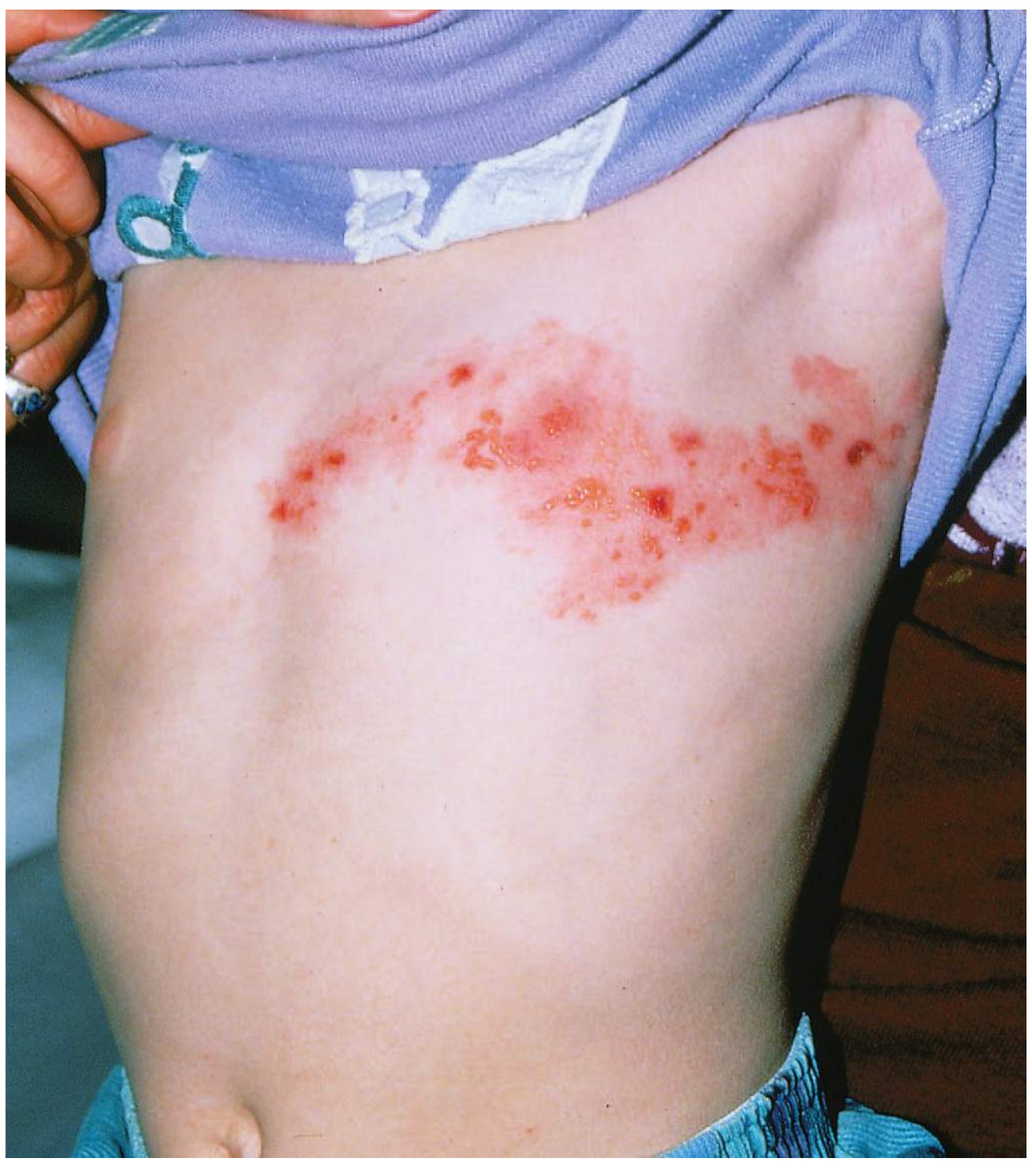


FIGURE 14.30 ■ **Herpes Zoster.** Vesicles in a classic thoracic dermatomal distribution are seen in this child. (Photo contributor: Frank Birinyi, MD.)

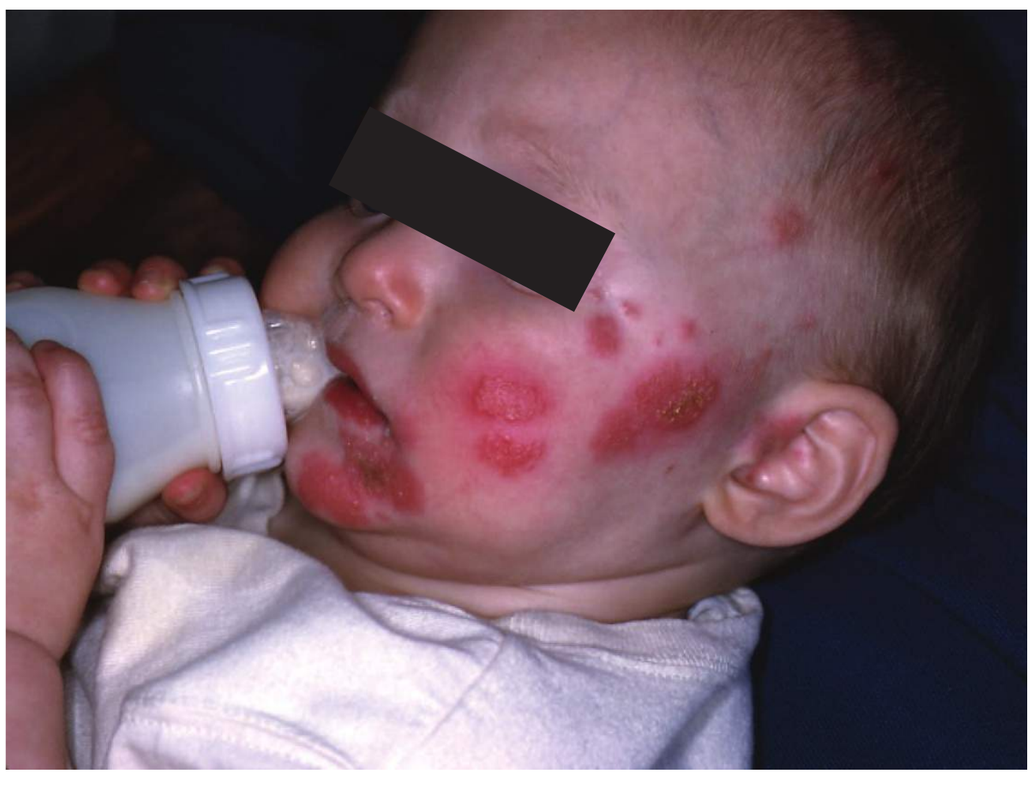


FIGURE 14.31 **Herpes Zoster.** Vesicles in a trigeminal nerve distribution. (Photo contributor: Anne W. Lucky, MD.)

HAND, FOOT, AND MOUTH SYNDROME

Clinical Summary

Hand, foot, and mouth syndrome is a seasonal (summer-fall) viral infection caused by coxsackievirus A16. Toddlers and school-aged children are affected most commonly. Adults may also be affected. It is characterized by a prodrome of fever, malaise, sore throat, and anorexia over 1 to 2 days, followed by the appearance of the characteristic enanthem in the posterior oropharynx and tonsillar pillars consisting of small, red macules evolving into small vesicles 1 to 3 mm in diameter that rapidly ulcerate. Oral manifestations are followed by a vesicular eruption characterized by 3- to 7-mm erythematous macules with a central gray vesicle on the hands and feet involving the palmar and plantar surfaces and interdigital surfaces. Nonvesicular rash may also be present on the buttocks, face, and legs.

Management and Disposition

Supportive therapy (hydration maintenance with fever and pain control) is the mainstay of treatment. Discuss the duration and characteristics of the illness with the parents. In most cases, the course is self-limited resolving in 2 to 3 days after the appearance of rash without further complication. Rare secondary complications such as myocarditis, pneumonia, pulmonary hemorrhage, and meningoencephalitis may occur.

Pearls

1. The child is contagious until all vesicles have resolved.
2. The oral lesions tend to involve the posterior oropharynx as contrasted with those of herpetic gingivostomatitis which typically involve the anterior structures of the mouth.
3. Varicella vesicles are located more centrally, are more extensive, and usually spare the palms and soles.

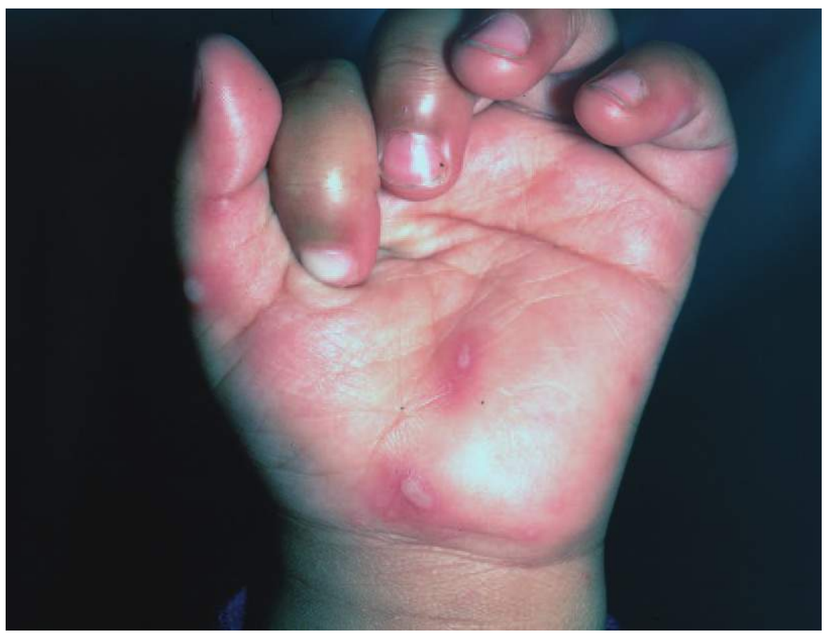


FIGURE 14.32 ■ **Hand, Foot, and Mouth Disease.** Erythematous vesicular rash scattered on the palms consistent with coxsackievirus. (Photo contributor: Lawrence B. Stack, MD.)

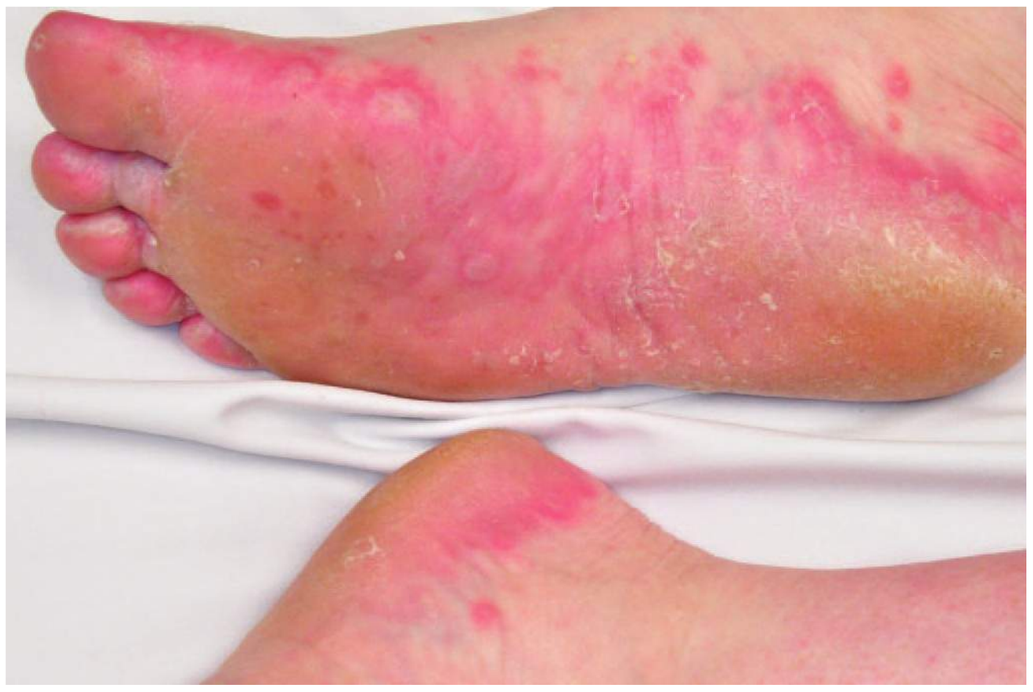


FIGURE 14.33 ■ **Hand, Foot, and Mouth Disease.** Vesicular rash of the feet consistent with coxsackievirus. (Photo contributor: Larry B. Mellick, MD.)

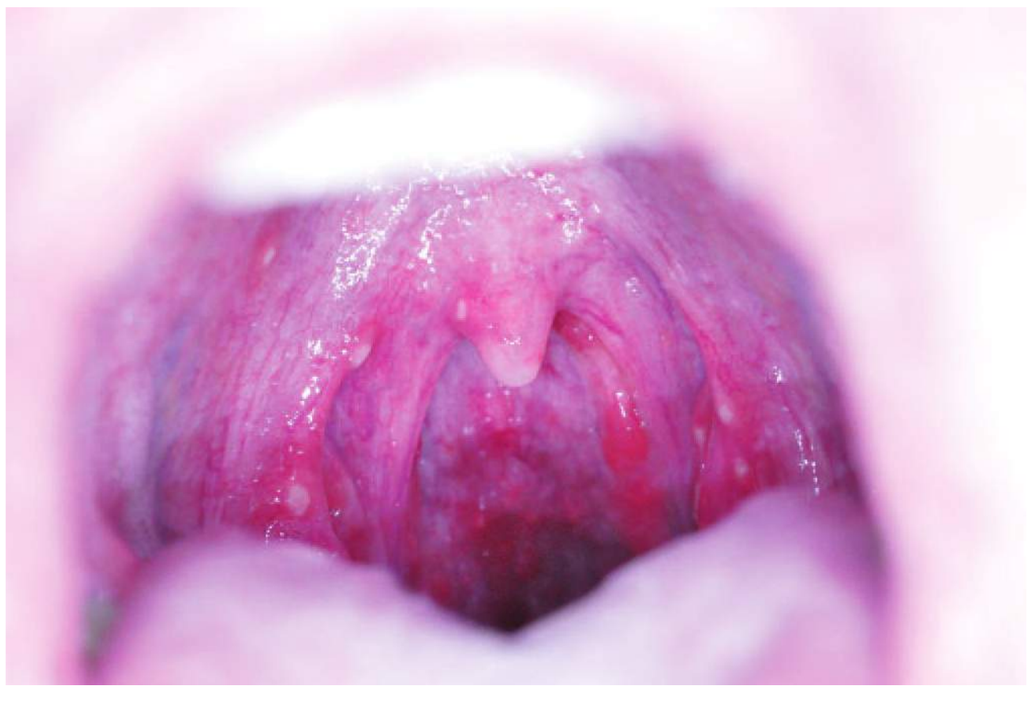


FIGURE 14.34 ■ **Hand, Foot, and Mouth Disease.** Discrete vesicular erosions on the posterior oropharynx and soft palate secondary to coxsackievirus. (Photo contributor: Larry B. Mellick, MD.)

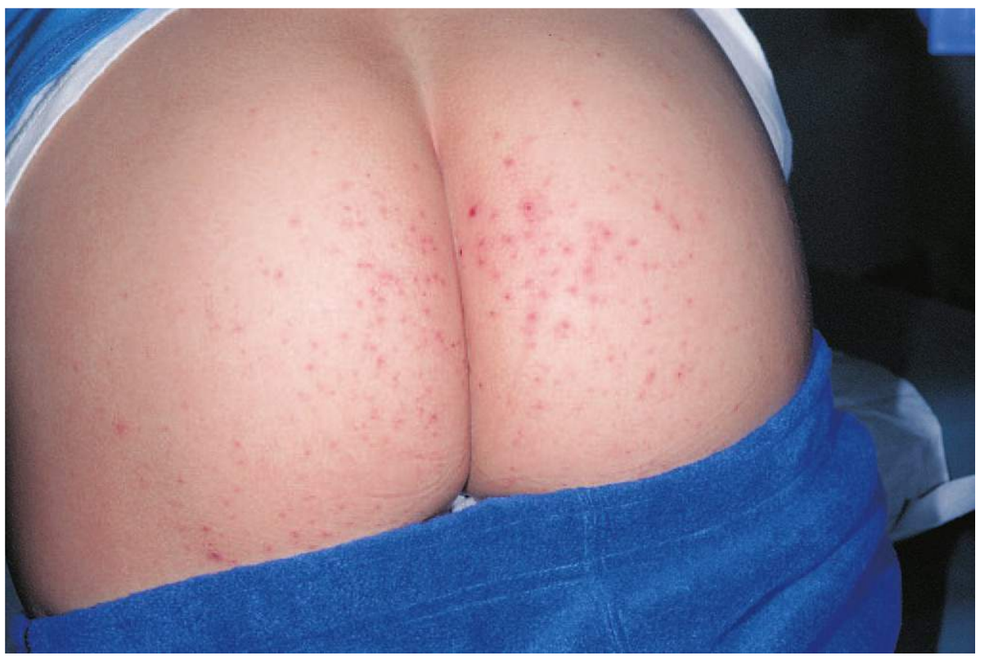


FIGURE 14.35 ■ **Hand, Foot, and Mouth Disease.** Nonvesicular rash on the buttocks, a common site. (Photo contributor: Binita R. Shah, MD. Used with permission from Shah B, Lucchesi M, Amodio J, Silverberg M. Atlas of Pediatric Emergency Medicine. 2nd ed. New York, NY: McGraw-Hill; 2013: Fig. 3.66D, p. 111.)

COLD PANNICULITIS (POPSICLE PANNICULITIS)

Clinical Summary

Cold panniculitis represents acute cold injury resulting in inflammation of the subcutaneous fat. It manifests as erythematous, indurated nodules, and plaques on exposed skin, especially the perioral areas and cheeks. Lesions appear 24 to 72 hours after exposure to cold and gradually soften and return to normal over 1 to 2 weeks usually without permanent sequelae. This phenomenon is caused by subcutaneous fat solidification and necrosis following exposure to cold temperatures. It is much more common in infants. The differential diagnosis includes facial cellulitis, frostbite, trauma, pressure erythema, giant urticaria, and contact dermatitis.

Management and Disposition

Treatment is supportive. Parental education and reassurance is important.

Pearls

1. Because these lesions may also be painful, the differentiation of cold panniculitis from cellulitis may be difficult. The absence of systemic symptoms, especially fever, and the history of cold exposure are more suggestive of cold panniculitis.
2. The lesions may resolve with resulting hyperpigmentation of the affected area.

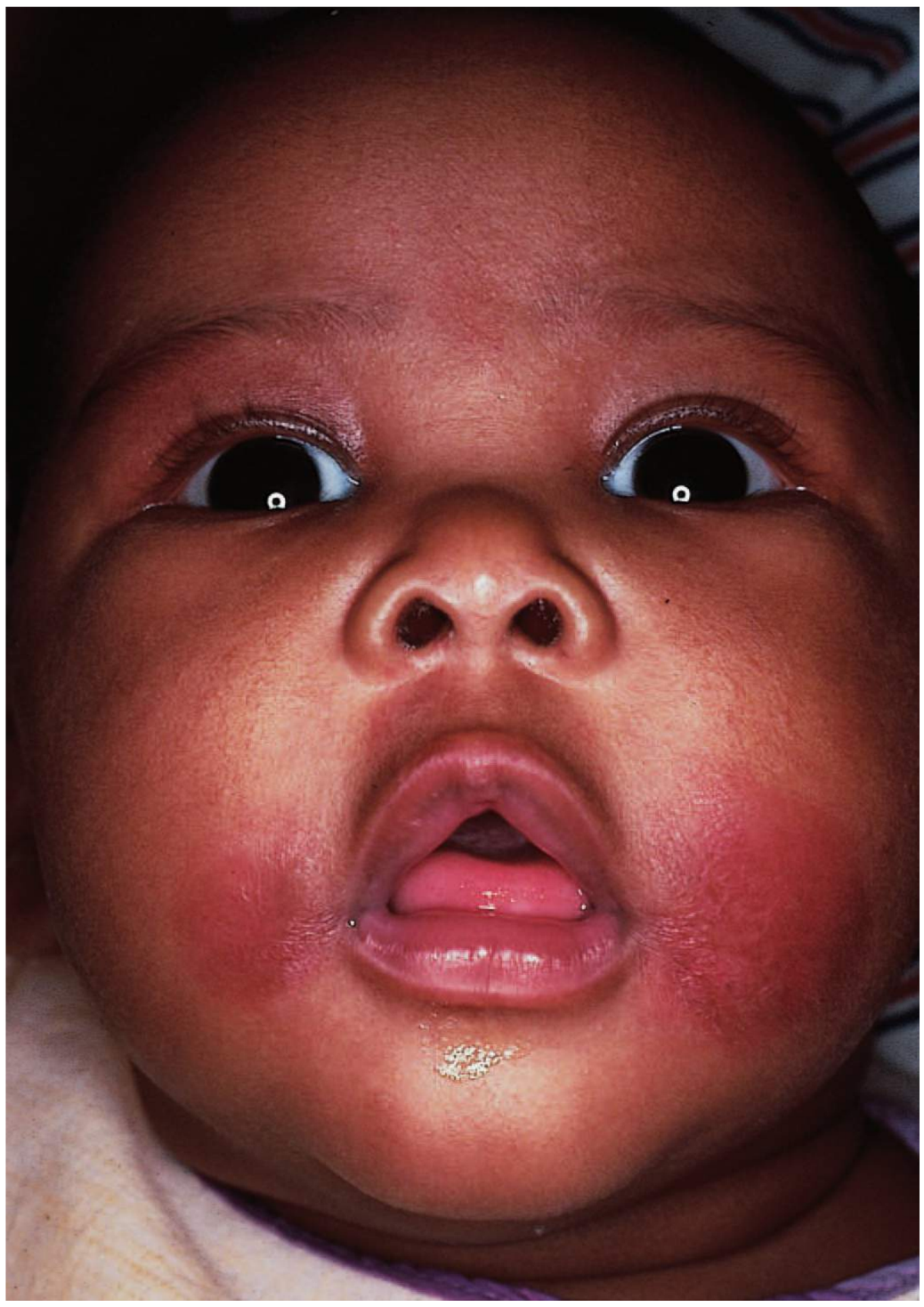


FIGURE 14.36 ■ **Cold Panniculitis.** Infant with cheek erythema, swelling, and discoloration consistent with popsicle panniculitis or cold injury. (Photo contributor: Anne W. Lucky, MD.)

HERPETIC GINGIVOSTOMATITIS

Clinical Summary

Herpetic gingivostomatitis is primary infection caused by HSV seen in up to 30% of children between 6 months and 5 years of age. Patients usually present with approximately 4 days of fever, malaise, decreased oral intake, cervical adenopathy, and pain in the mouth and throat. Following the prodrome, vesicular and ulcerative lesions appear throughout the oral cavity. The gingiva becomes very friable and inflamed, especially around the alveolar rim. Increased salivation, foul breath, and cervical lymphadenitis may be present. Although fever resolves in 3 to 5 days, children may have difficulty eating for 7 to 14 days. Sometimes, autoinoculation produces vesicular lesions on the fingers (herpetic whitlow).

Management and Disposition

Treatment includes pain control, hydration, and consideration of oral acyclovir therapy. The pain may be significant and often requires oral narcotic pain medications. Control of the pain will allow the patient to consume fluids and remain well hydrated. Acyclovir has been shown to reduce the duration of pain, gingival swelling, oral lesions, fever, and viral shedding if initiated within the first 72 to 96 hours of illness. Avoidance of citrus juices or spicy food is recommended. Cold clear fluids, popsicles, and ice cream may be useful in small children. Not infrequently, admission for IV hydration is necessary. Topical pain control may be achieved by using mixtures of antihistamine (diphenhydramine elixir) and antacid (1:1) applied to lesions with a cotton swab. Local application of viscous lidocaine should be avoided in children, since patients may develop toxic serum levels due to altered absorption from inflamed oral mucosa leading to seizures.

Pearls

1. Most lesions are in the anterior two-thirds of the oral cavity. Posterior lesions sparing the gingiva are most commonly seen in coxsackievirus infections.
2. Primary HSV infection in childhood is usually asymptomatic.
3. After primary oral infection, HSV remains latent in the trigeminal ganglion until reactivation as herpes labialis.

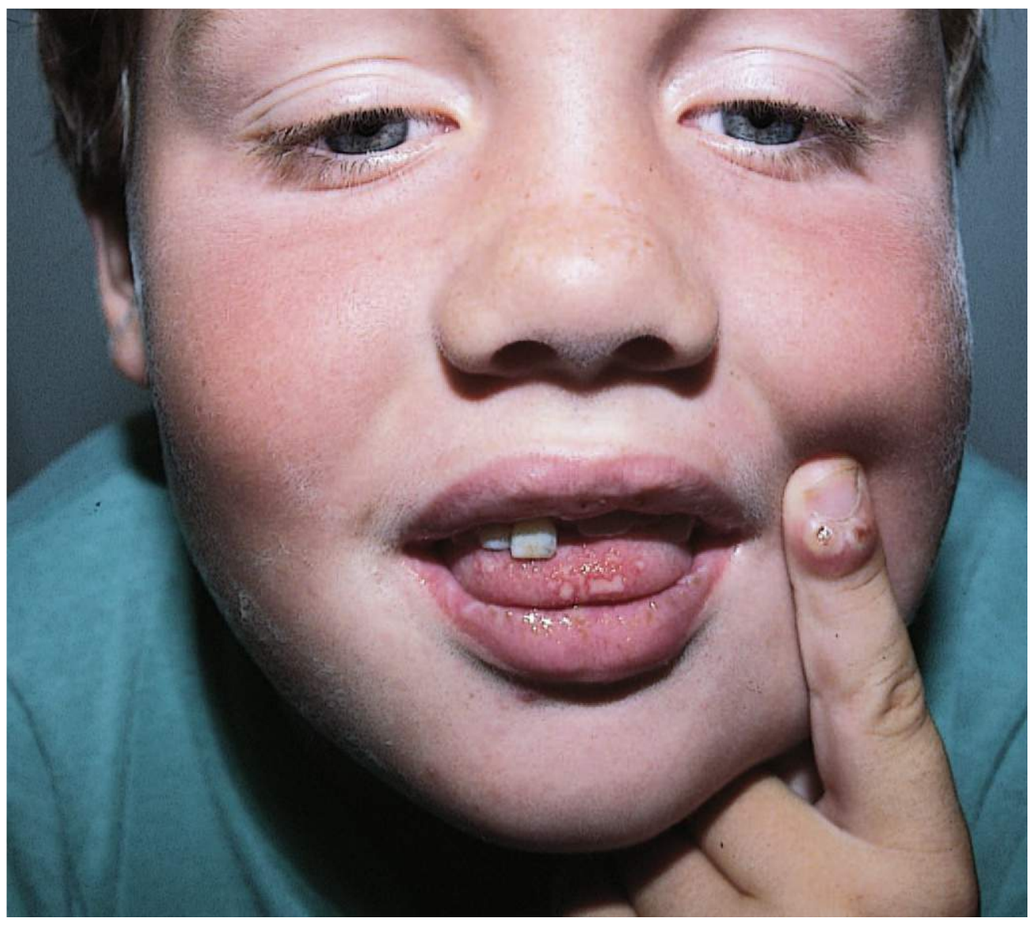


FIGURE 14.37 ■ **Herpetic Gingivostomatitis.** Multiple oral vesicular lesions and tongue ulcerations consistent with herpes gingivostomatitis. Vesicular lesions from autoinoculation are present on the finger (herpetic whitlow). (Photo contributor: Michael J. Nowicki, MD.)

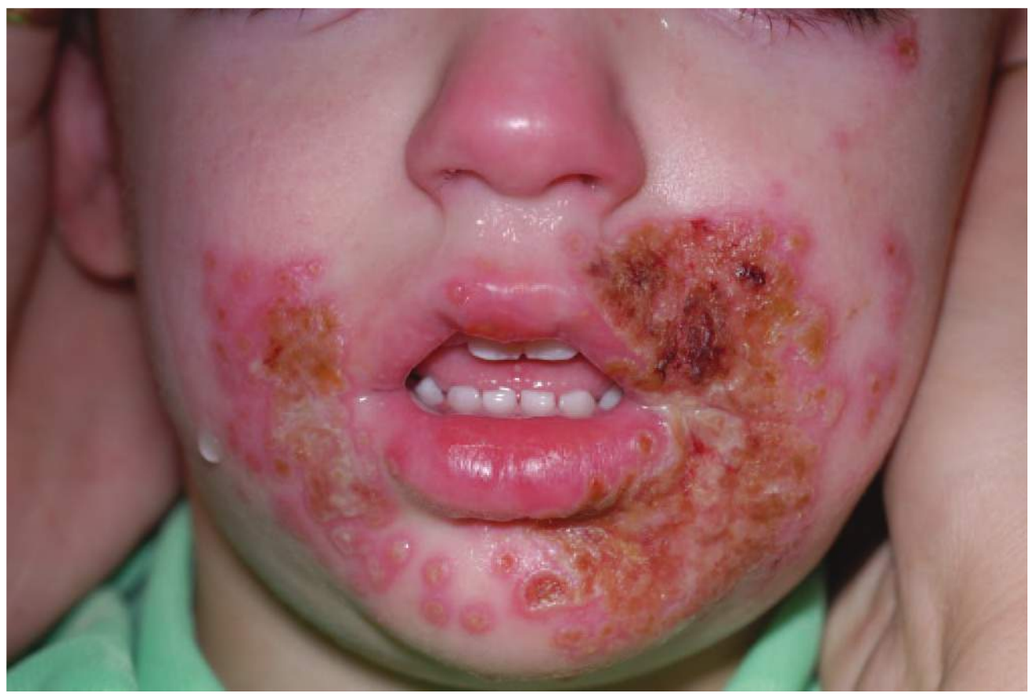


FIGURE 14.38 ■ **Herpetic Stomatitis.** Multiple perioral vesicular lesions consistent with herpes gingivostomatitis. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Meningococcemia is an acute febrile illness caused by Neisseria meningitidis bacteremia characterized by its generally rapid onset, significant toxicity, and petechial rash involving the skin and mucous membranes. The petechiae progress to become palpable purpura and may coalesce to become purpura fulminans. It progresses rapidly to decompensated septic shock with hypotension and multisystem organ failure. In cases of fulminant disease, progressive shock is accompanied by disseminated intravascular coagulation and massive mucosal hemorrhages. Prodromal symptoms may include cough, coryza, headache, neck pain, and malaise. Children less than 5 years of age and college students not receiving the meningococcal vaccine during high school are at greatest risk.

Management and Disposition

In stable patients in which meningococcemia is in the differential diagnosis, obtain cultures of blood and spinal fluid and from the nasopharynx, along with a CBC and coagulation studies. Consider bedside screening studies (eg, blood gas to assess acid-base status), liver function tests, and other studies as clinically indicated. These patients should be admitted for intensive monitoring to institutions capable of delivering critical care services. Broad-spectrum parenteral antibiotics should be administered initially until the organism is identified and sensitivities are available as with any patient with suspected sepsis. In the unstable septic patient, support end organ perfusion and oxygenation via early goal-directed therapy. Hemodynamic monitoring and blood pressure support (fluids and vasoactive drugs) are of paramount importance. Peripheral and central venous catheters and urinary and arterial catheters are usually necessary for optimal management of these patients.

Pearls

1. Skin scrapings of the purpuric lesion can be cultured and microscopically examined for the presence of gram-negative diplococci.
2. A child with a fever and an evolving petechial rash is presumed to have meningococcemia until proven otherwise.
3. A quadrivalent meningococcal conjugate vaccine is now available and recommended for all adolescents between ages 11 and 12 and then again at age 15 years or high school entry (whichever comes first).
4. Prophylaxis for all close contacts is recommended with rifampin for 48 hours or a single dose of ceftriaxone or ciprofloxacin.

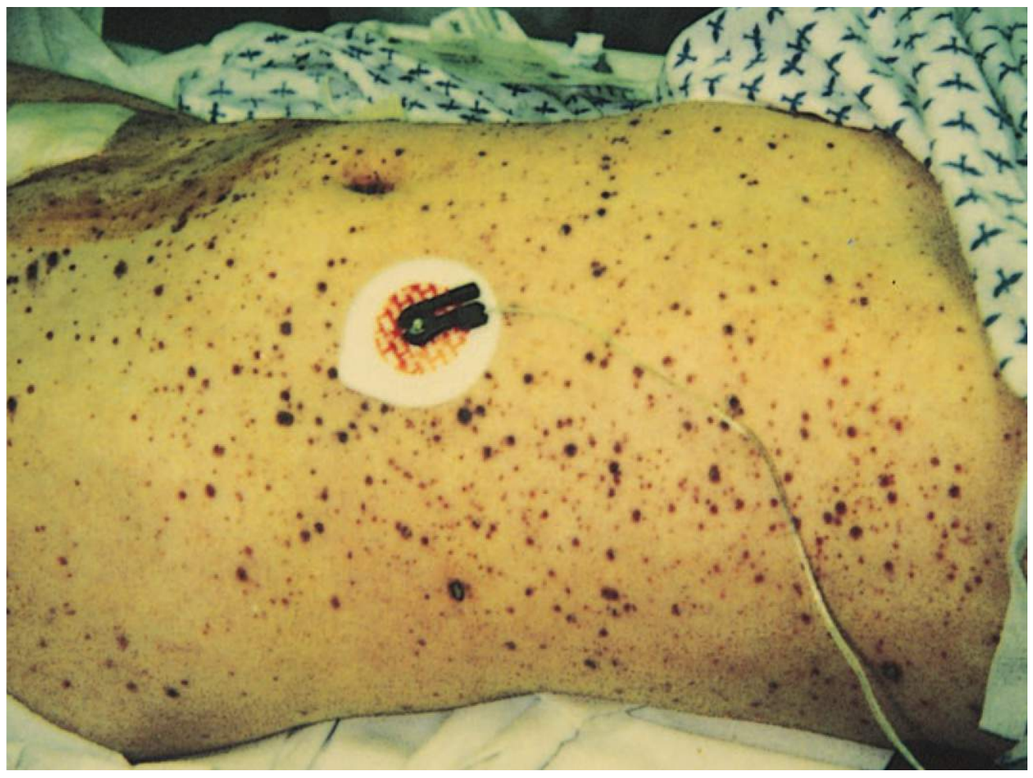


FIGURE 14.39 ■ **Meningococcemia.** Diffuse petechiae in a patient with meningococcemia. (Photo contributor: Richard Strait, MD.)

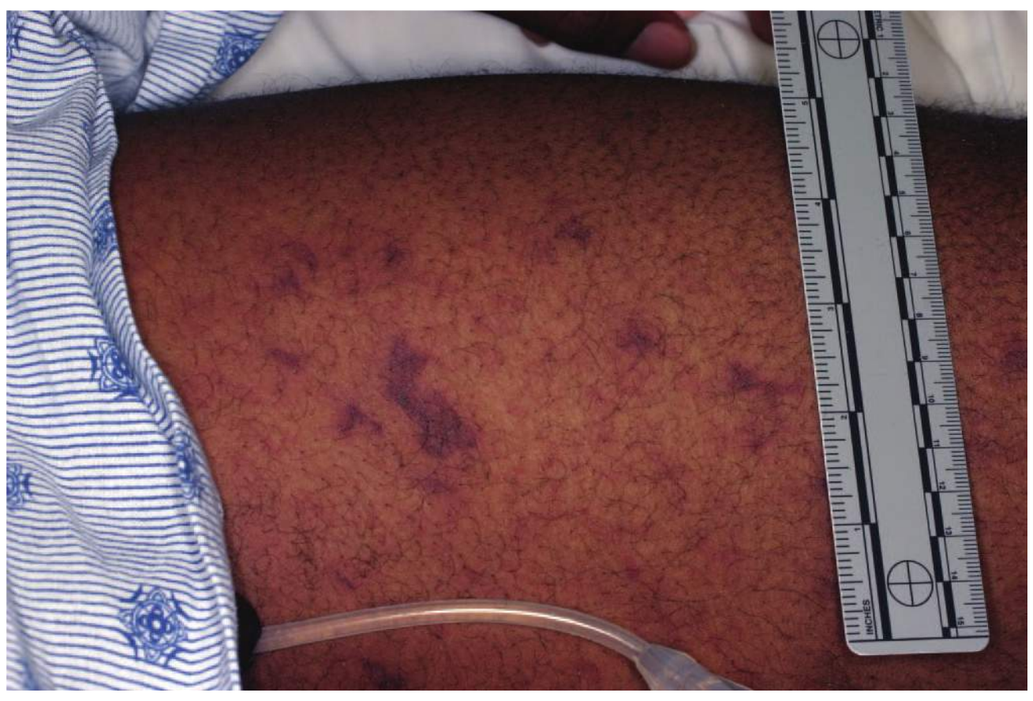


FIGURE 14.40 ■ **Meningococcemia.** Petechiae and purpura in an adolescent patient with meningococcemia. (Photo contributor: Kevin J. Knoop, MD, MS.)

Clinical Summary

Acute urticaria (defined as <6 weeks' duration) is a common condition in children caused by inflammatory mediator (histamine and others) release from mast cells and basophils in the superficial dermis. These lesions are often associated with an infection, an insect sting or bite, or ingestion of certain foods or medications, though in many cases the precise etiology cannot be identified. It is characterized by the sudden onset of intensely pruritic lesions that are raised, erythematous, well-circumscribed, and blanching. They may occur anywhere on the body and can vary in size from pinpoint to several centimeters in diameter. They can have central clearing or associated edema. Individual lesions usually resolve in 1 to 3 hours, but some may last for up to 24 to 48 hours. Rarely, they may take weeks to resolve completely.

Anaphylaxis is a systemic allergic reaction defined by multiorgan system involvement that can have an urticarial component along with wheezing, stridor, angioedema, vomiting, and hypotension. The differential diagnosis for urticaria includes erythema multiforme, Henoch-Schönlein purpura (HSP), arthropod bites, contact dermatitis, reactive erythemas, allergic vasculitis, juvenile rheumatoid arthritis, mastocytosis, and pityriasis rosea.

Management and Disposition

Treatment is symptomatic. If the offending agent can be identified, avoidance is recommended. Oral antihistamines are useful in the control of pruritus and often lead to improvement or complete resolution of the rash within an hour of administration. As the effect of the antihistamine wanes, the urticaria will often recur. If the patient has systemic signs suggesting anaphylaxis, administer intramuscular (IM) epinephrine as soon as possible. Antihistamines (H1 and H2 blockers), steroids, and IV fluids may also be helpful. Unless there is evidence of acute angioedema or anaphylaxis, most cases can be discharged home on oral antihistamines to follow up with a primary care physician. Specific allergen testing is not recommended in the emergency department.

Pearls

1. Ninety percent of patients with anaphylaxis have cutaneous reactions, which are often urticarial. IM epinephrine is the only medication that decreases the risk of mortality in anaphylaxis.
2. By definition, a lesion of urticaria must change or resolve within 24 hours of its appearance.
3. The most common causes of urticaria in children are viral illnesses and foods. Egg, milk, soy, peanuts, and wheat are the most common allergens in young children. In older children, fish, seafood, nuts, and peanuts are more common causes.
4. The addition of an H2 antagonist (ranitidine or famotidine) may be helpful when H1 agents alone are ineffective.

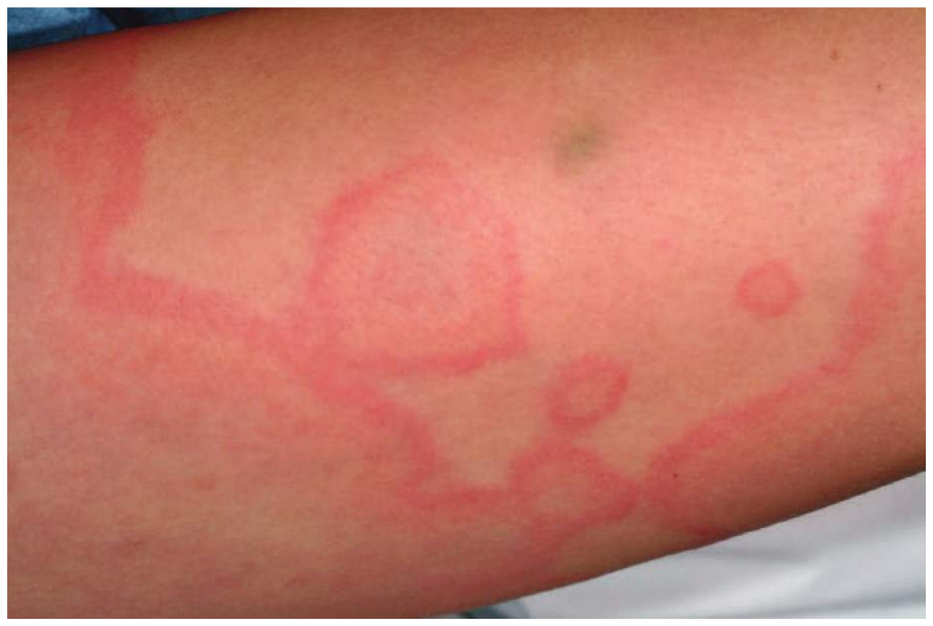


FIGURE 14.41 ■ **Urticaria.** Well-circumscribed, raised, pruritic extremity lesions consistent with urticaria. (Photo contributor: Anita P. Sheth, MD.)

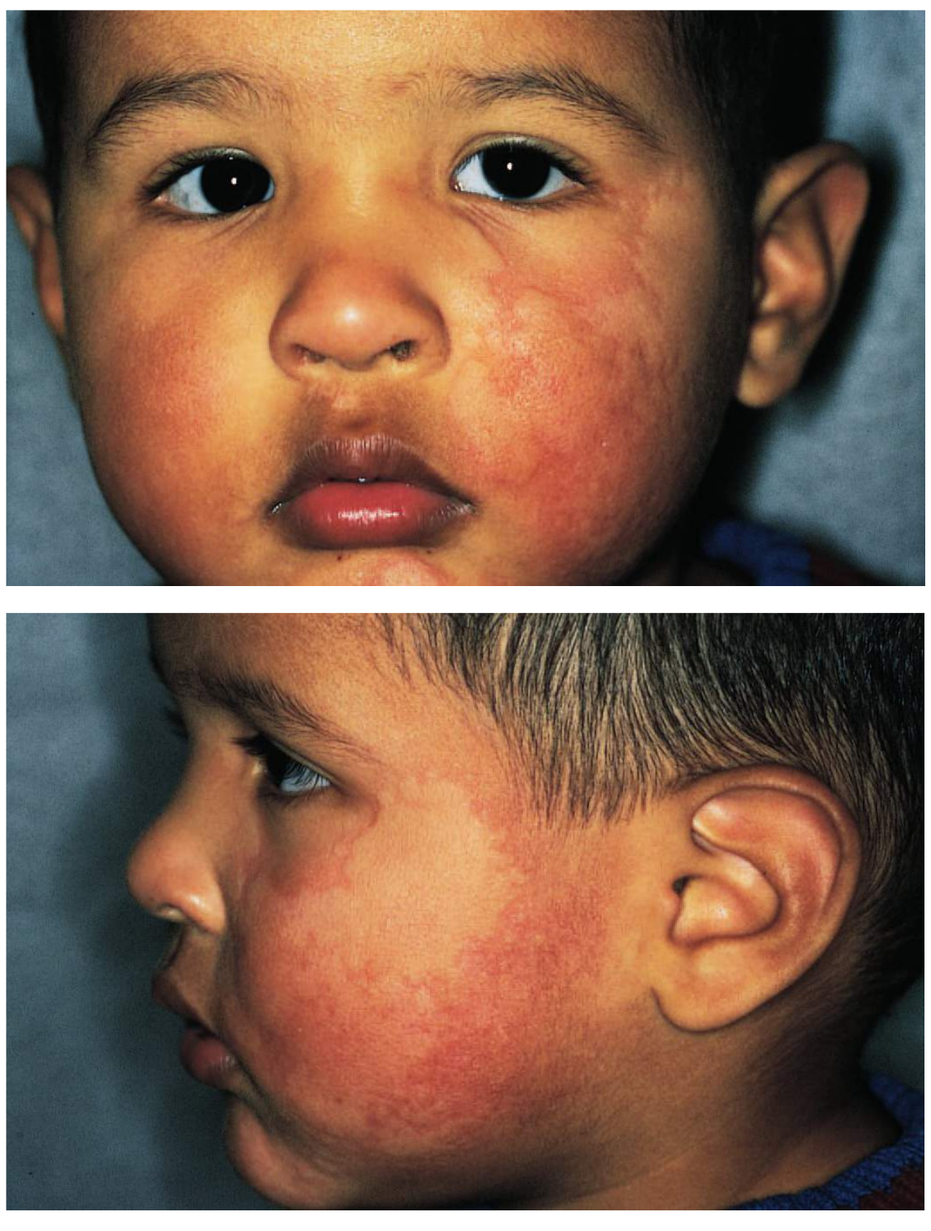


FIGURE 14.42 ■ **Urticaria.** Preschool child with annular, raised pruritic lesions with the central clearing and tense edema of polycyclic urticaria. The lesions had completely disappeared after about 5 minutes. (Photo contributor: Kevin J. Knoop, MD, MS.)

Clinical Summary

Summer penile syndrome is a benign hypersensitivity reaction involving the skin of the penile shaft. As the name suggests, it is seen during the warm weather months. It is also known as seasonal acute hypersensitivity reaction and lion's mane penis. The history may include recent play outside in the grass or a wooded area within the last 24 hours. It is most commonly associated with insect bites, usually chiggers, but may also be caused by exposure to plants (poison ivy, sumac, and oak). The diagnosis is made clinically and is suspected when the skin of the shaft of the penis, most often just proximal to the glans, is markedly edematous. There is minimal erythema and no fluctuance. Four out of five patients have pruritus and few have urinary symptoms. Symptoms (both swelling and pruritus) can last up to 2 to 3 weeks, but most patients have resolution in 4 to 5 days. The differential diagnosis includes trauma, nephrotic syndrome, HSP, balanitis, phimosis, paraphimosis, and priapism.

FIGURE 14.43 ■ **Summer Penile Syndrome.** Edema of the distal penile shaft and left scrotum. An insect bite is seen at the base of the left mons pubis. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

Treatment consists of an oral antihistamine if the patient has pruritus and cool compresses. Systemic corticosteroids should only be considered if the child is in significant pain or is having trouble with the voiding stream. Patients can be discharged home after education and reassurance. Urology involvement and referral is usually not necessary.

Pearls

1. Summer penile syndrome is usually painless.
2. Pruritus is the most common symptom.
3. Testing is unnecessary, but the diagnosis must be distinguished from paraphimosis.

FIGURE 14.44 ■ **Summer Penile Syndrome.** Also known as "lion's mane penis" due to the marked circumferential edema just proximal to the glans. (Photo contributor: Larry B. Mellick, MD.)

STAPHYLOCOCCAL SCALDED SKIN SYNDROME

Clinical Summary

Staphylococcal scalded skin syndrome (Ritter disease) most commonly affects infants and children less than 5 years of age and is caused by an exfoliative exotoxin-producing strain of S aureus. Initial presentation includes fever, malaise, and irritability following an upper respiratory infection with pharyngitis or conjunctivitis. Patients first develop a diffuse faint blanching erythematous rash that is tender to the touch. Crusting around the mouth, eyes, and neck may be seen. Within 2 to 3 days, flaccid blisters and bullae develop especially in flexor creases, the buttocks, hands, and feet. These bullae are sterile. In some patients, widespread desquamation occurs. The differential diagnosis includes toxic epidermal necrolysis, exfoliative erythroderma, bullous erythema multiforme, bullous pemphigoid, bullous impetigo, sunburn, acute mercury poisoning, toxic shock syndrome, and epidermolysis bullosa.

Management and Disposition

If staphylococcal scalded skin syndrome is suspected, obtain cultures from the blood, urine, nasopharynx, abnormal area of skin, or other suspected foci of infection. Treatment is directed at the eradication of S aureus, thus terminating the production of toxin. Semisynthetic penicillinase-resistant penicillins or clindamycin should be used intravenously. Consider the addition of vancomycin in areas with a high prevalence of methicillin-resistant S aureus (MRSA). Admission is usually necessary, especially in young infants. This age group requires careful attention to fluid and electrolyte losses and the prevention of secondary infection of the denuded skin. Management of pain associated with the rash may require narcotics.

Pearls

1. The wrinkling or peeling of the upper layer of the epidermis (pressure applied with a Q-tip or gloved finger) that occurs within 2 or 3 days of the onset of this illness is known as Nikolsky sign.
2. The fluid in the bullae of staphylococcal scalded skin syndrome is sterile. The toxin is produced at a remote site and delivered to the skin via the bloodstream.
3. Infants with involvement of large areas of the body surface are at increased risk for hypothermia and fluid/electrolyte losses.
4. Corticosteroids are contraindicated in the treatment of staphylococcal scalded skin syndrome.
5. These lesions do not typically scar as the cleavage plane is intraepidermal.

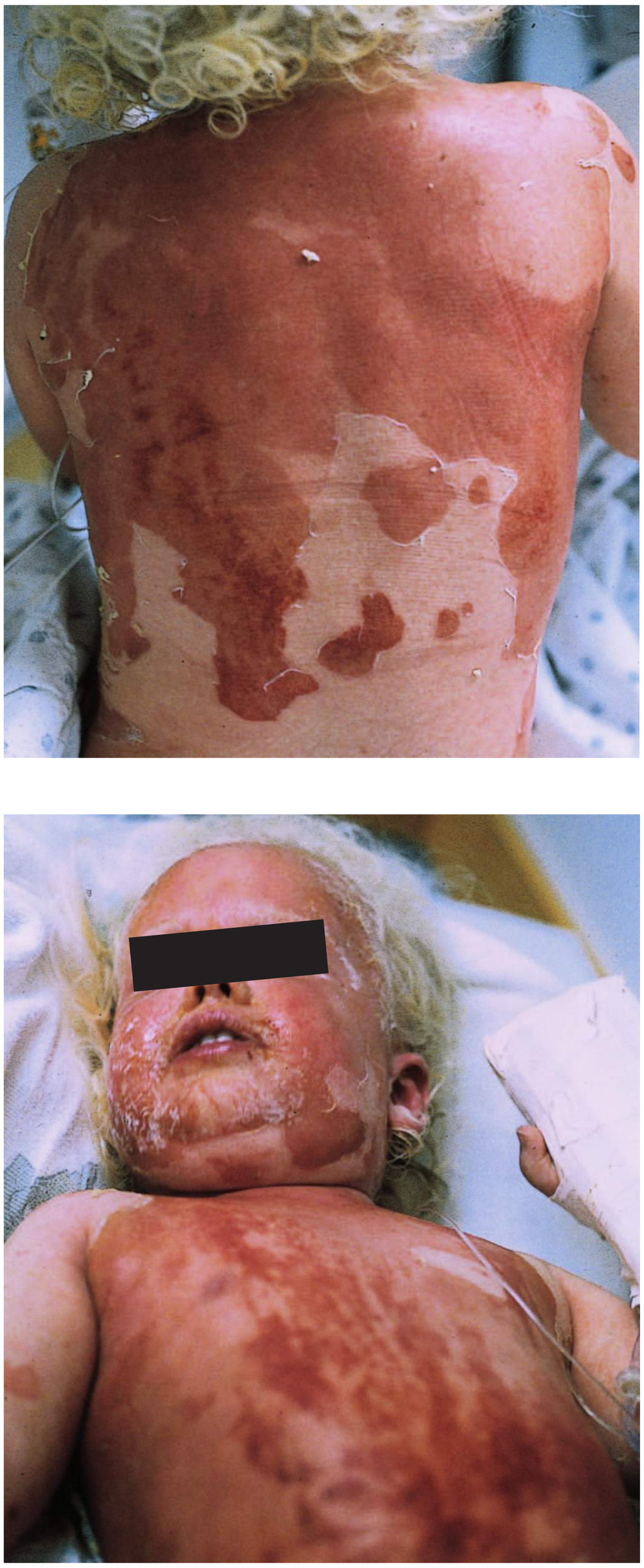


FIGURE 14.45 ■ **Staphylococcal Scalded Skin Syndrome.** Toddler with diffuse macular peeling eruption consistent with scalded skin syndrome from S aureus. (Photo contributor: Judith C. Bausher, MD.)

Clinical Summary

Scarlet fever manifests as diffuse blanching "sandpaper-like" erythematous macules and papules caused by erythrogenic toxin production from group A β-hemolytic Streptococcus pharyngitis. Occasionally, the site of infection is skin (impetigo) or perianal. The disease usually occurs in children 2 to 10 years of age. The typical presentation of scarlet fever includes fever, headache, sore throat, nausea, vomiting, and malaise followed by the characteristic scarlatiniform rash. The rash initially occurs centrally on the face (often with perioral sparing), neck, and upper trunk but quickly becomes generalized. Desquamation occurs after 5 to 7 days. On the tongue, a thick, white coat and swollen papillae may be seen ("strawberry tongue"). Palatal petechiae and tender anterior cervical lymphadenopathy may be present. The differential diagnosis includes enteroviral infections, staphylococcal scalded skin syndrome, viral hepatitis, infectious mononucleosis, toxic shock syndrome, drug eruptions, rubella, mercury poisoning, and Kawasaki disease.

Management and Disposition

Penicillin, either a single IM dose of benzathine penicillin G or oral amoxicillin for 10 days, is the treatment of choice. Alternatives include erythromycin or clindamycin in penicillin-allergic patients.

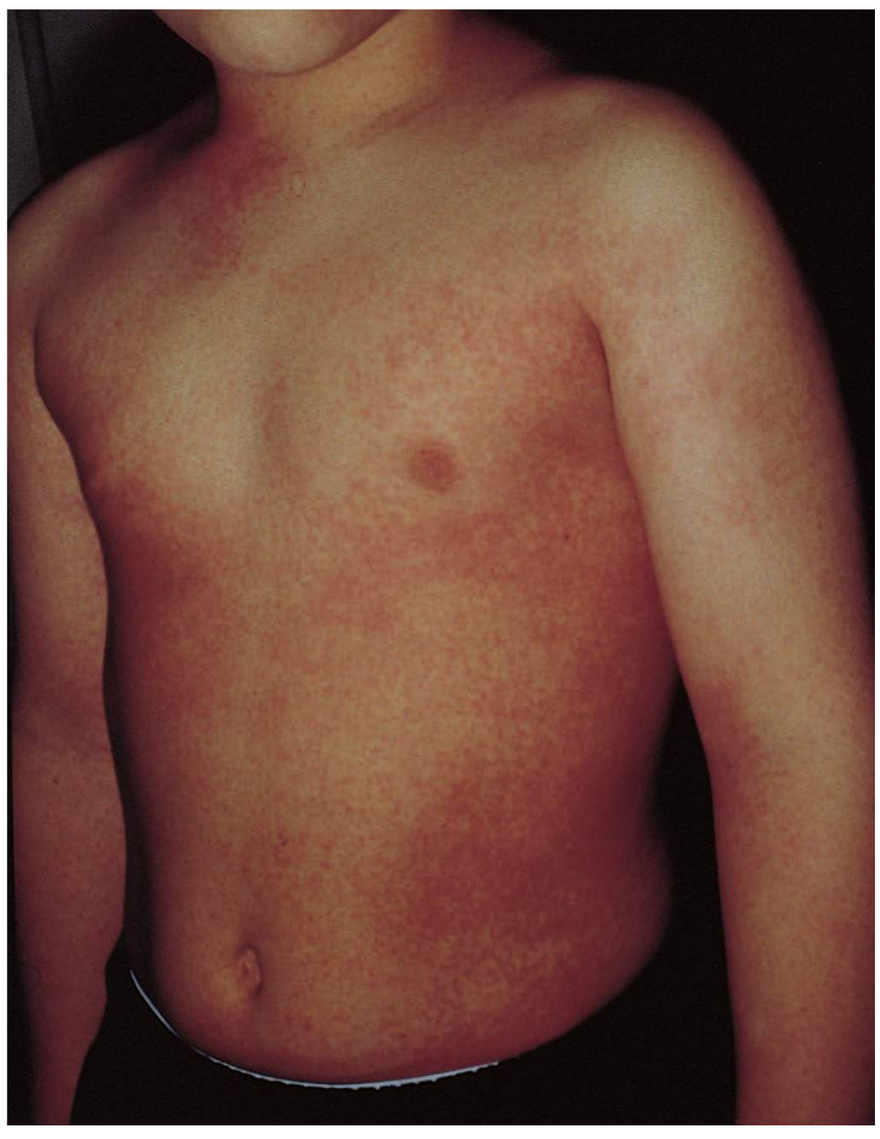


FIGURE 14.46 ■ **Scarlatina.** Erythematous scarlatiniform rash of scarlet fever. (Photo contributor: Lawrence B. Stack, MD.)

Pearls

1. In scarlet fever, petechiae in a linear pattern along the major skin folds in the axillae and antecubital fossae are known as "Pastia lines."
2. In dark-skinned individuals, the rash may be difficult to differentiate and may consist only of punctate papular elevations called "goose flesh."
3. There has never been an isolate of group A Streptococcus that is resistant to penicillin.

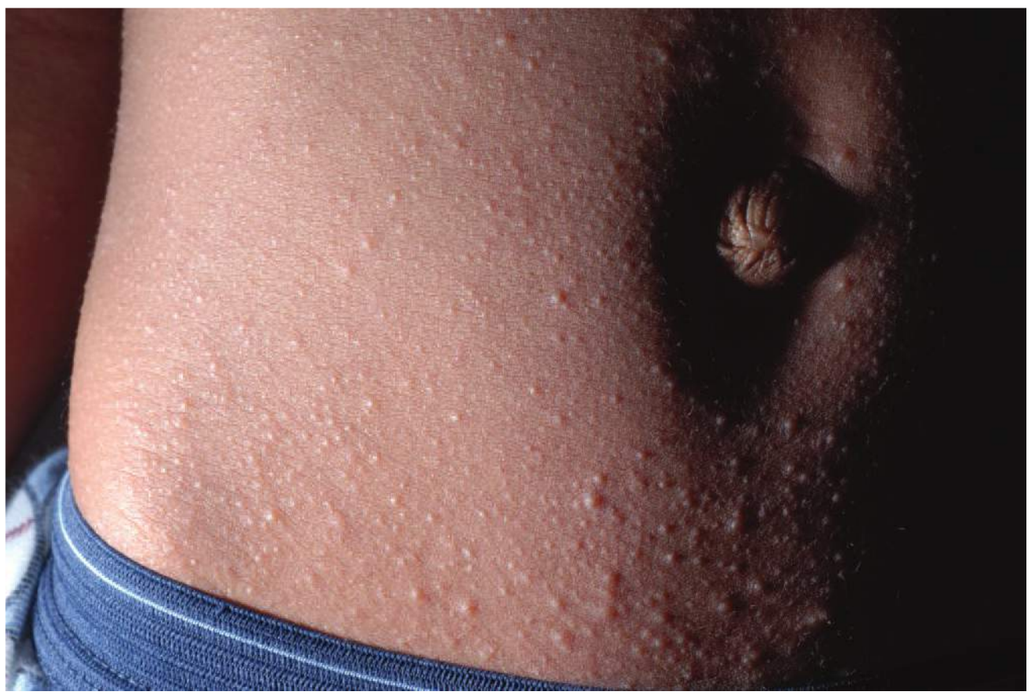


FIGURE 14.47 ■ **Goose Flesh.** This sandpaper rash started 2 days after sore throat and fever began in this 6-year-old. The grouping of the fine papules gives the skin a "goose flesh" texture in darker skin colors. (Photo contributor: Lawrence B. Stack, MD.)

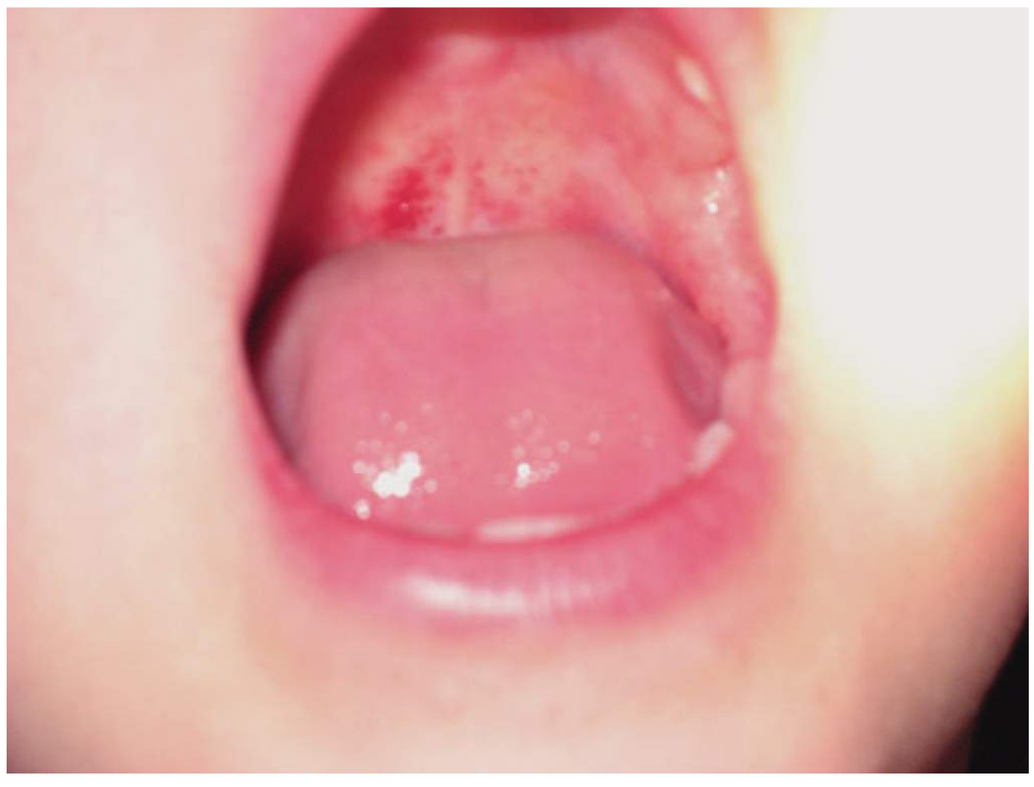


FIGURE 14.48 ■ **Palatal Petechiae.** Petechiae present on the posterior soft palate of a child with group A streptococcal infection. (Photo contributor: Hannah F. Smitherman, MD.)

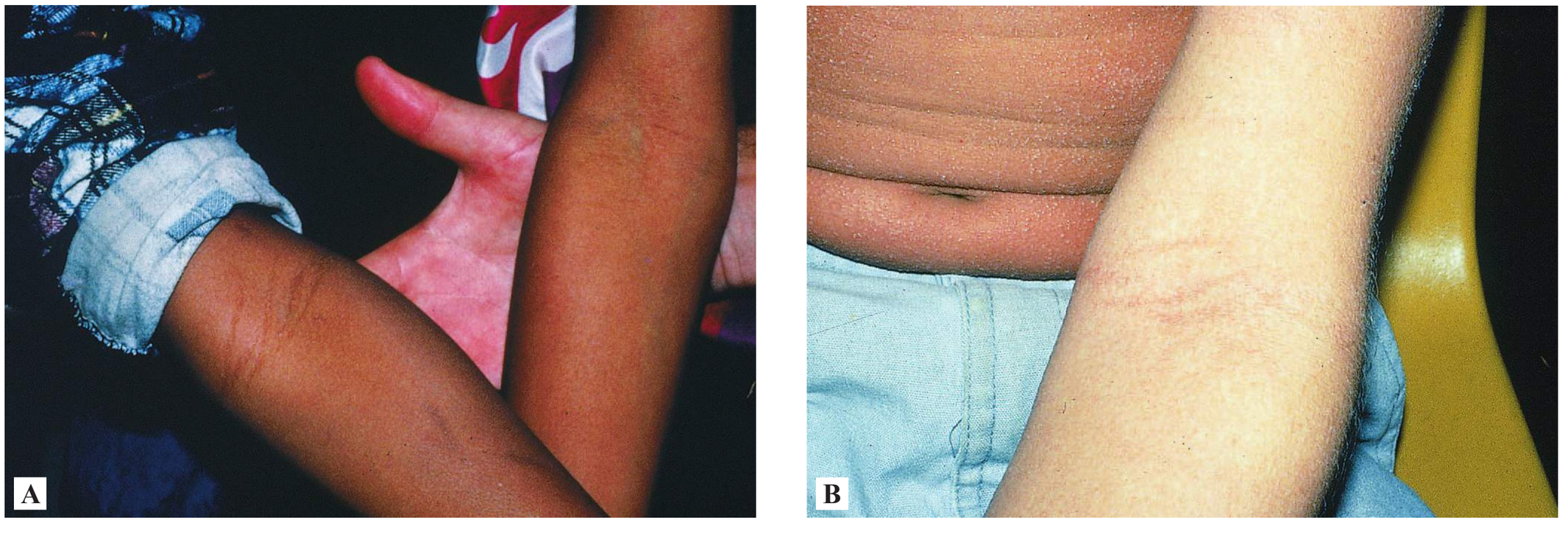


FIGURE 14.49 ■ **Pastia Lines.** Confluent petechiae in a linear pattern in the antecubital fossa consistent with Pastia lines are seen in these patients with scarlet fever. (**A**) The forearm on the right belongs to the patient's sister and does not show Pastia lines. (**B**) Pastia lines in a Caucasian patient. A classic sandpaper rash is also evident on the arm and trunk. (Photo contributor: Stephen W. Corbett, MD.)

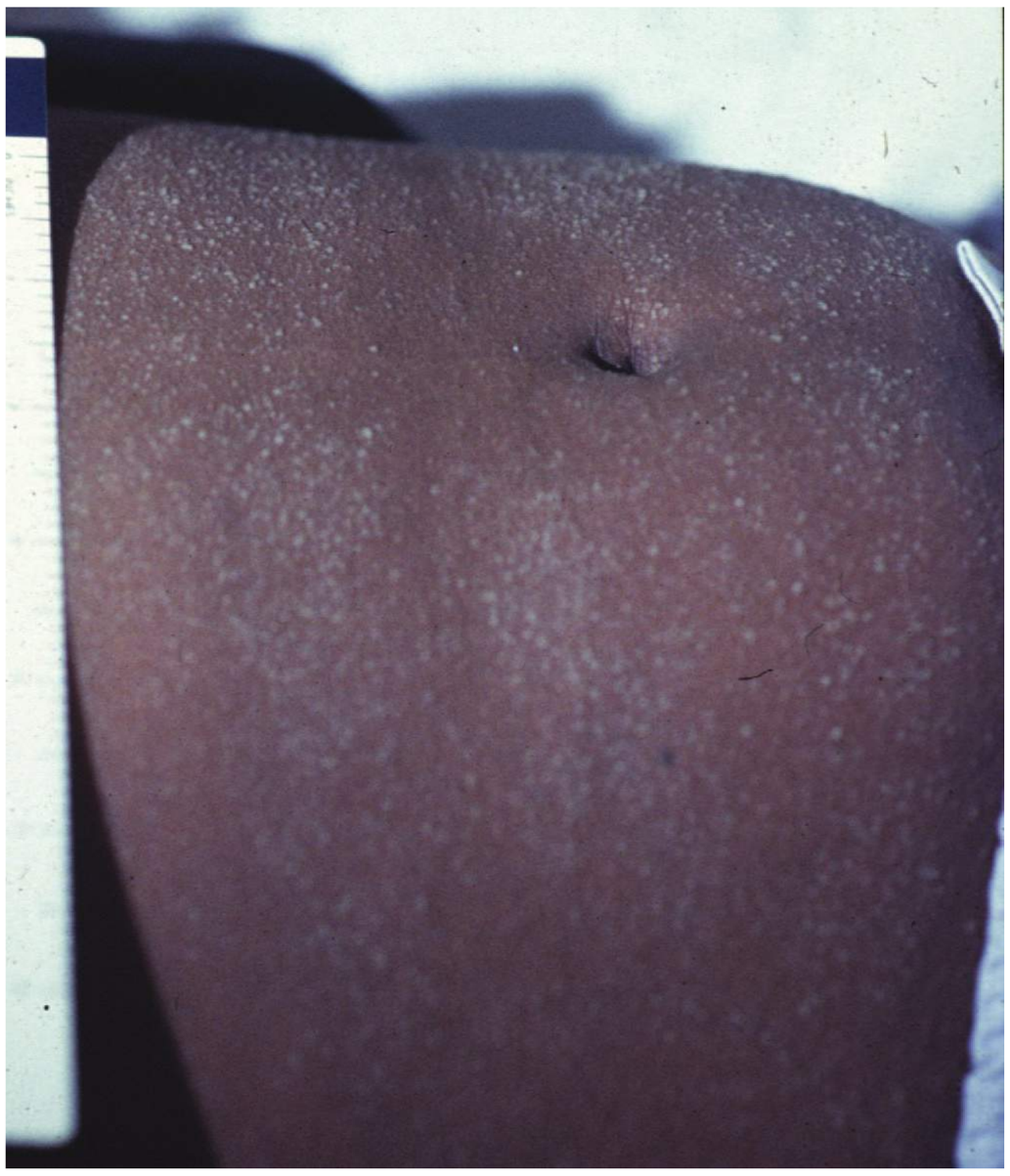


FIGURE 14.50 ■ **GABHS Toxic Shock Syndrome.** Nine-year-old girl with strawberry tongue, nausea, vomiting, fever, and sandpaper rash over trunk, arms, and legs for 4 days suggestive of toxic shock syndrome. Vital signs on presentation: HR: 152, BP: 84/55. (Photo contributor: Eric M. Sergienko, MD.)

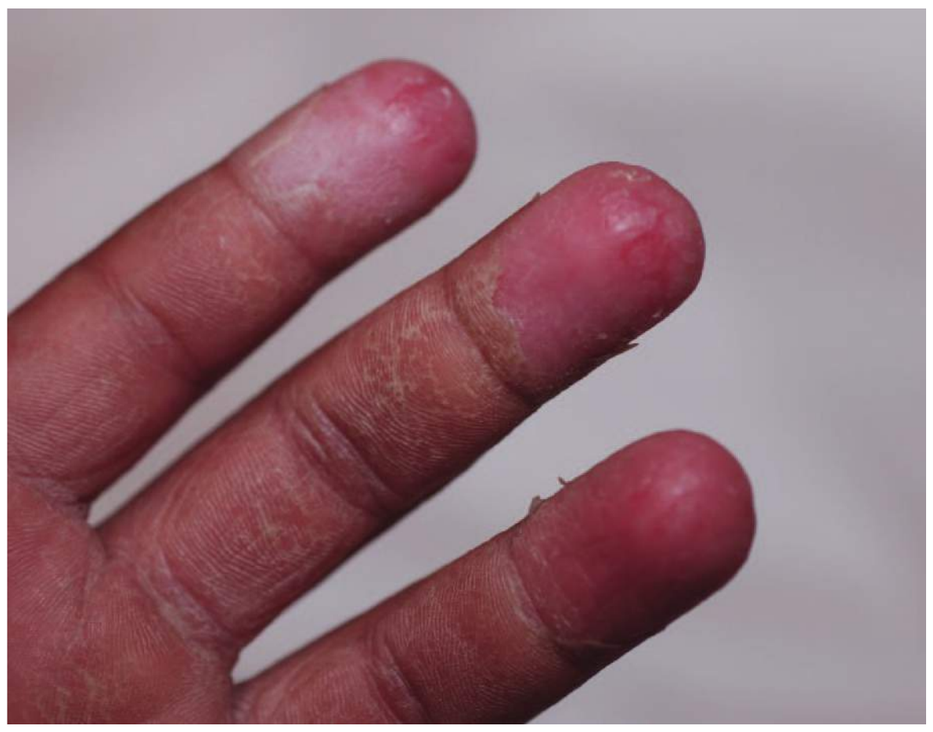


FIGURE 14.51 ■ **Desquamating Rash.** Desquamation of the fingertips occurred as the scarlatiniform rash began to fade. Desquamation also occurred in the groin and toes. (Photo contributor: Clay. B. Smith, MD.)

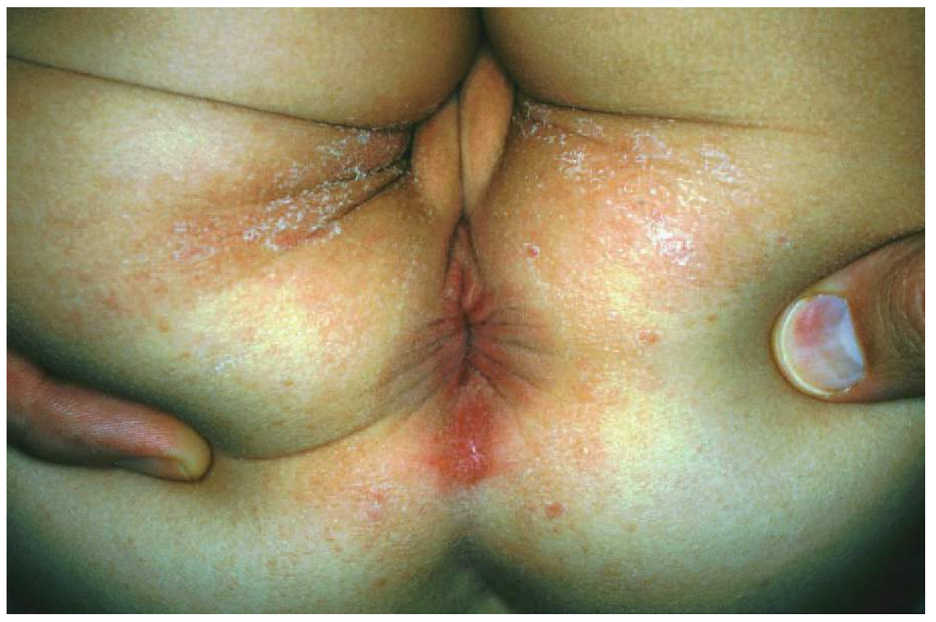


FIGURE 14.52 ■ **Perianal Strep Desquamation.** Desquamating rash in the perineum of a child with GABHS infection. (Photo contributor: Kevin J. Knoop, MD.)

BLISTERING DISTAL DACTYLITIS

Clinical Summary

Blistering distal dactylitis is a cellulitis of the fingertip caused by group A β-hemolytic Streptococcus or S aureus infection in children from infancy to teenage years. The typical lesion is a fluid-filled, painful, tense blister with surrounding erythema located over the volar fat pad on the distal portion of a finger or toe. Polymorphonuclear leukocytes and gram-positive cocci can be found in the Gram stain of the purulent exudate from the lesion. The differential diagnosis includes bullous impetigo, burns, friction blisters, felon, and herpetic whitlow.

Management and Disposition

There is usually a rapid response to incision and drainage of the blister and a 10-day course of antibiotic therapy. Consider use of agents active against MRSA (clindamycin or trimethoprim/sulfamethoxazole) if there is a high community prevalence.

Pearl

1. Nonpurulent vesicular lesions that become confluent multilocular bullae are characteristic of herpetic whitlow and help distinguish it from blistering distal dactylitis.

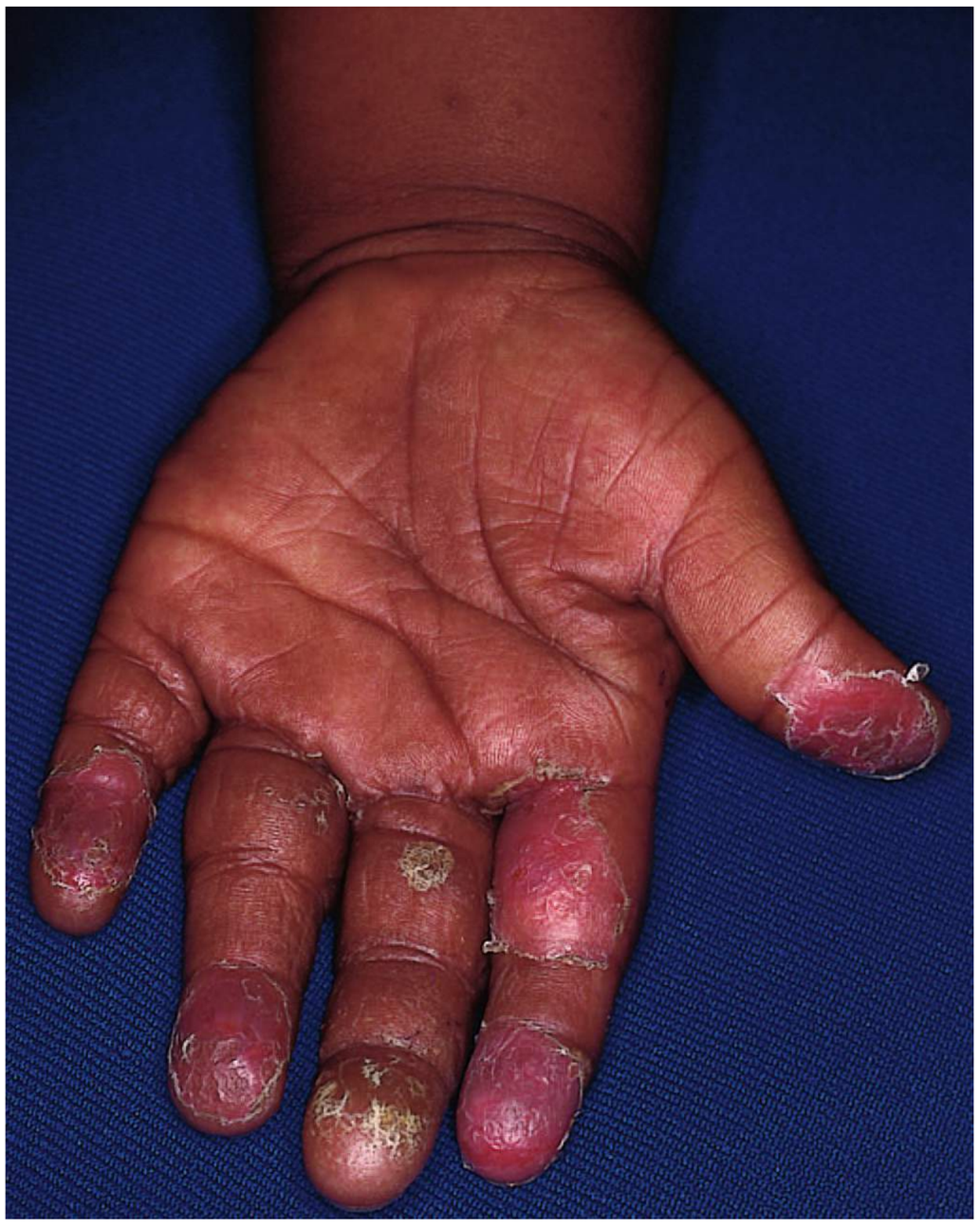


FIGURE 14.53 ■ **Blistering Distal Dactylitis.** Blistering rash of the distal fingers with surrounding erythema typically caused by Streptococcus. Note the location of the rash over the volar finger pads. (Photo contributor: Anne W. Lucky, MD.)

HENOCH-SCHÖNLEIN PURPURA

Clinical Summary

Henoch-Schönlein purpura (HSP) is the most common systemic vasculitis in children, with a peak incidence between ages 3 and 15 years. It is characterized by four main clinical manifestations. The classic **exanthem** of HSP begins with erythematous macules or urticaria that eventually coalesce and evolve into ecchymotic lesions and palpable purpura. The lesions are more often located on the buttocks and gravity dependent areas (lower extremities) in ambulatory children. In nonambulatory children, the lesions can be seen on the face, trunk, and upper extremities as well. Mucosal involvement is rare; however, edema of the scalp, hands, scrotum, and periorbital tissue occurs.

Migratory oligoarticular (1 to 4 joints) **arthritis** and **arthralgia** are eventually seen in 84% of patients. **Gastrointestinal symptoms** (colicky abdominal pain, occult or gross blood in the stool, and small bowel-small bowel intussusception) usually develop within a week of the rash, but may precede it. **Renal involvement** is the most frequent serious complication and usually occurs during the first month. It commonly manifests as microscopic hematuria and may progress to glomerulonephritis. One percent progress to end-stage renal disease. Hypertension is uncommon.

Diagnosis is made by history and clinical examination. Laboratory tests are usually normal (platelets, complement level, coagulation studies, and antinuclear antibodies) except for the urinalysis, which may be positive for blood or protein in 50%. The overall prognosis is excellent, with full recovery in most. The course is marked by relapses and remissions in 50% (the rash tends to recur within the first 6 weeks). Prognosis is linked to renal involvement. The rash may be confused with drug reactions, erythema multiforme, urticaria, and even physical abuse. Consider other causes of purpura, such as bleeding disorders or infection (meningococcemia).

Management and Disposition

Treatment is supportive. Patients with HSP limited to the skin and joints with minimal urinary findings can be managed as outpatients. Hospitalization is warranted when patients cannot maintain oral intake, have severe abdominal pain, GI hemorrhage, and intussusception, are unable to ambulate or care for themselves because of significant joint pain, or have evidence of renal injury. Ibuprofen or naproxen can be effective for symptomatic pain relief. Corticosteroids may shorten the duration of abdominal pain in patients with severe symptoms that are unable to maintain oral intake. They are not recommended routinely for rash or joint pains. In cases where intussusception is suspected, prompt surgical consultation and diagnostic ultrasonography or air-contrast enema are indicated. In contrast to ileocolic intussusception, small bowel-small bowel intussusception generally resolves spontaneously and rarely requires surgical intervention.

Pearls

1. Obtain a stool occult blood test and urinalysis in patients with abdominal pain and suspected HSP. The fecal occult blood test is positive in more than half.
2. Intussusception associated with HSP is seen in 2% of patients, most commonly in boys, particularly those about 6 years of age. The intussusception is often ileo-ileal.
3. The typical rash occurs in nearly 100% and is the presenting feature in at least 50%. Joint symptoms may precede the rash as the presenting complaint in 25% of patients. Ankles and knees are the most commonly affected joints.
4. Scrotal swelling and pain may be the initial presenting sign in boys and can mimic testicular torsion.

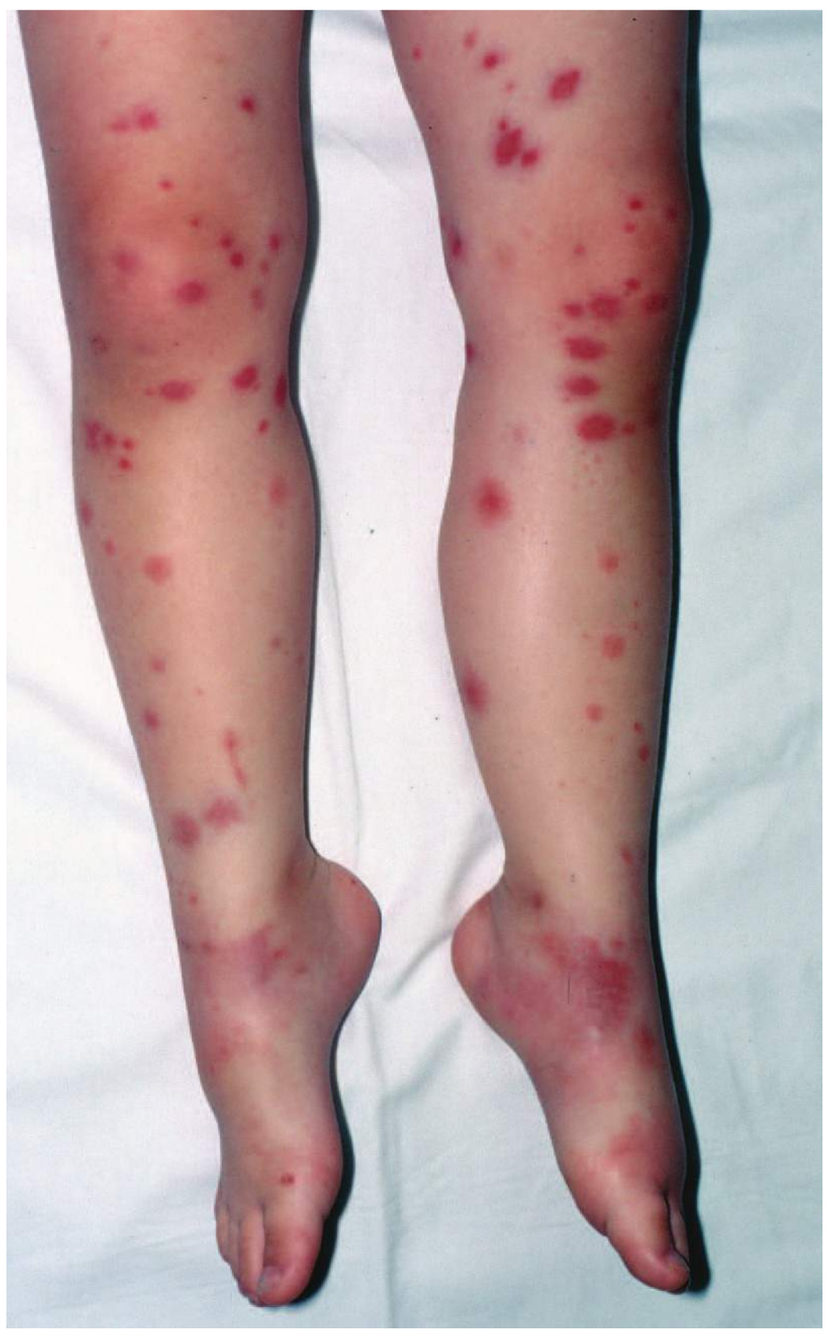


FIGURE 14.54 ■ **Henoch-Schönlein Purpura.** These erythematous, hemorrhagic papules and petechiae in a symmetric acral distribution are classic findings in HSP. This child presented with no other symptoms. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

It is characterized by rash, fever, and polyarthralgias with onset 1 to 2 weeks following exposure to an offending agent, and resolves within 1 to 2 weeks after exposure is discontinued. It is felt to occur via an immune complex–mediated mechanism and can be precipitated by exposure to a number of drugs, with cefaclor, trimethoprim-sulfamethoxazole, and penicillins being the most common. Almost all patients develop a polymorphous pruritic rash that starts in the trunk, groin, and axillae, eventually spreading to the limbs. The urticarial lesions are longer lasting than typical hives. Some patients can exhibit palpable purpura, maculopapular lesions, or erythema multiforme–type exanthema. In all patients, the mucous membranes are spared. Almost all patients develop remittent fever without temporal spikes. Two-thirds will have arthralgias, most often in the peripheral extremities. Frank arthritis is more rare, but may lead to a child being unable/unwilling to walk. The differential diagnosis includes viral exanthems, hypersensitivity vasculitis, scarlet fever, acute rheumatic fever, meningococcemia, disseminated gonococcemia, reactive arthritis, Lyme disease, Still disease, and Stevens-Johnson syndrome.

Management and Disposition

The diagnosis is clinical and based on history of exposure to a potential offending agent. Treatment consists of discontinuing the causative agent and supportive care. Glucocorticoids may benefit patients with severe symptoms. Diphenhydramine can be used in cases where pruritus is a prominent symptom. Patients who are ill-appearing or do not have a history of a potential offending agent should have a CBC with differential, erythrocyte sedimentation rate (ESR) and C-reactive protein (CRP), urinalysis, blood urea nitrogen (BUN), creatinine, serum electrolytes, urinalysis, and blood culture obtained to exclude other inflammatory or infectious causes.

Pearls

1. Serum sickness, as originally described, is typically in response to administration of a nonhuman species protein antigen. Present-day examples include sheep-derived Fab snake antivenom, heterologous immunomodulators containing murine components (rituximab and infliximab), streptokinase, and the human diploid cell rabies vaccine.
2. Patients with a history of serum sickness–like reaction will have a more rapid onset of symptoms if reexposed to the causative agent.
3. Mucous membrane and eye involvement does not occur in serum sickness–like reactions. These findings should prompt consideration for Stevens-Johnson syndrome, a potentially fatal hypersensitivity reaction.

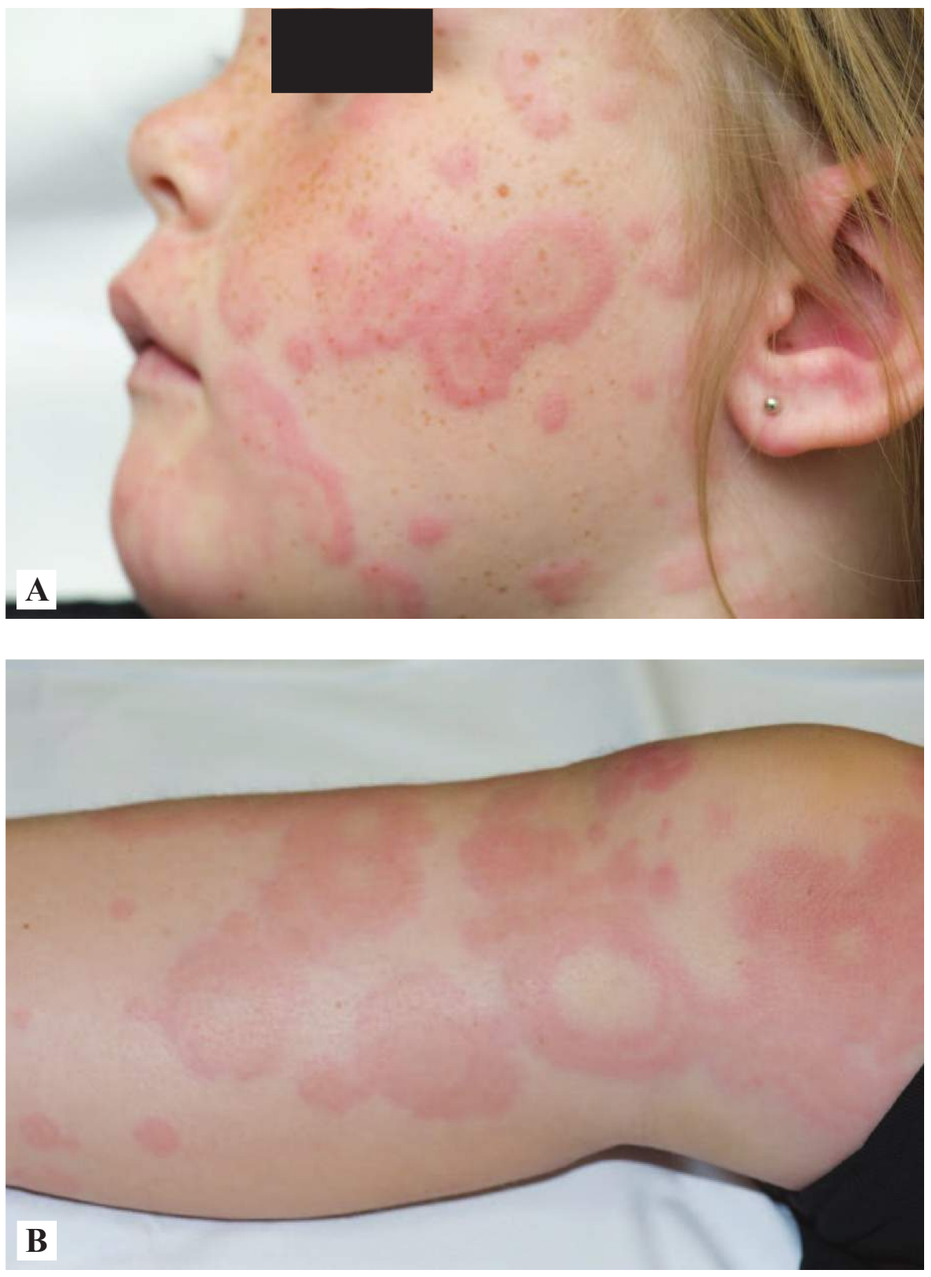


FIGURE 14.55 ■ **Serum Sickness–Like Reaction.** Fever, joint pain, and erythematous plaques to face (**A**) and legs (**B**) 14 days after receiving amoxicillin in a 7-year-old girl. These clinical findings suggest serum sickness. (Photo contributor: Lawrence B. Stack, MD.)

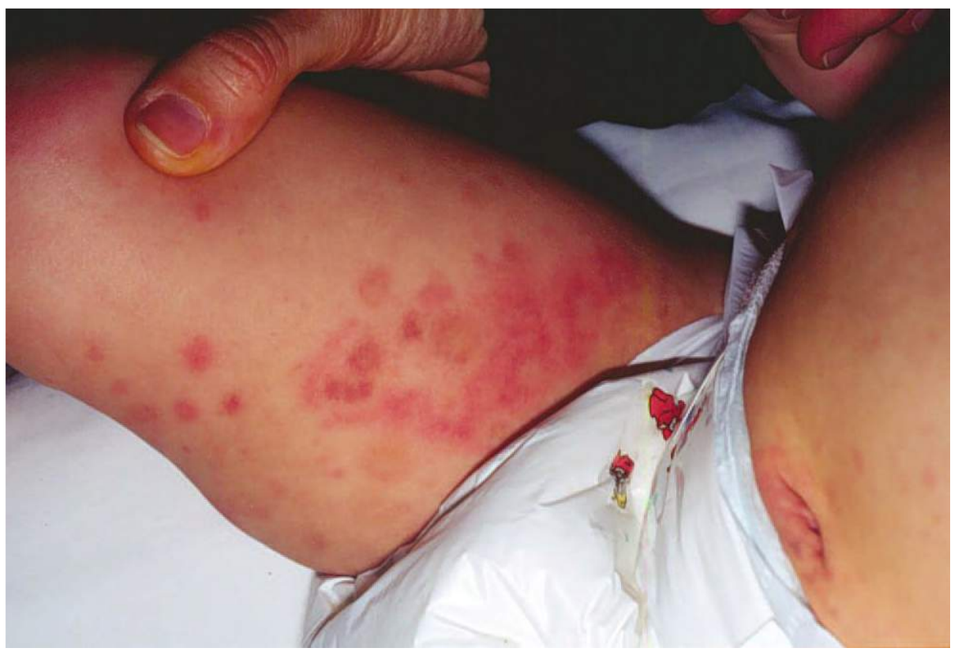


FIGURE 14.56 ■ **Serum Sickness–Like Reaction.** Fever, joint pain, ankle swelling, and erythematous plaques seen here in an infant 11 days after taking ceclor, suggesting serum sickness. (Photo contributor: Lawrence B. Stack, MD.)

STRAWBERRY HEMANGIOMA

Clinical Summary

Hemangiomas are benign vascular tumors characterized by a rapid proliferative phase followed by a spontaneous involutional phase. They are the most common soft tissue tumors of infancy. The appearance of hemangiomas is defined by the lesion's depth, location, and stage of evolution. A strawberry hemangioma lies in the upper dermis and often originates as an erythematous macular patch, a pale macule, or a localized telangiectasia with a pale halo. The lesion grows and becomes vascularized during the first 2 months of life. The classic presentation is a bright red, slightly raised, noncompressible plaque. It commonly regresses by 2 to 3 years of age but may persist throughout life. Hemangiomas can also affect the airway, eyes, and liver or cause high-output cardiac failure if sufficiently large. The most important local complication is ulceration which can be exquisitely painful. The differential diagnosis includes vascular malformations, malignant vascular neoplasms, pyogenic granulomas, and giant melanocytic birthmarks.

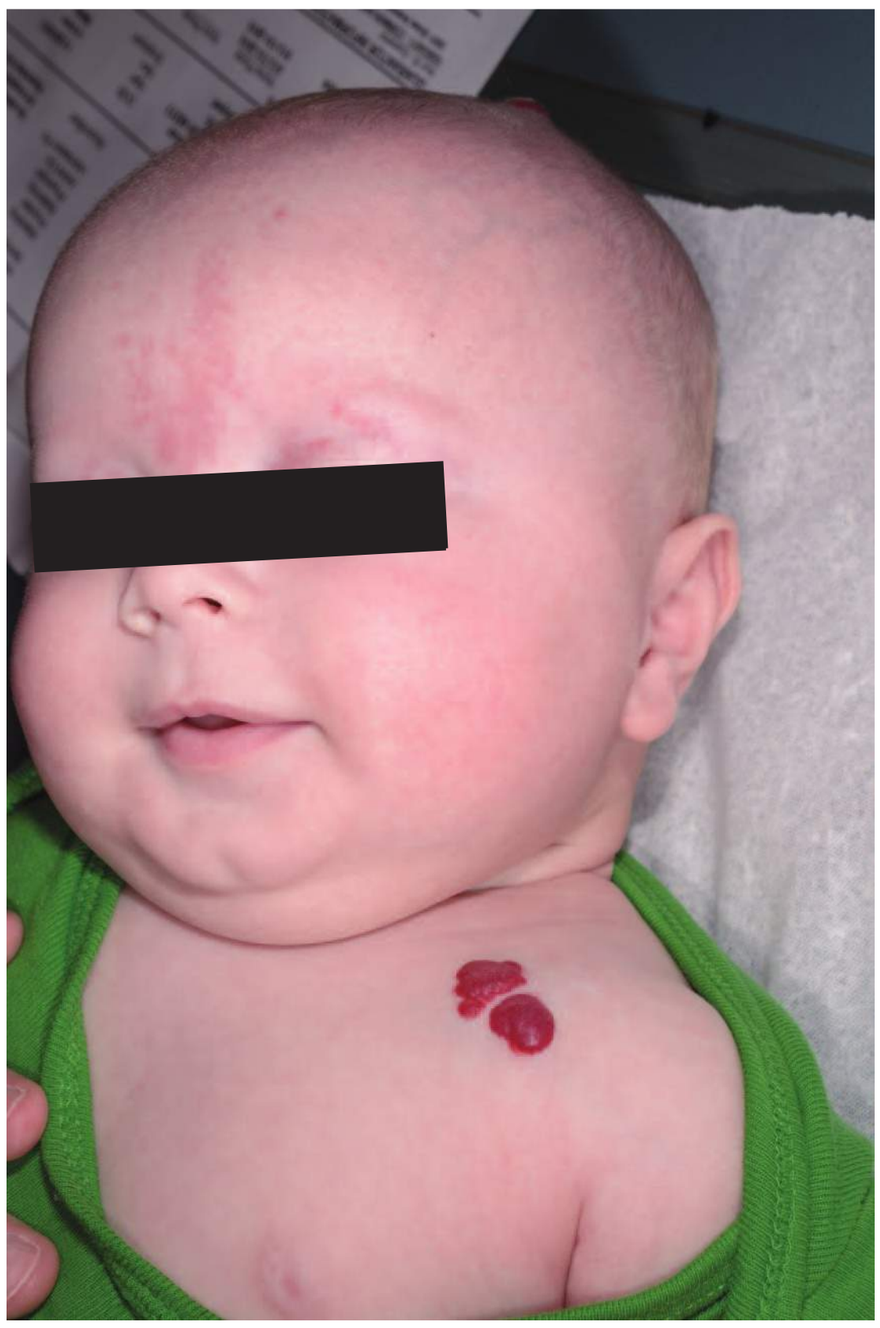


FIGURE 14.57 ■ **Strawberry Hemangioma.** Raised umbilicated vascular lesion on the left upper chest consistent with strawberry hemangioma. (Photo contributors: Kara Shah, MD, PhD, and Katharine Hanlon.)

Management and Disposition

Most cases require no therapy because strawberry hemangiomas usually regress without residual problems. Treatment of hemangiomas in general is indicated when there is an obstruction of a vital orifice (ie, airway, mouth, or nares) or vision (eyelids) or if hematologic or cardiovascular complications are present. Education and parental reassurance are important because there is great pressure to treat for cosmetic reasons. Oral corticosteroids, intralesional corticosteroids, pulsed dye laser, and recombinant interferon-α are some of the therapeutic modalities available. In complex cases, consultation with a vascular malformation specialist or dermatologist is recommended.

Pearls

1. Kasabach-Merritt phenomena (hemolytic anemia, thrombocytopenia, and coagulopathy) is associated with Kaposiform hemangioendothelioma or tufted angiomas and not with common hemangiomas as was previously thought.
2. As many as 20% of affected infants have multiple lesions.
3. In contrast to hemangiomas, vascular malformations, such as port-wine stains, do not proliferate or involute. They persist throughout life and grow in proportion with the child.
4. Patients with hemangiomas of the face, and especially in the "beard" distribution, have a higher risk of also having an airway hemangioma. Hemangiomas over the midline lumbar spine should prompt an evaluation for spinal dysraphism.

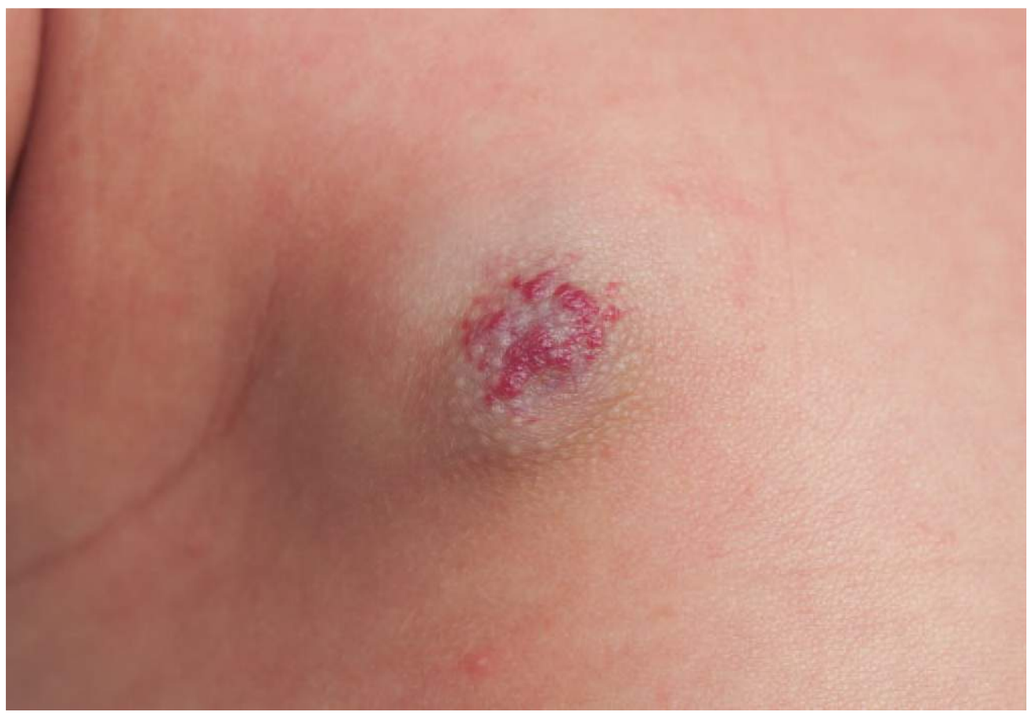


FIGURE 14.58 ■ **Strawberry Hemangioma.** Vascular lesion on the nipple. (Photo contributor: Lawrence B. Stack, MD.)

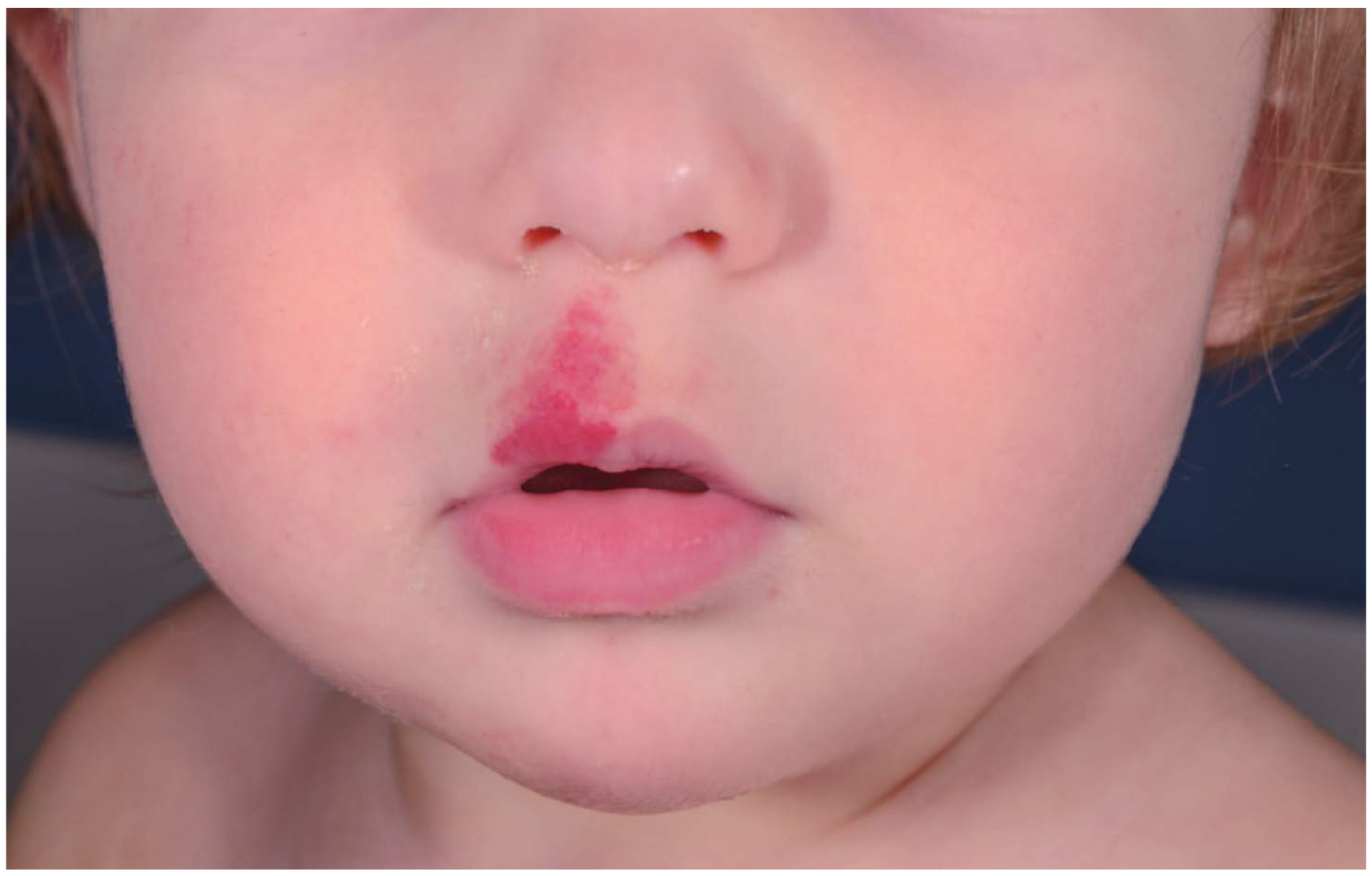


FIGURE 14.59 ■ **Strawberry Hemangioma.** Vascular lesion on the upper lip consistent with strawberry hemangioma. (Photo contributors: Kara Shah, MD, PhD, and Katharine Hanlon.)

ORBITAL AND PRESEPTAL (PERIORBITAL) CELLULITIS

Clinical Summary

Orbital cellulitis is a serious bacterial infection characterized by fever, painful purple-red eyelid swelling, ophthalmoplegia, pain with extraocular movements, proptosis, and variable decreased visual acuity. It may begin with eye pain and fever. In general, it is caused by Streptococcus pneumoniae, Haemophilus influenzae, Moraxella catarrhalis, and S aureus. It usually arises as a complication of ethmoid or maxillary sinusitis. If not treated promptly, it can lead to blindness, cavernous venous sinus thrombosis, meningitis, subdural empyema, or brain abscess.

Preseptal (periorbital) cellulitis is much more common and usually presents with edema and circumferential erythema of the eyelids and periorbital skin, fever, and minimal pain. Proptosis and ophthalmoplegia are not characteristic as it does not involve the orbit or other ocular structures. Preseptal cellulitis usually results from sinusitis or contiguous infection due to local skin trauma, insect bite, or hordeolum. Common organisms are S aureus and group A Streptococcus.

Management and Disposition

A broad-spectrum parenteral antibiotic with staphylococcal coverage, ophthalmologic consultation, and admission are indicated in cases of orbital cellulitis. Orbital CT with contrast is needed to assess for abscess requiring surgical drainage. In febrile or ill-appearing patients with preseptal cellulitis, admission with broad-spectrum IV antibiotic therapy is also indicated. Adults and children greater than 1 year with mild cases (especially those with a history of trauma such as abrasion or insect bite or sting) can be treated with outpatient oral antibiotic therapy with close follow-up. Consider the use of antimicrobials with MRSA coverage.

Pearls

1. Obtain orbital and sinus CT if the diagnosis is uncertain and in patients who have pain with eye movement, vision changes, or if unable to assess vision (usually age less than 1 year).

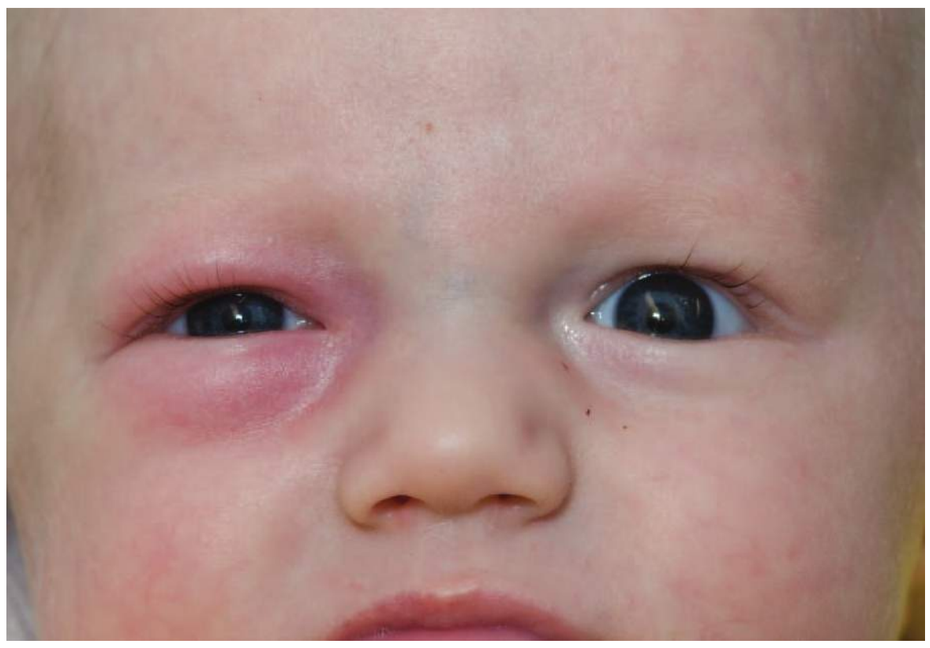


FIGURE 14.60 ■ **Orbital Cellulitis.** CT confirmed orbital cellulitis in this 2-month-old. (Photo contributor: Lawrence B. Stack, MD.)

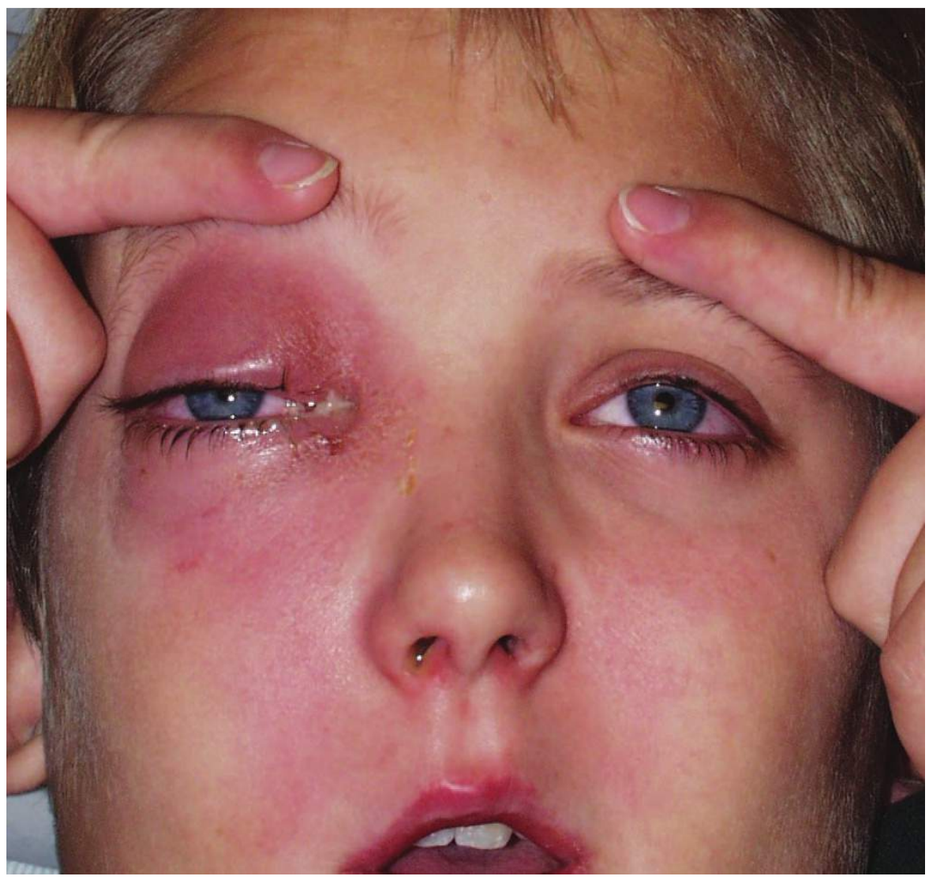


FIGURE 14.61 ■ **Orbital Cellulitis.** Eye redness, swelling, purulent drainage, and mild entrapment are seen in this patient with painful orbital cellulitis. (Photo contributor: Kevin J. Knoop, MD, MS.)

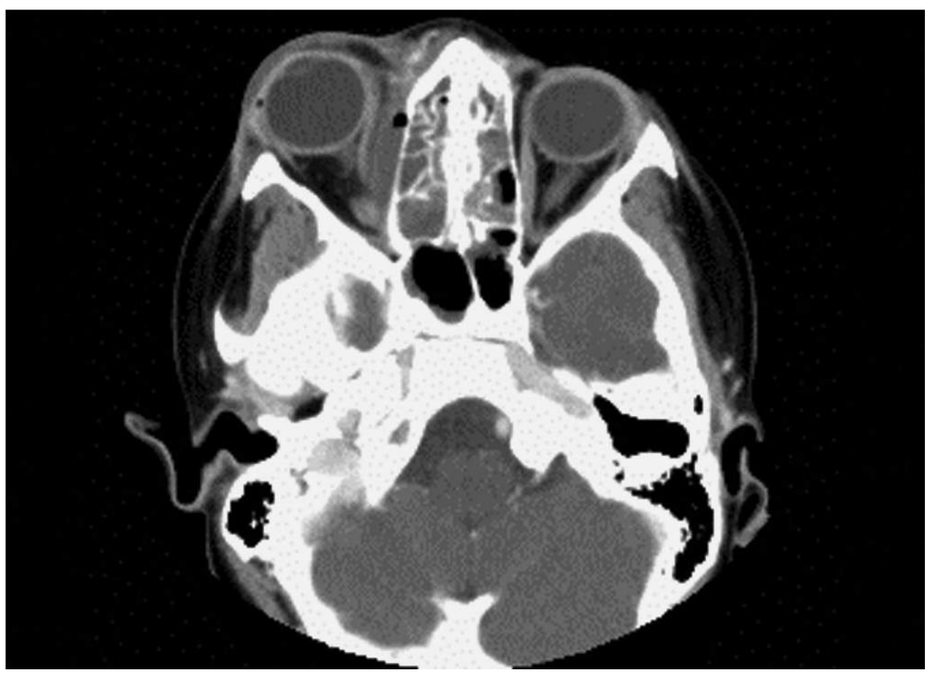


FIGURE 14.62 ■ **Orbital Cellulitis.** Right ethmoid sinusitis with subperiosteal abscess and extension into the orbital space in above patient. (Photo contributor: Kevin J. Knoop, MD, MS.)

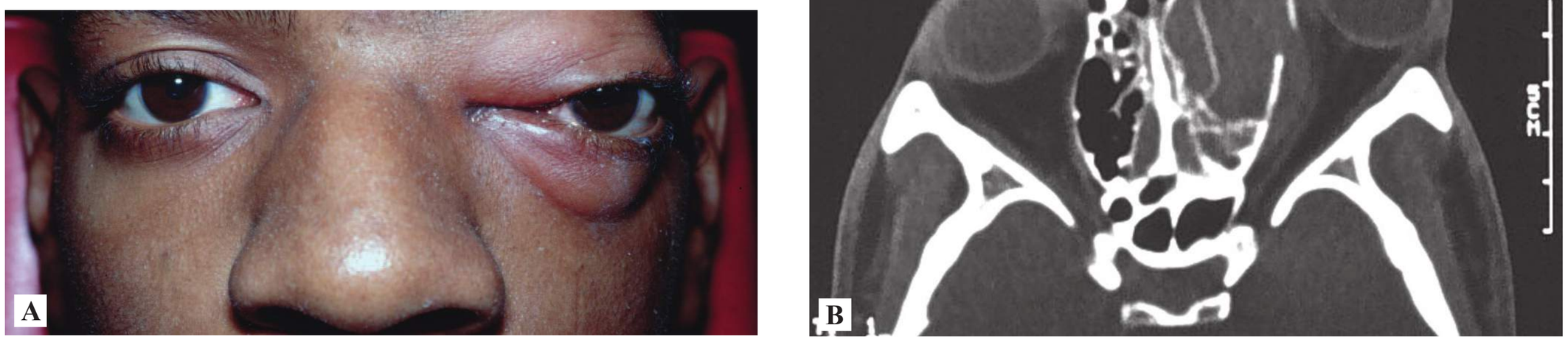


FIGURE 14.63 ■ **Orbital Cellulitis.** Left ethmoid sinusitis with extension into the orbital space, periosteal abscess formation, and proptosis is seen in this patient (**A**) and on CT (**B**). (Photo contributor: Lawrence B. Stack, MD.)

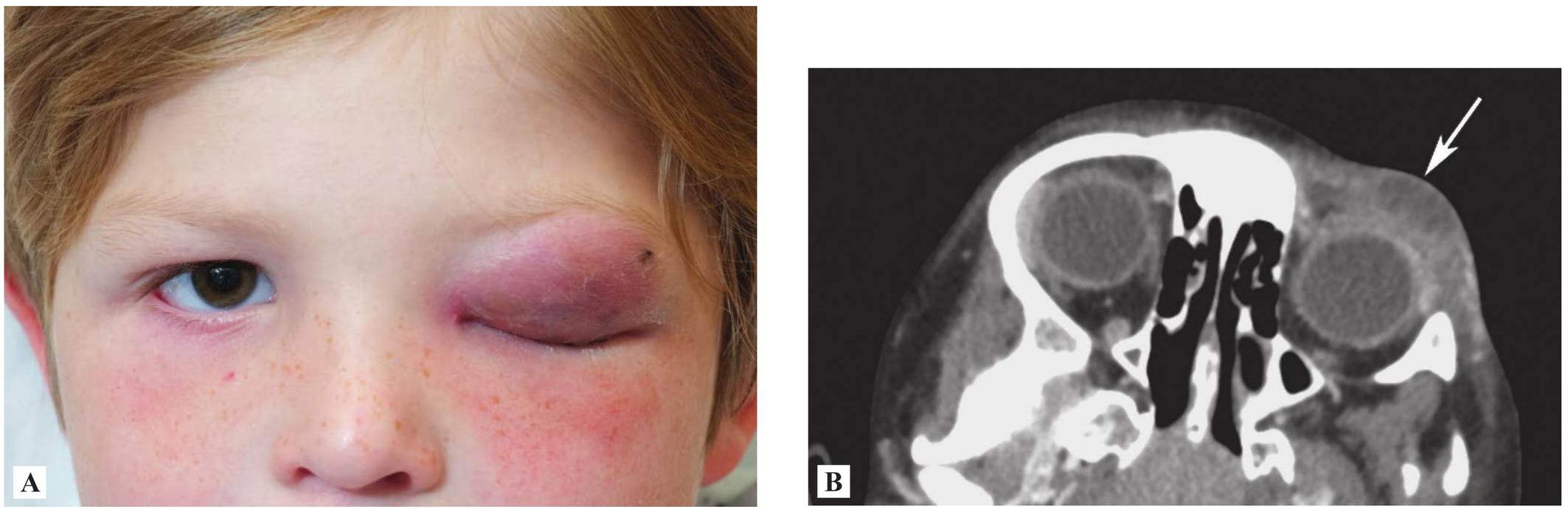


FIGURE 14.64 ■ **Preseptal Cellulitis.** Periorbital cellulitis with abscess formation seen in this patient (**A**) and on CT (**B**). (Photo contributor: Eftitan Akam.)

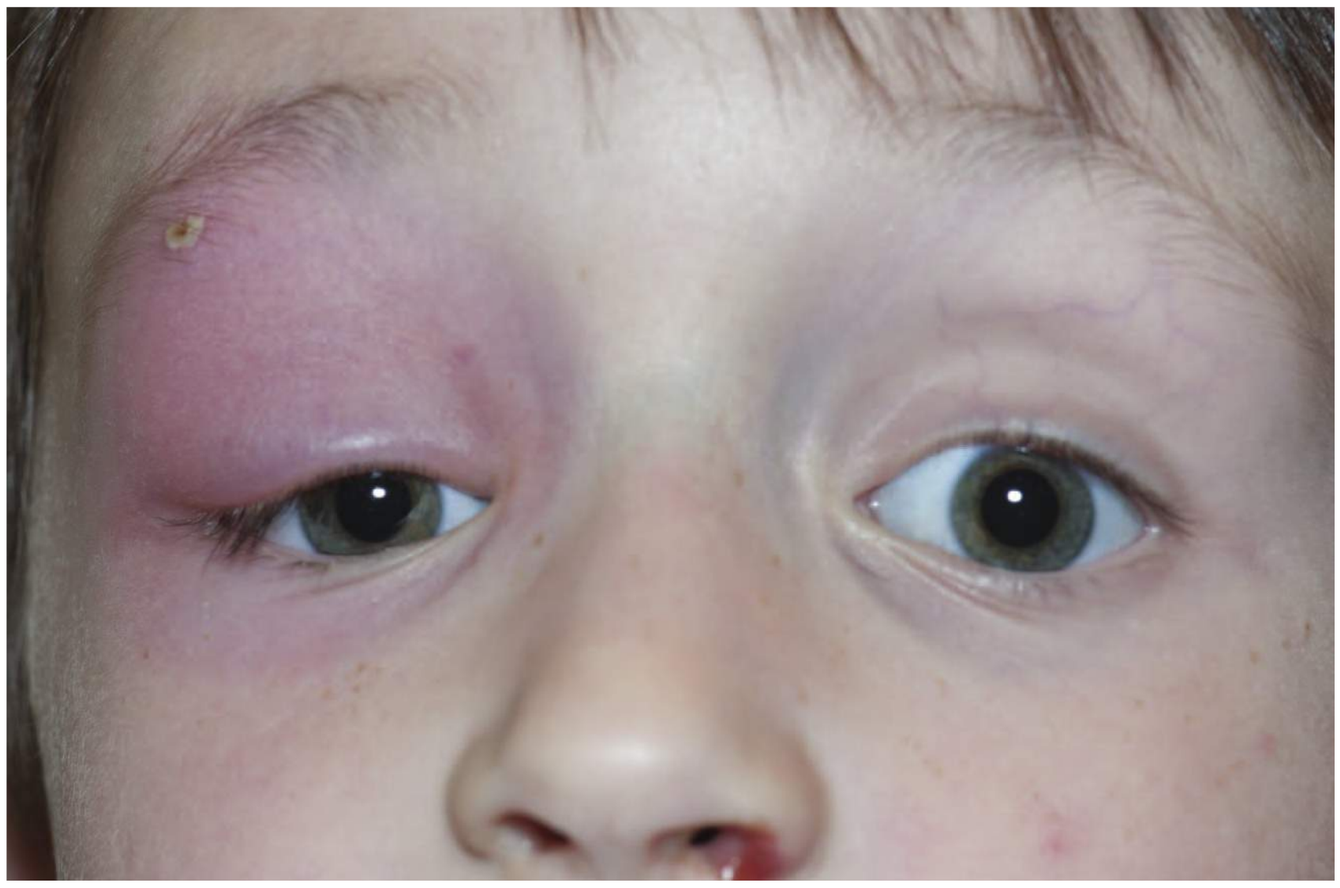


FIGURE 14.65 ■ **Preseptal Cellulitis.** Cellulitis originating from local skin trauma in the left eyebrow. (Photo contributor: Lawrence B. Stack, MD.)

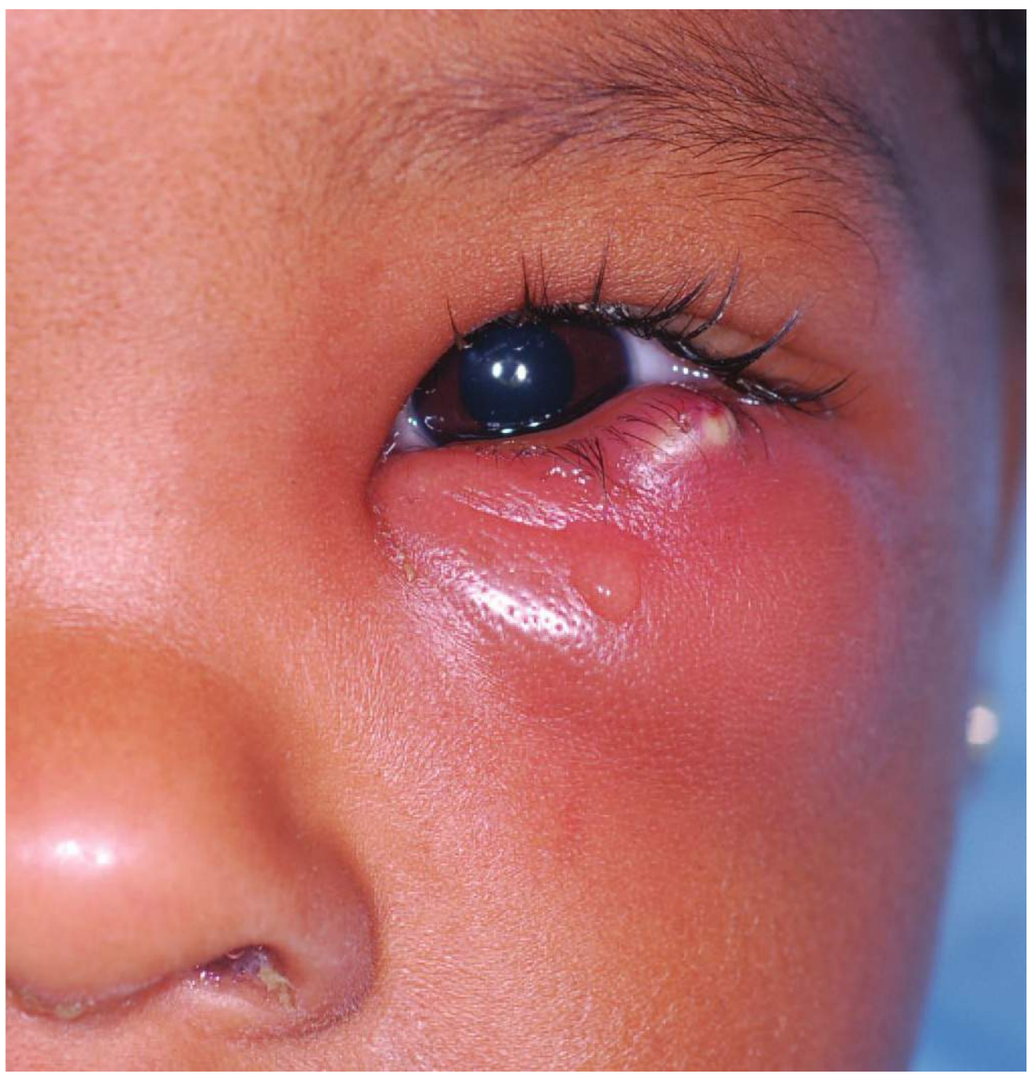


FIGURE 14.66 ■ **Preseptal Cellulitis.** Preseptal cellulitis extending from a hordeolum. (Photo contributor: David Effron, MD.)

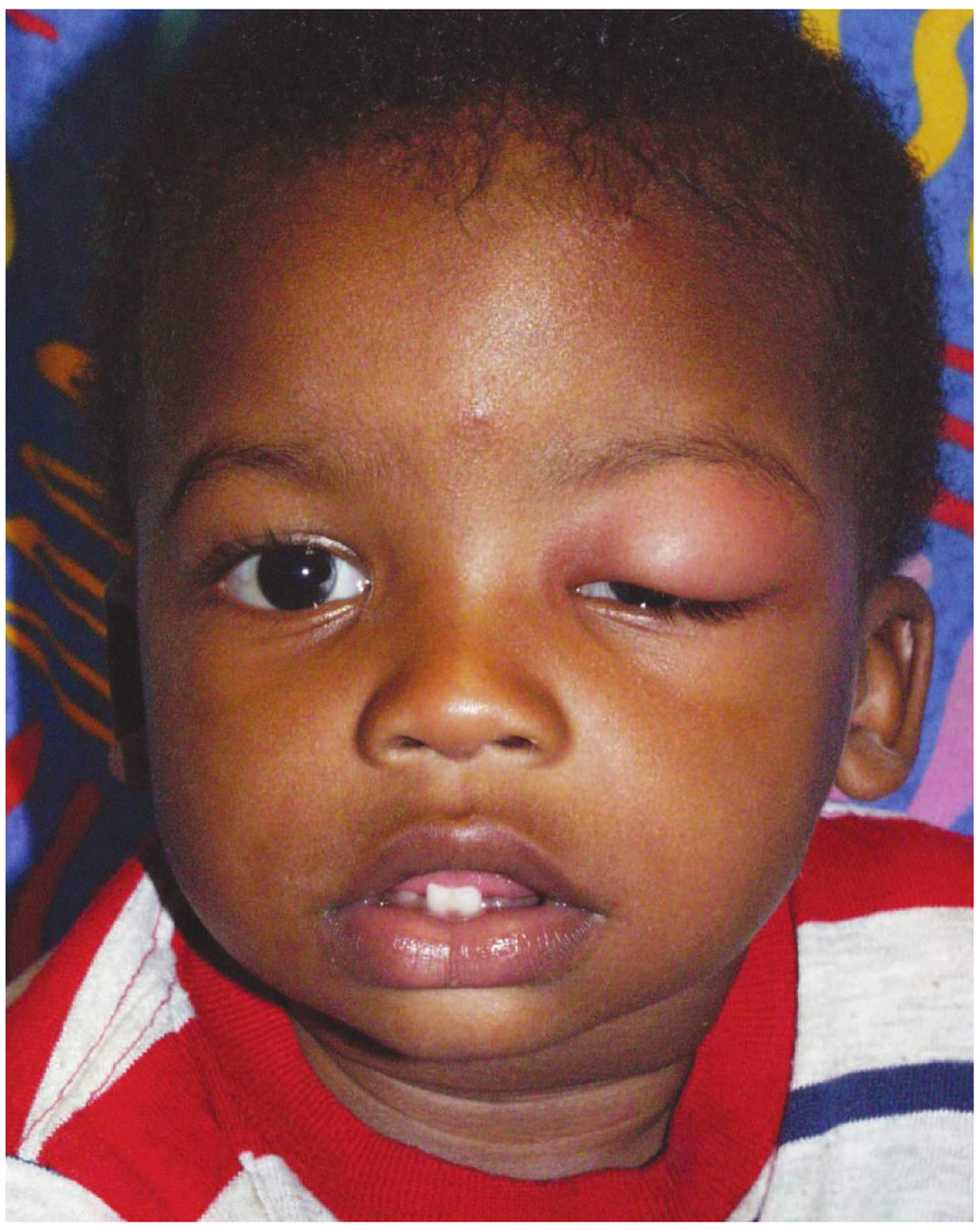


FIGURE 14.67 ■ **Preseptal Cellulitis.** Left periorbital cellulitis with edema and erythema of the eyelids in a nontoxic cooperative toddler with a normal ocular exam. (Photo contributor: Kevin J. Knoop, MD, MS.)

Clinical Summary

A branchial cleft cyst arises from the incomplete obliteration of one of the four branchial clefts during embryogenesis. As the obliteration of the clefts occurs, a portion may remain, forming a cystic space. The anatomic location of a branchial cleft cyst depends upon the specific arch/cleft involved. Involvement of the first branchial cleft may result in a cyst in the region of the parotid gland, the preauricular or postauricular area, or inferior to the angle of the mandible. A second cleft anomaly represents 95% of these malformations, and may be found along the anterior border of the sternocleidomastoid muscle or deeper, in the vicinity of the carotid arteries. Third and fourth arch/cleft anomalies are very rare. Cysts usually present clinically when they become infected and enlarge acutely usually in association with an upper respiratory infection.

Management and Disposition

The diagnosis is suggested by history and physical examination. Treatment includes antibiotics to decrease the inflammation if there is an associated infection. Ensure that there is no evidence of airway compromise. Complete excision by a specialist is necessary in order to prevent recurrence. CT or ultrasound may better define the extent of the lesion.

Pearls

1. Branchial cleft anomalies are second only to thyroglossal duct cysts in frequency of congenital head and neck lesions in children.
2. These lesions may be intimately involved with major vessels and nerves. Excision by a specialist familiar with the underlying anatomy is recommended.

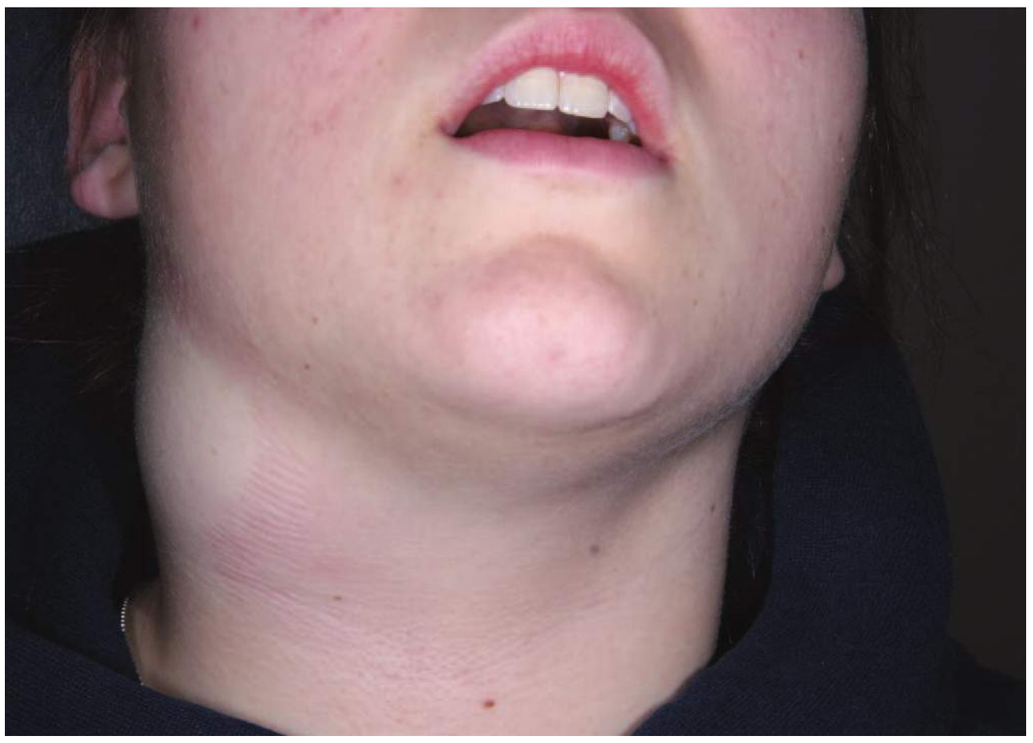


FIGURE 14.69 ■ **Second Branchial Cleft Cyst.** Right neck mass seen in second branchial cleft cyst. (Photo contributor: Scott Manning, MD.)

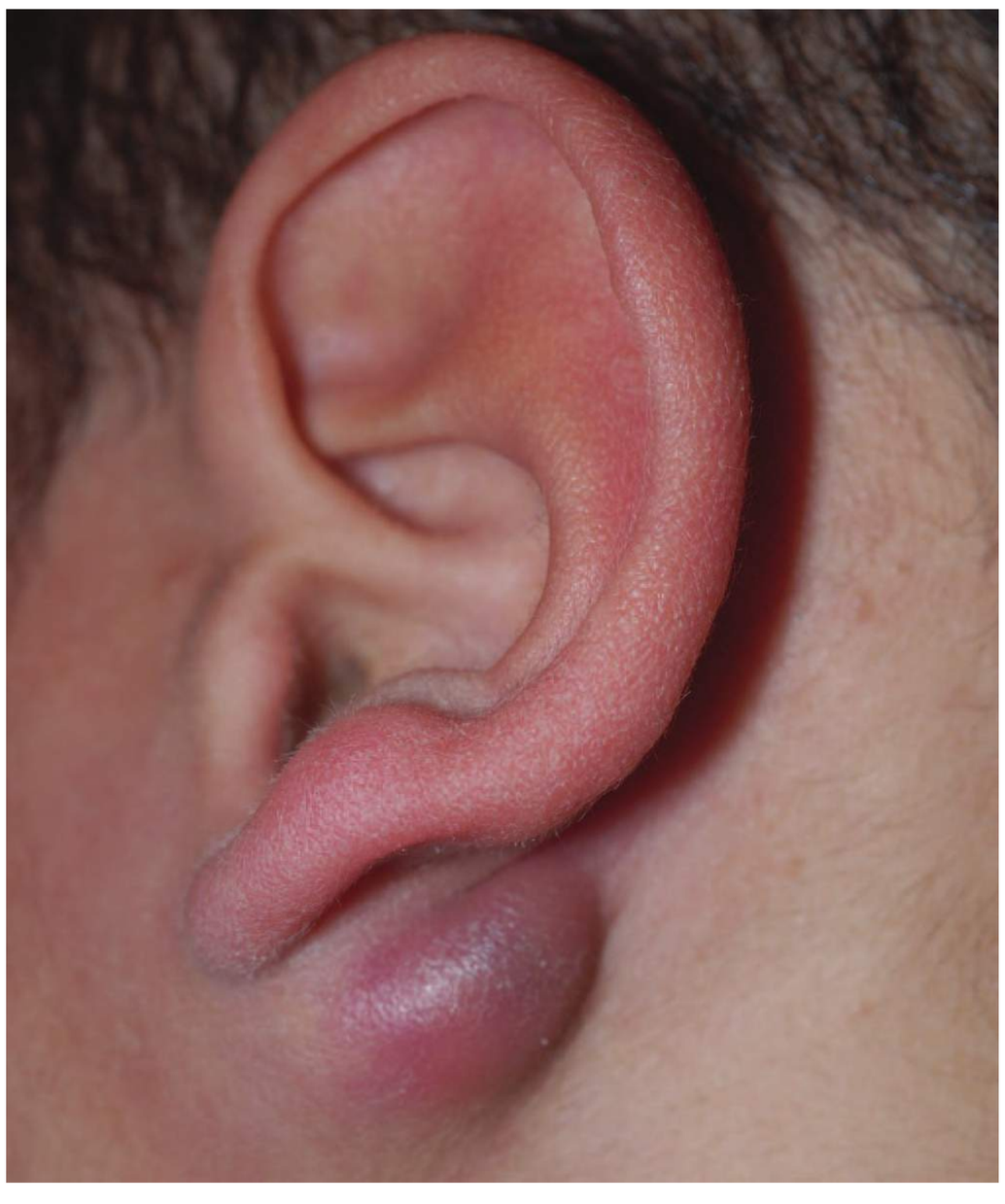


FIGURE 14.68 ■ **First Branchial Cleft Cyst.** Periauricular mass from a first brachial cleft cyst. (Photo contributor: Lawrence B. Stack, MD.)

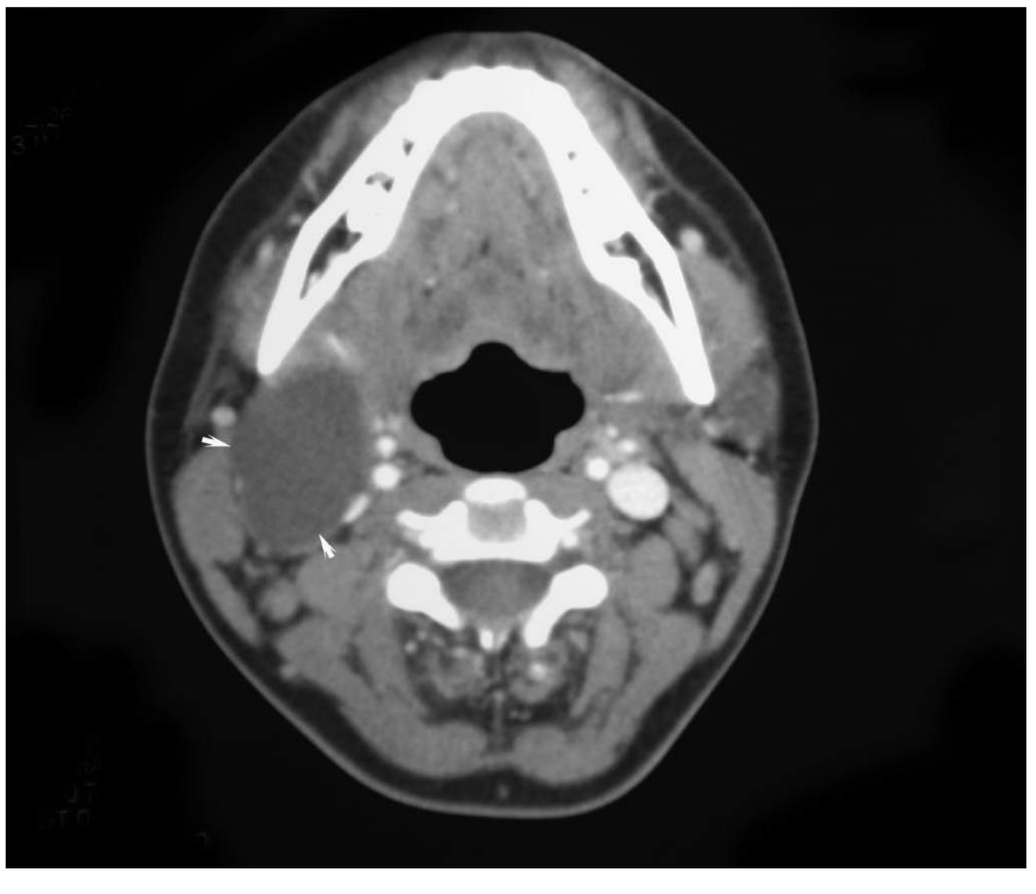


FIGURE 14.70 ■ **Second Branchial Cleft Cyst.** Neck CT showing second branchial cleft cyst. (Photo contributor: Scott Manning, MD.)

THYROGLOSSAL DUCT CYST

Clinical Summary

A thyroglossal duct cyst arises from the incomplete obliteration of the thyroglossal duct during fetal development. It usually presents as a painless, midline, anterior neck mass that moves with swallowing and protrusion of the tongue. It may occur anywhere from the base of the tongue to the sternal notch but is usually located at or below the hyoid bone adjacent to the thyrohyoid membrane. These cysts may rapidly enlarge if infected, which often occurs in association with upper respiratory symptoms.

Management and Disposition

The diagnosis of this lesion is suggested by history and physical examination. Antibiotics are indicated if the lesion has rapidly enlarged due to infection. Common pathogens include H influenzae, S aureus, and Staphylococcus epidermidis. Treatment involves excision of the cyst and midportion of the hyoid bone if involved (Sistrunk procedure) following resolution of any associated infection. Referral to an otolaryngologist is appropriate.

Pearls

1. A thyroglossal duct cyst is the most frequent congenital head and neck lesion in children.
2. In approximately 1% of patients with this lesion, the only functional thyroid tissue is located within the cyst. Therefore, patients should be screened by history for symptoms of hypothyroidism. If symptoms are present, a serum thyroid-stimulating hormone (TSH) should be sent, and ultrasound of the midline neck should be considered prior to surgical removal.
3. The cyst typically moves up with protrusion of the tongue or with swallowing due to its relationship with the hyoid bone and larynx.

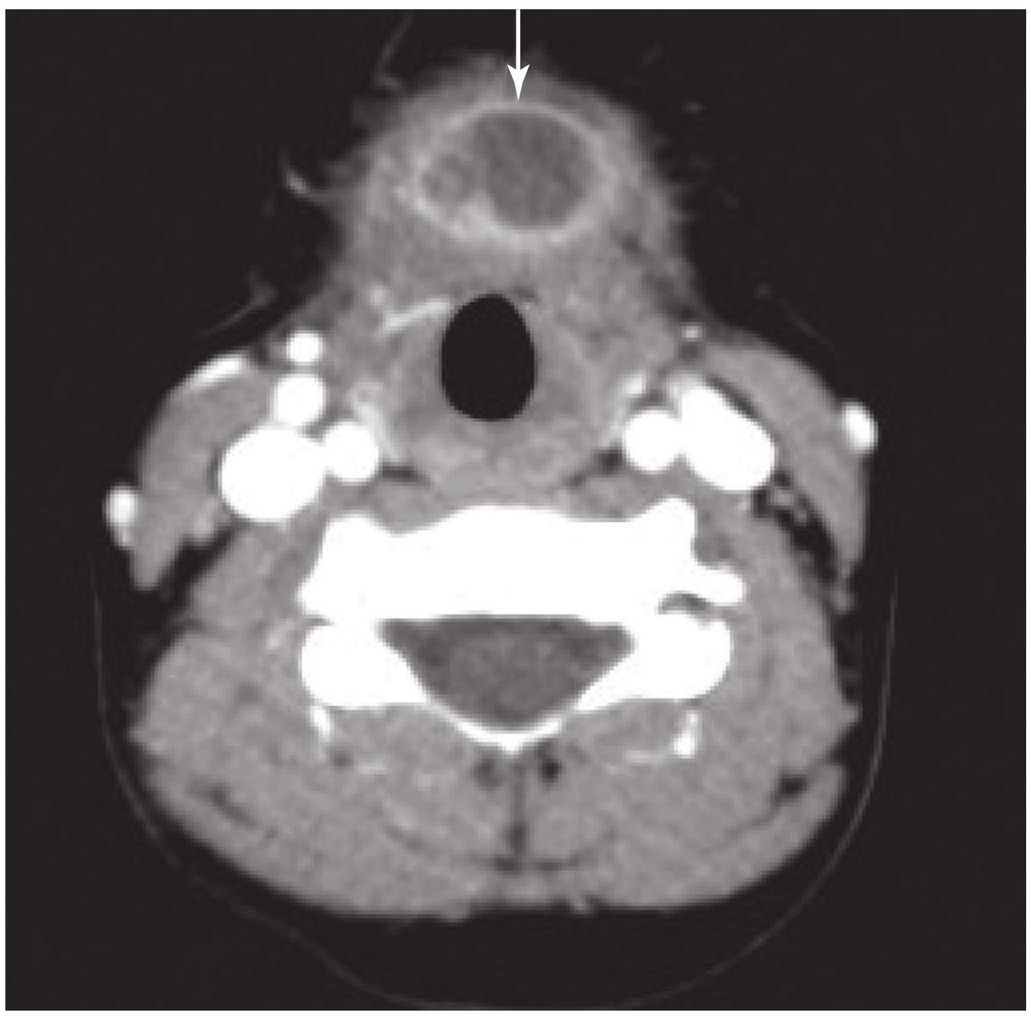


FIGURE 14.72 ■ **Thyroglossal Duct Cyst.** CT of the neck showing anterior thyroglossal duct cyst. (Photo contributor: Lawrence B. Stack, MD.)

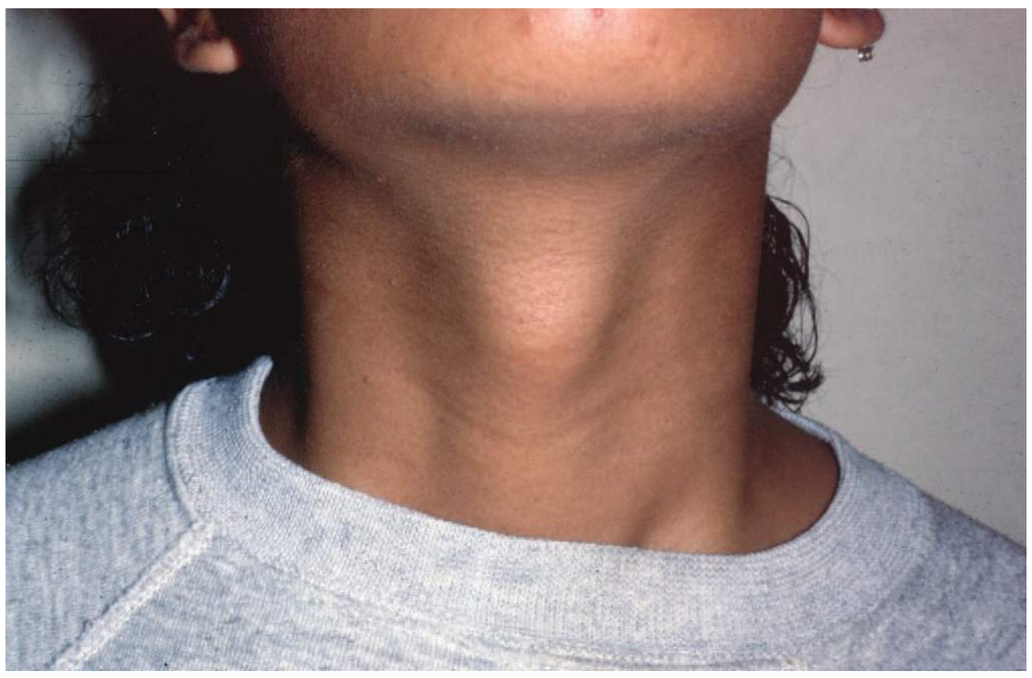


FIGURE 14.71 ■ **Thyroglossal Duct Cyst.** A midline mass is seen in thyroglossal duct cyst. (Photo contributor: Lawrence B. Stack, MD.)

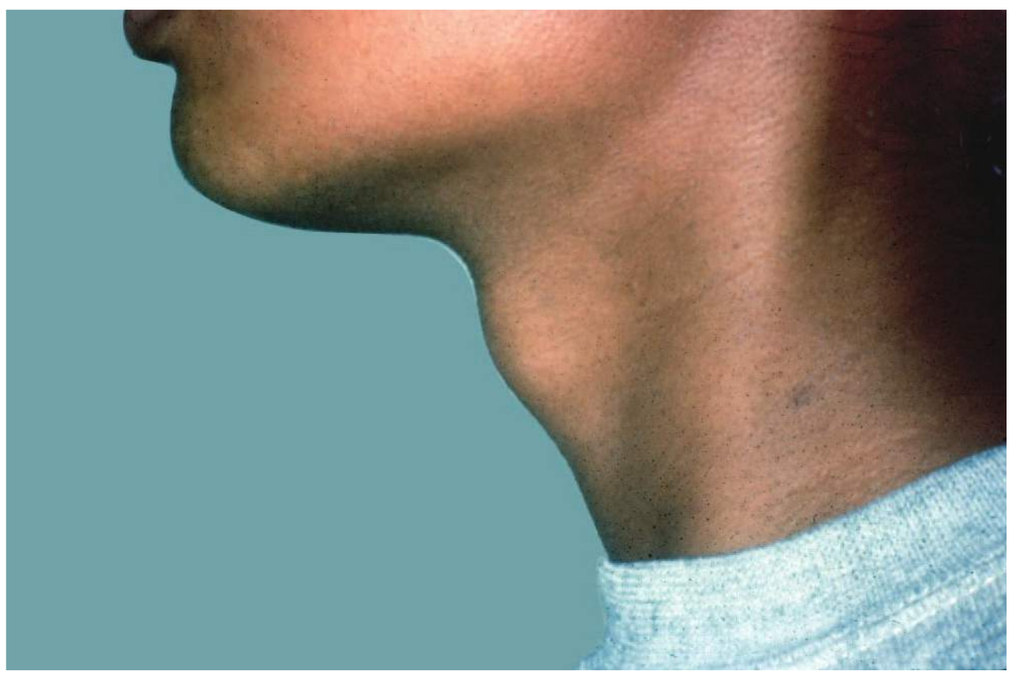


FIGURE 14.73 ■ **Thyroglossal Duct Cyst.** Lateral view of thyroglossal duct cyst. (Photo contributor: Lawrence B. Stack, MD.)

Clinical Summary

Cystic hygromas are congenital lymphatic malformations found most commonly in the neck in infants and children less than 2 years of age. They present as nontender, compressible, unilocular, or multilocular masses with thin, transparent walls filled with straw-colored fluid. Unlike hemangiomas, these lesions rarely undergo spontaneous involution. The vast majority tend to grow and infiltrate adjacent structures. In cases where the tongue is involved, they may lead to upper airway obstruction. The differential diagnosis includes branchial arch remnants, thyroglossal duct cysts, cystic teratomas, cervical lymphadenopathy, and other primary neoplastic diseases.

Elective surgical removal is the treatment of choice in the vast majority of cases, since these lesions do not regress and may affect local tissues. Extent of the lesion should be evaluated prior to its removal (CT). The earlier these lesions can be removed, the better the cosmetic result. Aspiration of the lesion, with or without injection of a sclerosing agent, is another therapeutic option.

Pearls

1. Rapid enlargement of a cystic hygroma is most likely due to bleeding or infection. This can result in airway compromise.
2. Chromosomal abnormalities are found in a significant number of infants with cystic hygromas. These lesions are frequently associated with Noonan, Turner, and Down syndromes.

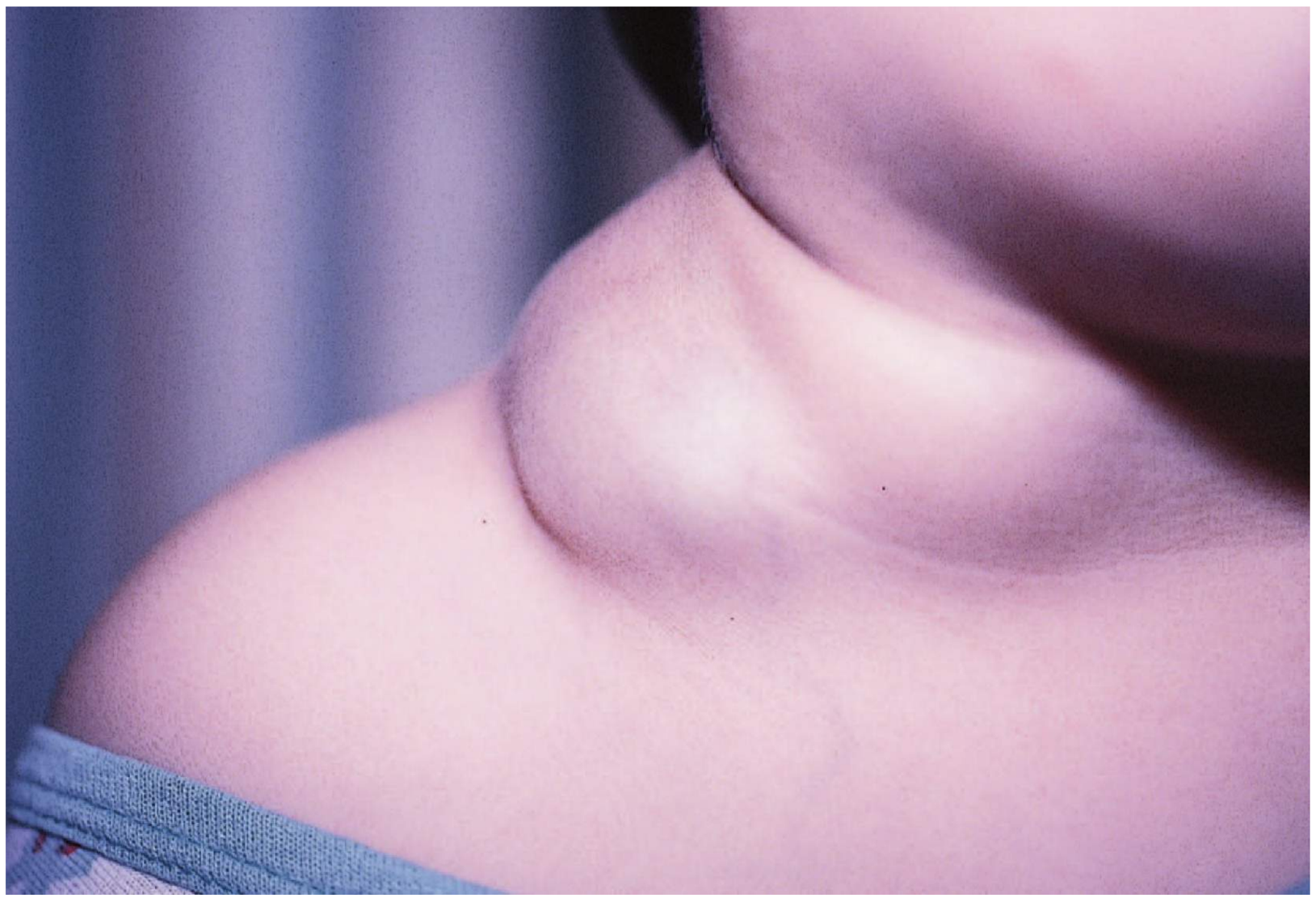


FIGURE 14.74 ■ **Cystic Hygroma.** A bright, supraclavicular, soft, boggy, compressible mass consistent with cystic hygroma. (Photo contributor: Richard M. Ruddy, MD.)

CAT-SCRATCH DISEASE

Clinical Summary

Cat-scratch disease is a benign, self-limited condition caused primarily by Bartonella henselae that usually manifests with regional lymphadenopathy, though visceral organ, neurologic, or ocular involvement can occur. A history of contact with or scratch from a cat (especially kittens with fleas) is usually present. Cat-scratch disease typically starts with an inoculation lesion, which sequentially will appear vesicular, erythematous, and then papular. Lymphadenopathy usually appears within 2 weeks of skin inoculation and may persist for months. In rare cases, patients may develop complications such as encephalitis, osteolytic lesions, hepatosplenic lesions, weight loss, prolonged fever, and fatigue. The differential diagnosis includes lymphogranuloma venereum, bacterial adenitis, sarcoidosis, infectious mononucleosis, tumors (benign or malignant), tuberculosis, tularemia, brucellosis, and histoplasmosis.

Management and Disposition

The disease is usually self-limited and management is primarily symptomatic. Parents and patients should be reassured that the nodes are benign and frequently resolve within 2 to 4 months. Though the evidence is inconclusive, there appears to be a benefit to treating immunocompetent patients with 5 days of azithromycin. Other options include clarithromycin, trimethoprim-sulfamethoxazole, rifampin, or ciprofloxacin. Patients with hepatosplenic, neurologic, or neuroretinal disease should be treated with parenteral antibiotics (rifampin plus gentamicin or azithromycin). If the diagnosis is in doubt, serologic assays for Bartonella species can be sent. Surgical excision of the affected nodes is generally unnecessary.

Pearls

1. Cat-scratch disease is the most common cause of regional adenopathy and should be considered in all children or adolescents with persistent lymphadenopathy.
2. Parinaud oculoglandular syndrome is characterized by a unilateral conjunctivitis and preauricular lymphadenopathy caused by B henselae.
3. Even in the presence of severe and multiple hepatic lesions, liver transaminase levels are normal and hepatomegaly is rare.
4. B henselae is a rare cause of unilateral neuroretinitis and vision loss.
5. In cases treated with antibiotics, a Jarisch-Herxheimer reaction may occur which manifests as fever, tachycardia, hyperventilation, hypotension, peripheral vasodilation, diffuse myalgias, and exacerbation of skin lesions.

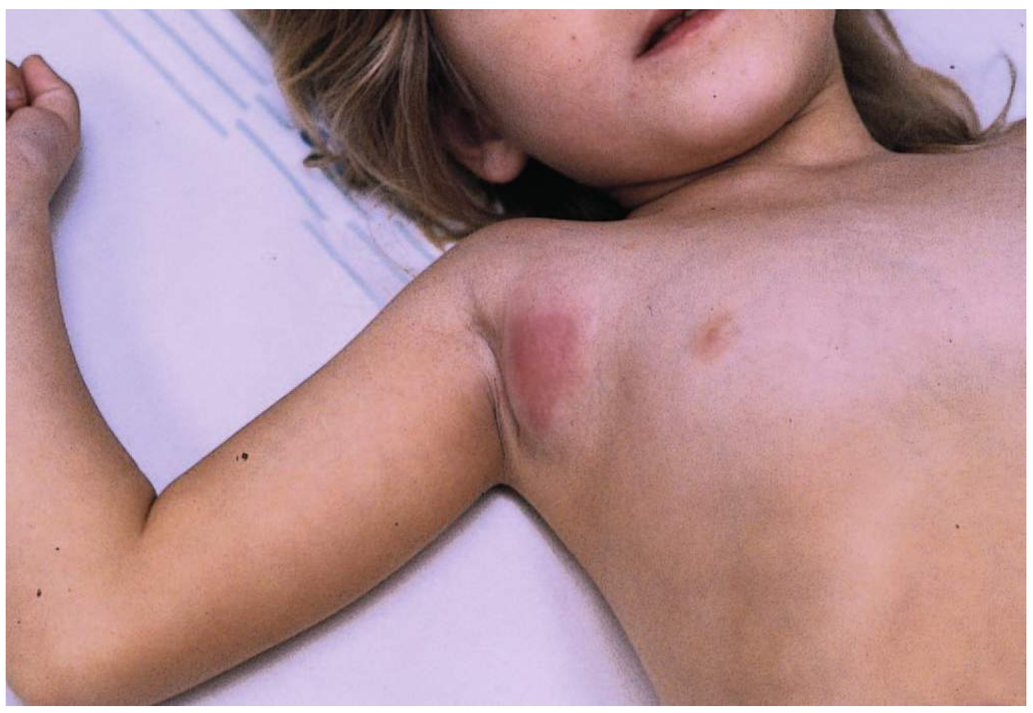


FIGURE 14.75 ■ **Cat-scratch Disease.** An erythematous, tender, suppurative node is seen in a young febrile patient with a history of cat scratch on the extremity. The node required drainage 2 days later. (Photo contributor: Kevin J. Knoop, MD, MS.)

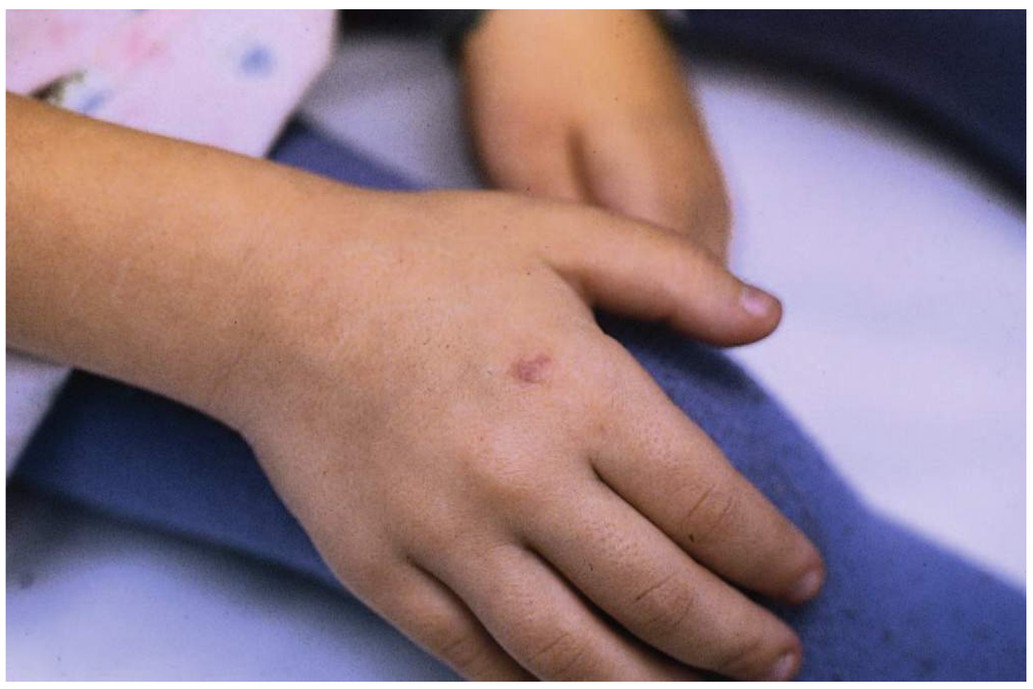


FIGURE 14.76 ■ **Cat-scratch Disease.** The precipitating wound that caused the suppurative node in Fig. 14.75. (Photo contributor: Kevin J. Knoop, MD, MS.)

Clinical Summary

Epiglottitis (AKA supraglottitis) is a life-threatening condition characterized by sudden onset of fever, irritability, sore throat, moderate to severe respiratory distress with stridor, and variable degrees of drooling. It results from a cellulitis of the epiglottis, aryepiglottic folds, and adjacent tissues. The patient generally appears toxic and prefers a sitting position, leaning forward with the neck extended in a sniffing position with an open mouth. With the addition of the H influenzae type B vaccine to the routine immunization schedule, there has been a dramatic decrease in the incidence of epiglottitis as well as a shift in the bacterial etiology. Though H influenzae type B is still the most common cause, many cases are now caused by nontypeable H influenzae, streptococci, staphylococci (especially MRSA), and Candida albicans. Adults typically have a more indolent course characterized by severe sore throat and odynophagia. Direct thermal injury has been reported as a noninfectious cause. On soft tissue lateral neck x-ray, the epiglottis is seen as rounded and blurred (thumbprint sign). Epiglottitis may progress to complete upper airway obstruction if not treated. Differential diagnosis includes acute infectious laryngitis, acute laryngotracheobronchitis (croup), acute spasmodic laryngitis, membranous (bacterial) tracheitis, anaphylactic reaction, foreign-body aspiration, retropharyngeal abscess (RPA), and extrinsic or intrinsic compression of the airway (tumors, trauma, cysts).

Management and Disposition

Immediate intervention is required. If epiglottitis is suspected, the child should be allowed to remain in a position of comfort if they are maintaining an adequate airway. If impending respiratory failure is present, an airway must be established. If possible, this should be done in the operating room or designated area where advanced airway management with sedation is available. An experienced anesthesiologist and surgeon should be readily available in case a surgical airway is necessary. Once the airway has been controlled, the patient should be sedated to avoid unplanned extubation. Parenteral antibiotic therapy should be initiated with ceftriaxone or cefotaxime. Antistaphylococcal coverage against MRSA with clindamycin or vancomycin is also indicated, and therapy can be adjusted once culture results are available.

Pearls

1. Definitive diagnosis of epiglottitis requires direct visualization of a red, swollen epiglottis, preferably in an operating room with advanced airway measures readily available.
2. Allow the child to remain undisturbed in a position of comfort while preparing for airway management. An agitated child is at increased risk for sudden, complete upper airway obstruction.
3. H influenzae type B can still cause epiglottis even in immunized children.

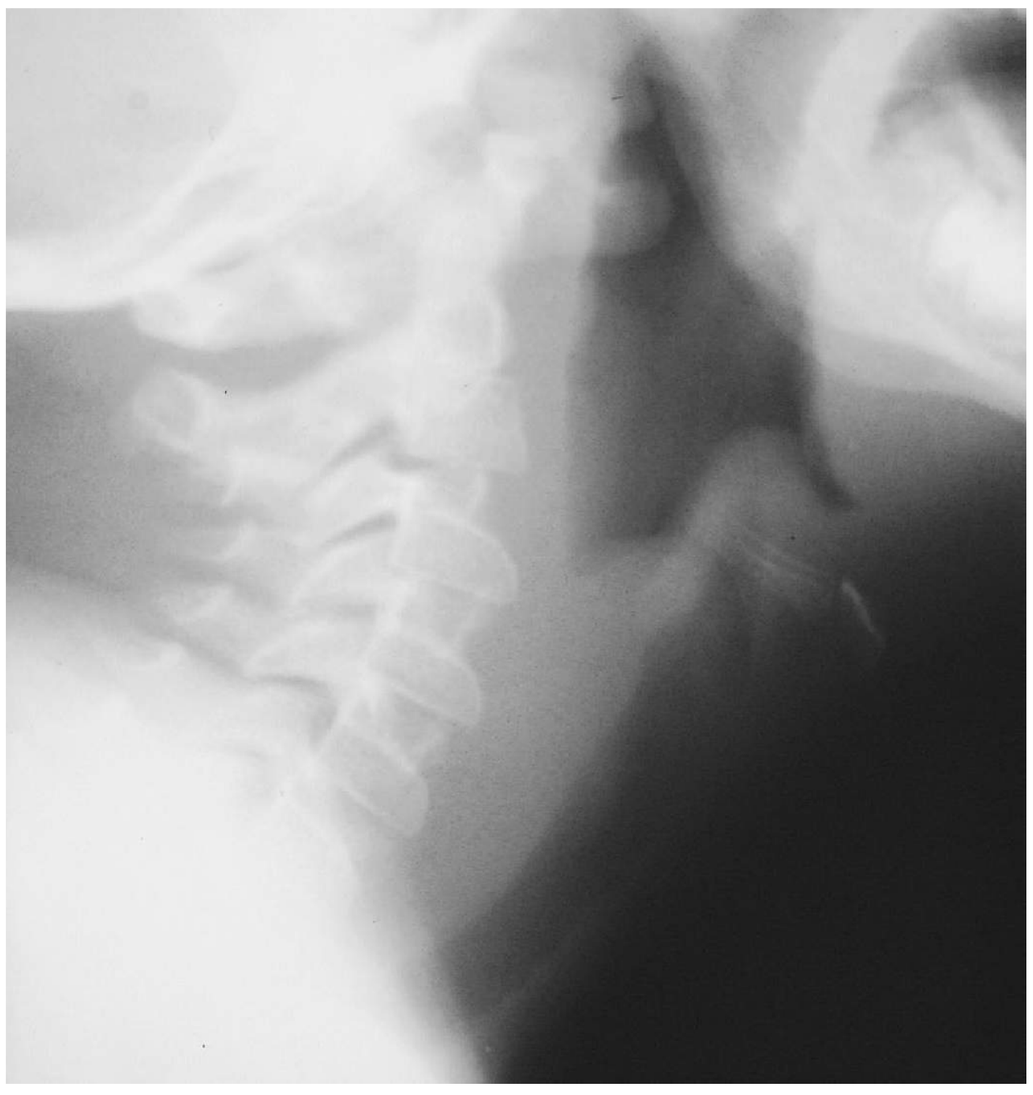


FIGURE 14.77 ■ **Epiglottitis.** Lateral soft-tissue x-ray of the neck demonstrating thickening of aryepiglottic folds and thumbprint sign of epiglottis. (Photo contributor: Richard M. Ruddy, MD.)

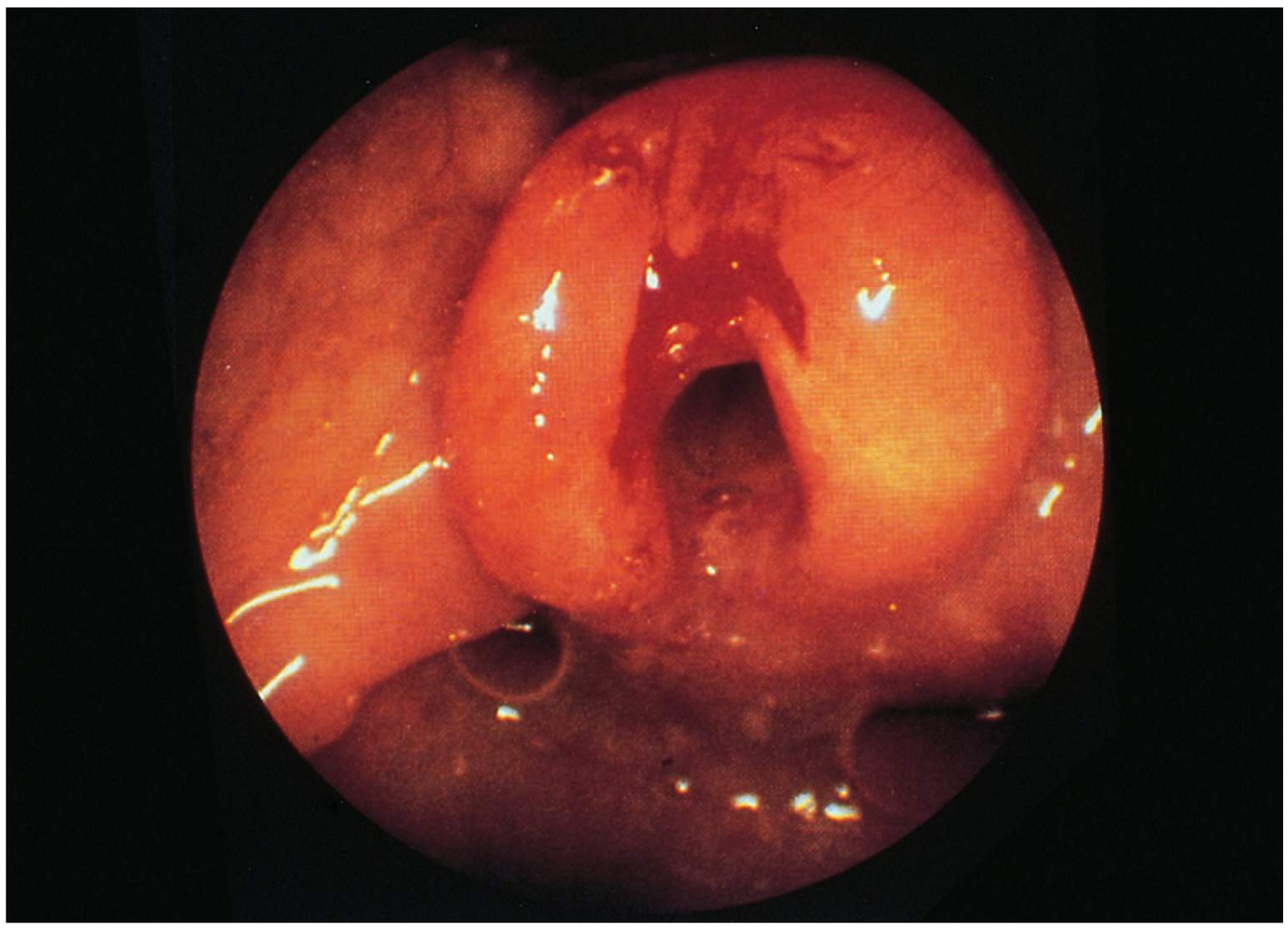


FIGURE 14.78 ■ **Epiglottitis.** The same patient immediately after extubation. Although erythema and some edema persist, the airway is widely patent. (Photo contributor: Department of Otolaryngology, Cincinnati Children's Hospital Medical Center, Cincinnati, OH.)

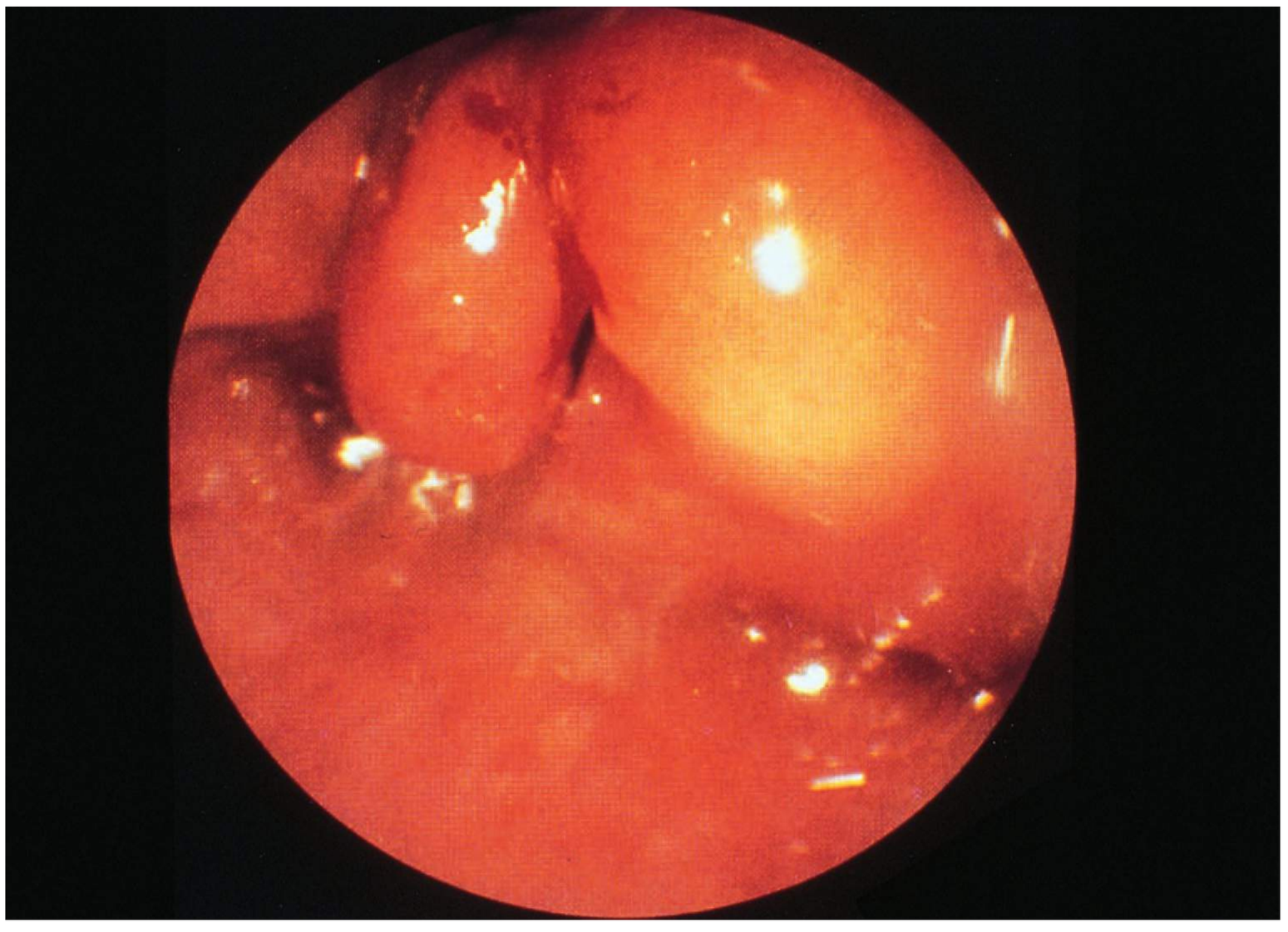


FIGURE 14.79 ■ **Epiglottitis.** Endoscopic view of almost complete airway obstruction secondary to epiglottitis. Note the slit-like opening of the airway. (Photo contributor: Department of Otolaryngology, Children's Hospital Medical Center, Cincinnati, OH.)

Clinical Summary

Retropharyngeal abscess (RPA) usually presents with fever, difficulty swallowing, excessive drooling, sore throat, changes in voice, or neck stiffness. Limitation of neck movement on examination, especially with hyperextension, or torticollis will often be seen. The stiff neck may mimic meningitis. The characteristic retropharyngeal edema is a result of cellulitis and suppurative adenitis of the lymph nodes located in the prevertebral fascia. It is seen on a soft tissue lateral x-ray of the neck as prevertebral soft tissue thickening. The RPA may be preceded by an upper respiratory infection, pharyngitis, otitis media, or a wound infection following a penetrating injury to the posterior pharynx. The differential diagnosis includes pharyngitis, acute laryngotracheobronchitis, epiglottitis, membranous (bacterial) tracheitis, cervical adenitis, infectious mononucleosis, peritonsillar abscess, foreign-body aspiration, and diphtheria.

Management and Disposition

RPA requires immediate assessment of the airway with establishment of a definitive airway if physical exam indicates progressive upper airway obstruction. Antibiotic coverage should be initiated immediately (clindamycin or a β-lactamase-resistant penicillin in areas where S aureus remains susceptible to methicillin). Analgesia should be administered as needed. Radiologic evaluation includes soft tissue lateral neck x-ray and neck CT with contrast to define the extent of infection. In the absence of airway obstruction, medical

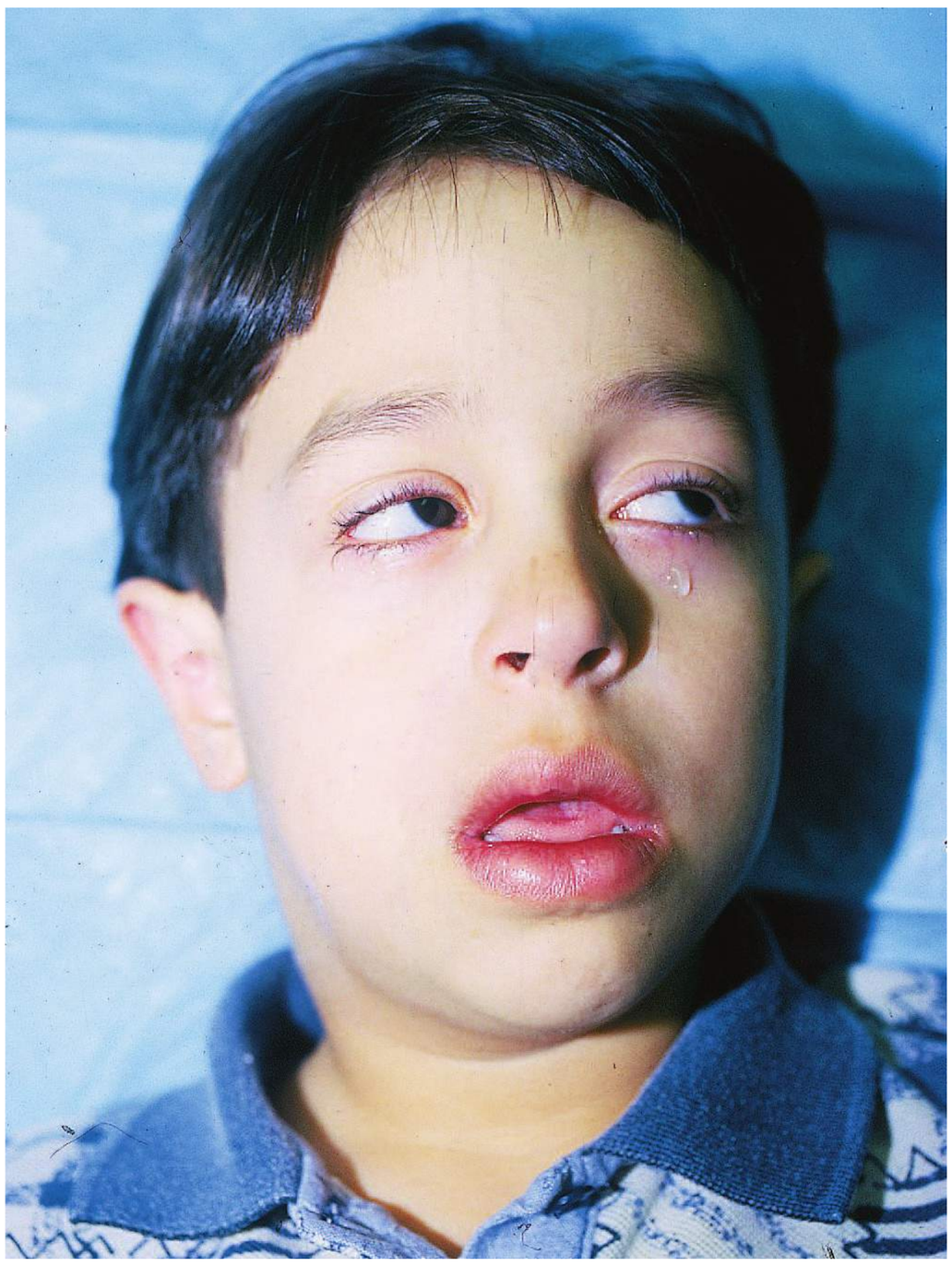


FIGURE 14.80 ■ **Retropharyngeal Abscess.** This ill-appearing 6-year-old child presented with a several-day history of fever, neck pain, sore throat, cough, and headache. Soft tissue lateral radiography of the neck showed thickened prevertebral tissues opposite C2 to C4. CT showed the airway narrowed to a width of 5 mm within the oropharynx. (Photo contributor: Mark Ralston, MD.)

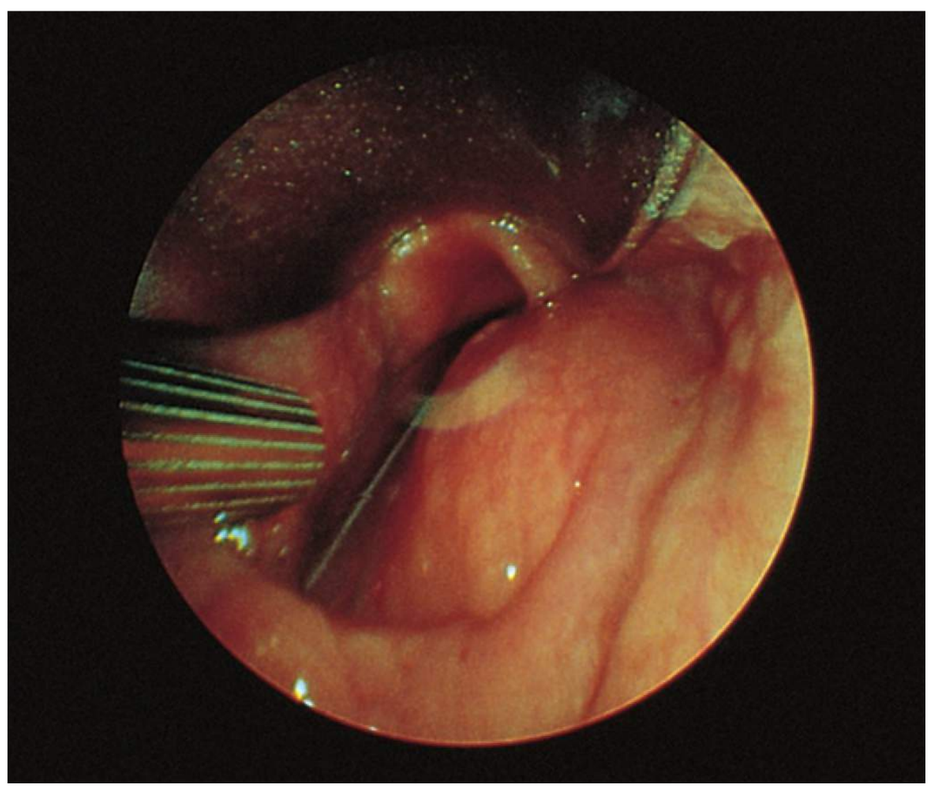


FIGURE 14.81 ■ **Retropharyngeal Abscess.** Endoscopic view of a retropharyngeal abscess. Note the massive swelling posteriorly. (Photo contributor: Department of Otolaryngology, Cincinnati Children's Hospital Medical Center, Cincinnati, OH.)

treatment with IV antibiotics for 24 to 48 hours is first-line therapy. If impending obstruction is present or the infection is unresponsive to IV antibiotic therapy, needle aspiration or incision and drainage should be performed in the operating room. These patients require hospitalization and immediate otolaryngologic or surgical consultation.

Pearls

1. On lateral soft tissue x-ray of the neck, the prevertebral soft tissue can measure up to 7 mm in width at the level of C2. At C6, it can measure up to 14 mm in width. This represents approximately one-half the width of the corresponding vertebral body.
2. The prevertebral soft tissue may appear falsely enlarged during neck flexion or crying.
3. The peak incidence occurs in 3- to 5-year-olds. It is rare beyond 6 years of age as the retropharyngeal lymph nodes involute.
4. RPAs in older patients most commonly arise as a complication of trauma or an immunocompromised state.

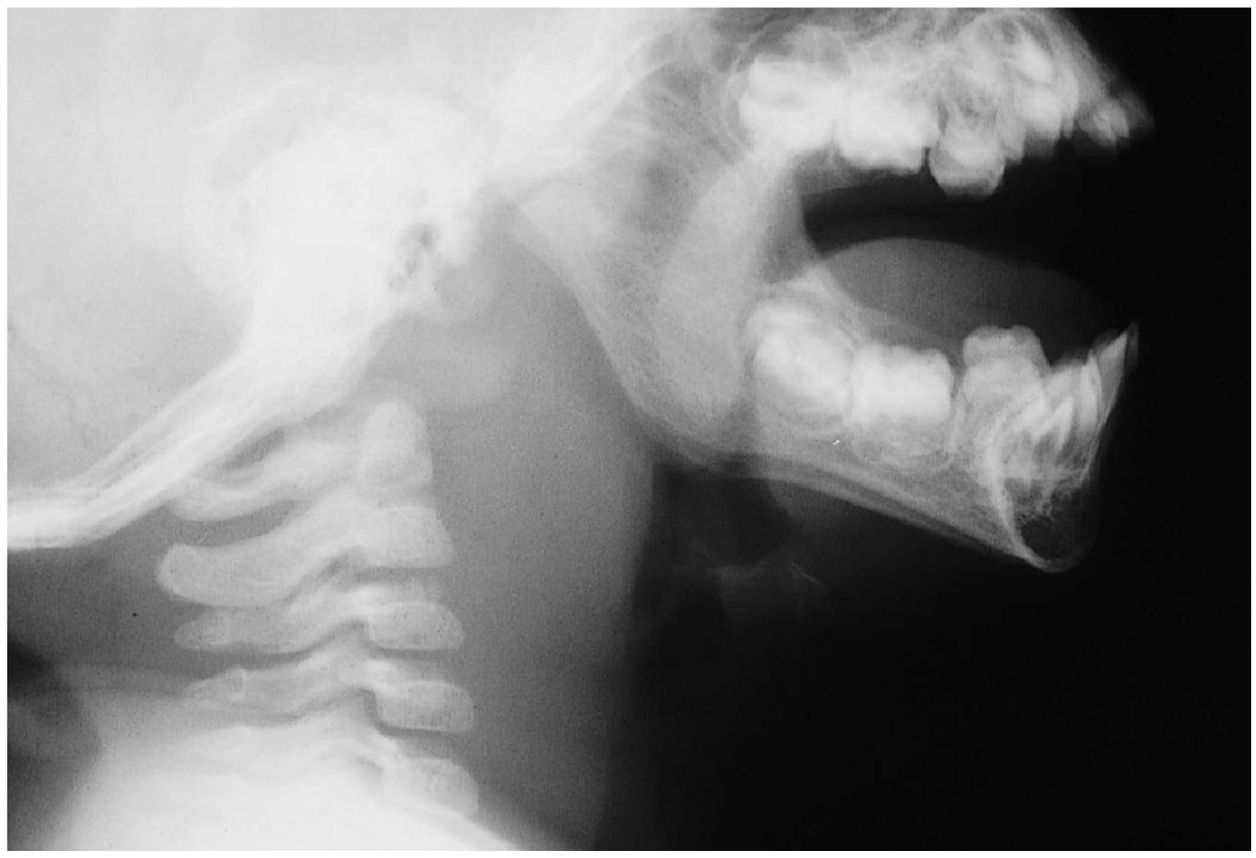
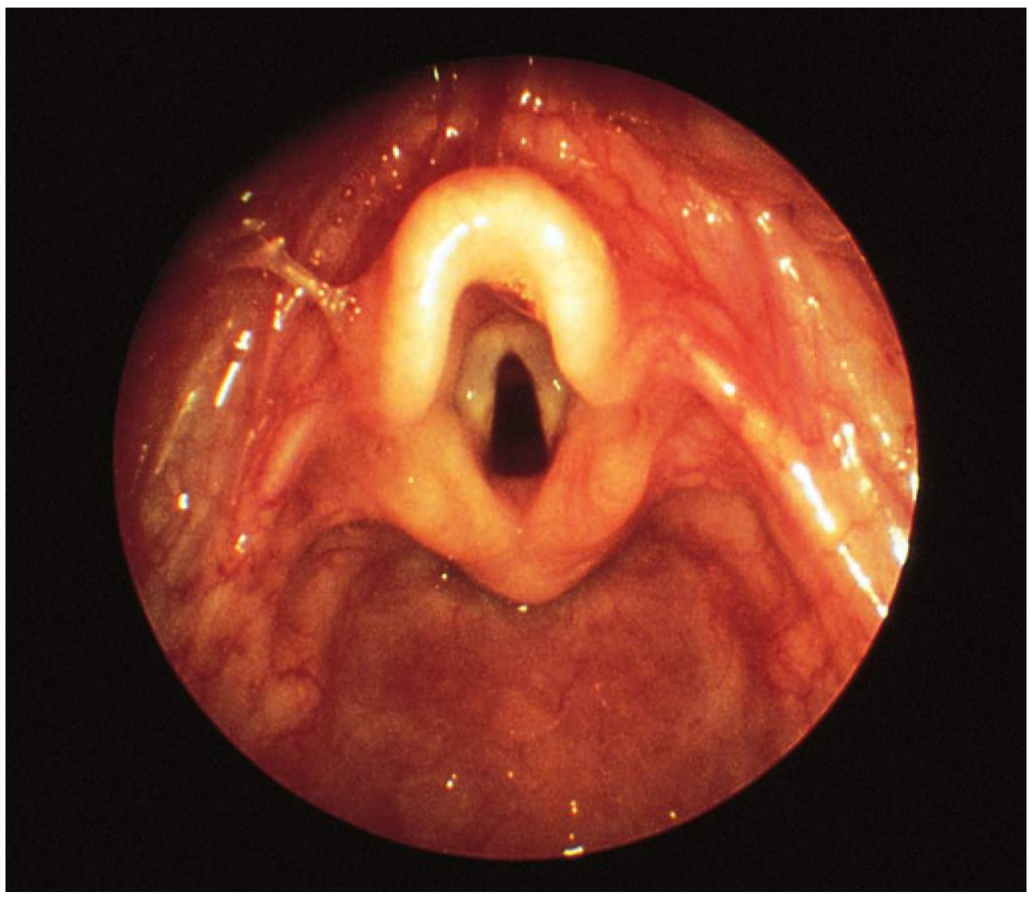


FIGURE 14.82 ■ **Retropharyngeal Abscess.** Lateral soft tissue neck x-ray demonstrating prevertebral soft tissue density consistent with retropharyngeal abscess. (Photo contributor: Richard M. Ruddy, MD.)



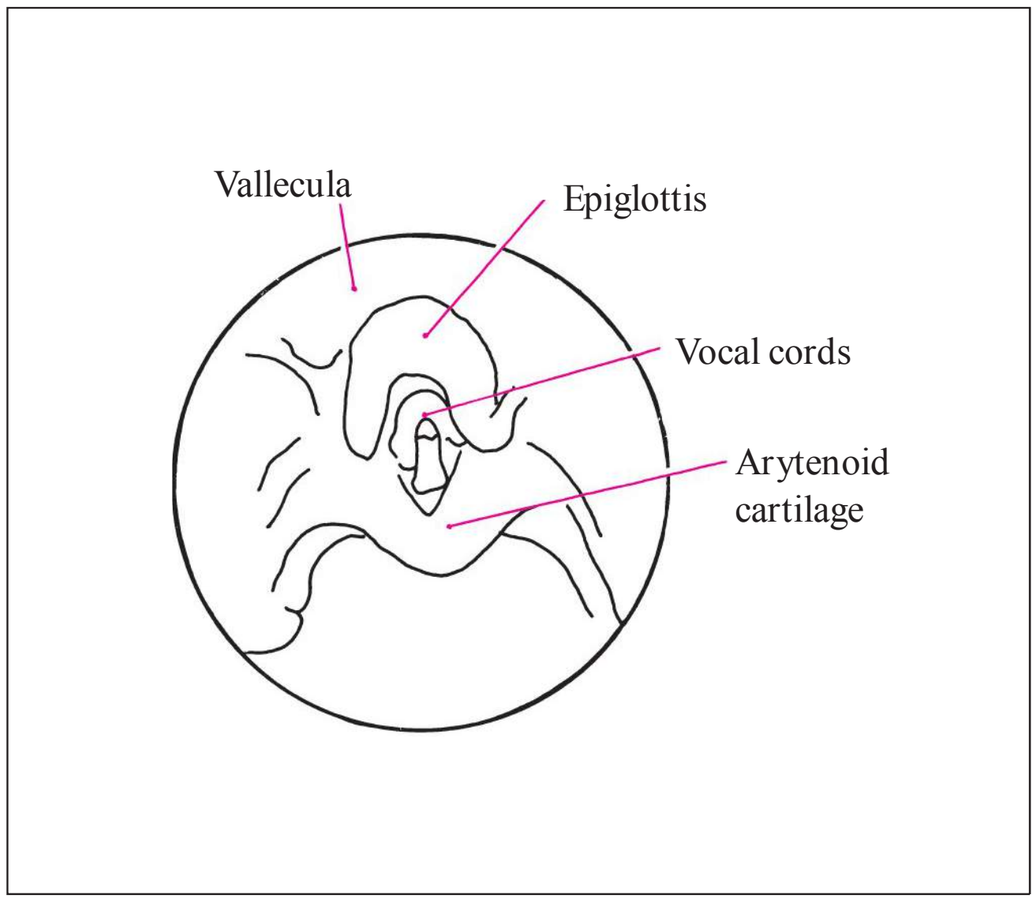


FIGURE 14.83 ■ **Normal Laryngeal Structures.** Endoscopic view of a normal epiglottis and surrounding structures. (Photo contributor: Department of Otolaryngology, Cincinnati Children's Hospital Medical Center, Cincinnati, OH.)

Clinical Summary

The majority of button battery ingestions occur in children less than 6 years of age, peaking between age 1 and 2 years. The most important factors in determining symptoms at presentation, as well as management, are location within the GI tract and duration of contact with the mucosal surface. Batteries that are lodged in the esophagus may be asymptomatic initially or can present with pain, drooling, dysphagia, poor oral intake, cough, vomiting, or fever. Mechanisms of injury associated with button battery ingestion include electrical-current-induced soft tissue injury, liquefactive necrosis resulting from alkali exposure due to battery leakage or the de novo synthesis of alkali at the surface of the battery, and pressure tissue necrosis.

Management and Disposition

Anteroposterior and lateral plain radiographs should be obtained to locate the battery within the GI tract. Button batteries can be distinguished from coins on plain x-ray by demonstration of a double contour. Batteries that are lodged in the esophagus can lead to potentially fatal complications and require immediate removal in consultation with a gastroenterologist or surgeon. This is best accomplished with direct visualization via endoscopy and with a definitive airway in place to prevent aspiration of the battery upon removal from the esophagus. Batteries that have passed into the stomach are likely to traverse the GI tract without complication. However, patients with symptoms of GI injury (pain, obstruction, hematemesis) or those that coingested a magnet are candidates for endoscopic removal. X-rays can be repeated at weekly intervals until passage is documented. Instructions detailing concerning symptoms (abdominal pain, abdominal distention, hematemesis, or blood in the stools) should be provided to the parents at the time of discharge.

Pearls

1. Button batteries with a diameter of 20 mm or greater that remain in the stomach beyond 48 hours should be considered for removal.
2. Button batteries lodged in the nasal cavity or external auditory canal also require emergent removal to prevent complications such as perforation or stenosis.
3. Heavy metal poisoning due to absorption of button battery components though the GI tract is extremely rare.

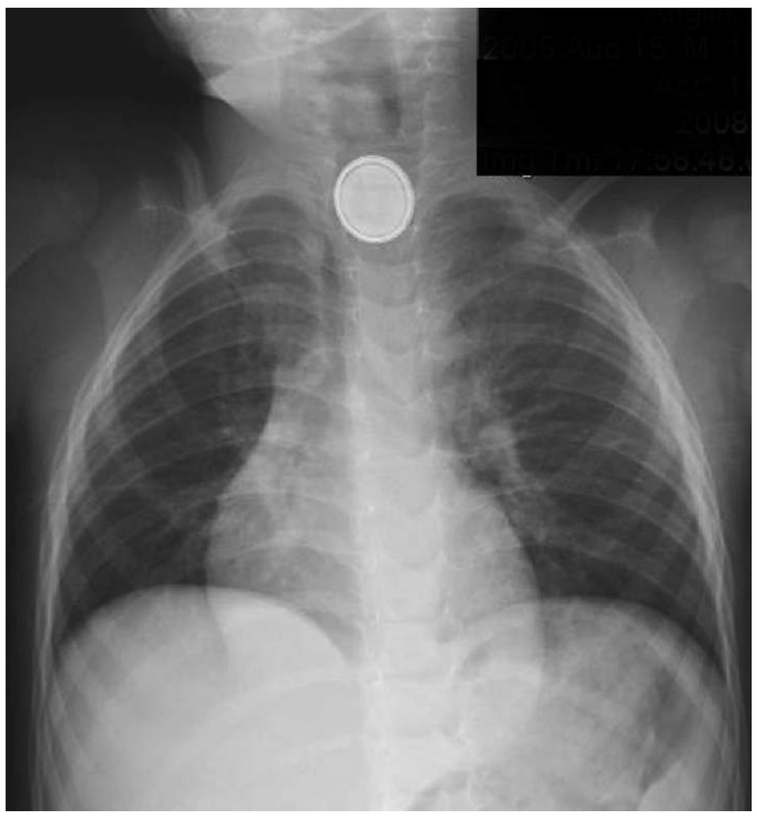


FIGURE 14.84 ■ **Disk Battery Ingestion.** Chest x-ray showing circular "coin-like" appearance with a second concentric ring from the plastic insulating grommet. This identifies the foreign body as a disk battery. (Photo contributor: Scott Manning, MD.)

FIGURE 14.85 ■ **Disk Battery.** This battery was retrieved after ingestion. (Photo contributor: Lawrence B. Stack, MD.)

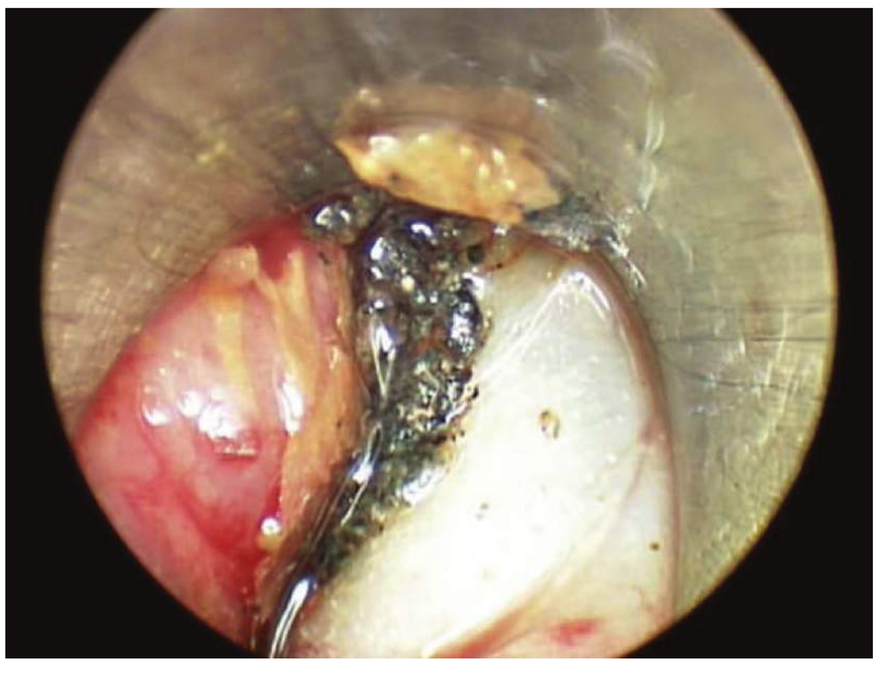


FIGURE 14.86 ■ **Disk Battery Ingestion.** This endoscopic view shows esophageal necrosis after disk battery lodged in the esophagus. (Photo contributor: Scott Manning, MD.)

MEMBRANOUS (BACTERIAL) TRACHEITIS

Clinical Summary

Membranous tracheitis is an acute exudative bacterial infection (S aureus, H influenzae, M catarrhalis, streptococci, and pneumococci) of the upper airway capable of causing life-threatening airway obstruction. It may present as a primary infection or occur as a secondary bacterial complication of a viral infection of the upper respiratory tract. This locally invasive infection of the tracheal mucosa below the vocal cords produces copious purulent secretions. The exudate can form a thick plug that may ultimately lead to an acute tracheal obstruction. Patients appear toxic, with high fever and a croup-like syndrome that can progress rapidly. The characteristic "membranes" may be seen on x-rays of the airway as edema with an irregular border of the subglottic tracheal mucosa. On direct laryngoscopy, profuse purulent secretions can be found in the presence of a normal epiglottis. The differential diagnosis includes acute laryngotracheobronchitis, RPA, epiglottitis, peritonsillar abscess, foreign-body aspiration, and acute diphtheric laryngitis.

Management and Disposition

Otolaryngologic consultation should be obtained as soon as the diagnosis is considered. Direct visualization of the trachea is more important than pursuing a radiologic diagnosis. Aggressive airway management, including endotracheal intubation, may be needed to protect the airway and allow for repeated suctioning to prevent acute airway obstruction. The patient should be admitted to the intensive care unit for close monitoring. Parenteral antibiotic coverage against suspected organisms should be instituted immediately.

Pearls

1. Bacterial tracheitis often presents with acute, severe airway obstruction after a short prodrome. It should be suspected in all patients with an atypical croup-like presentation: unusual age group, toxic appearance, not improving with routine croup therapy, and unusual appearance of the tracheal lumen on plain radiographs.
2. In bacterial tracheitis, up to 50% of soft tissue films may delineate a subglottic membrane.

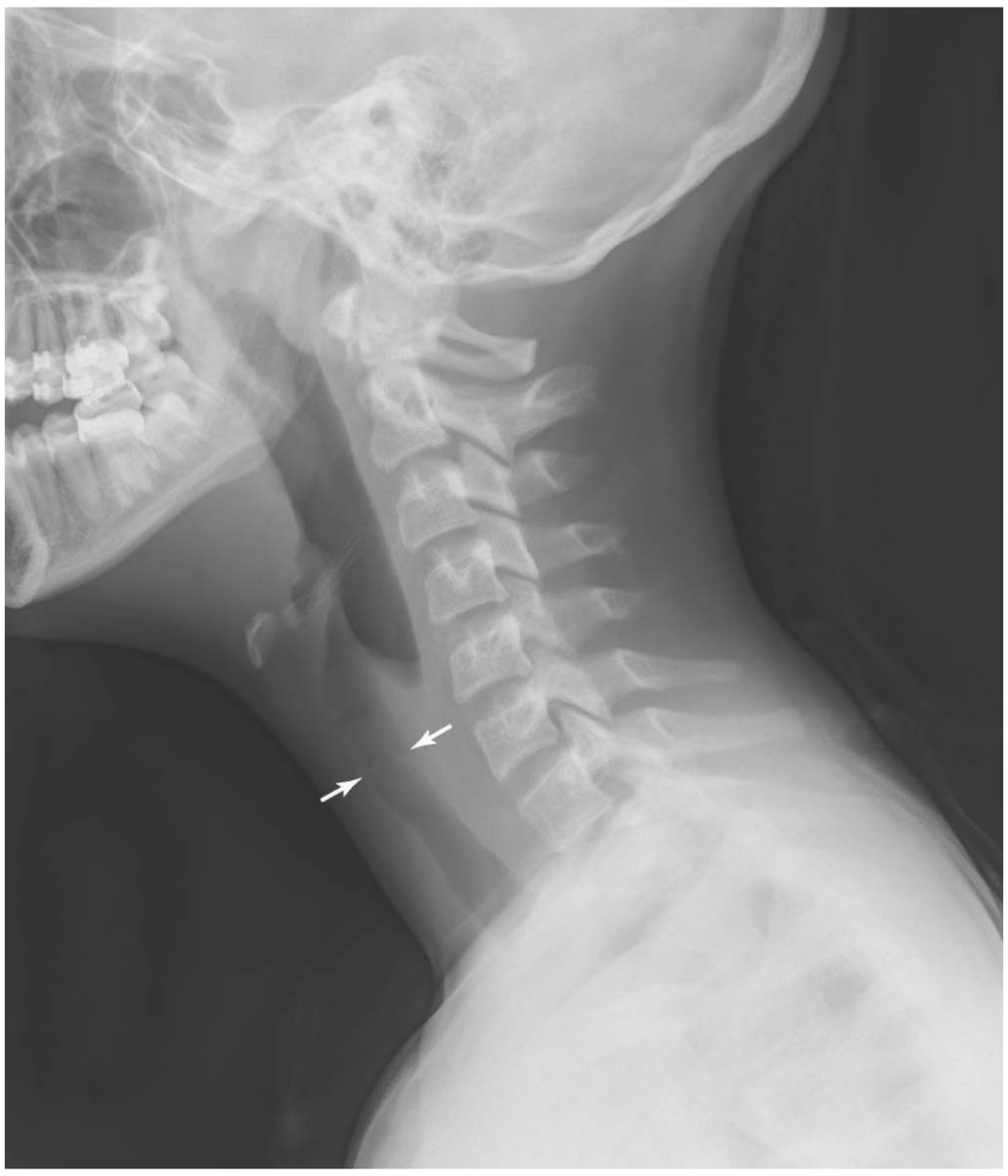


FIGURE 14.87 ■ **Membranous Tracheitis.** Lateral soft tissue x-ray of the neck in a 13-year-old girl with the acute onset of stridor after 3 days of sore throat. Membranes (arrows) are visible in the subglottic region. (Photo contributor: Matthew R. Mittiga, MD.)

Clinical Summary

The nose is the most common site of aerodigestive tract foreign bodies in children. Most children present with a history of witnessed or suspected foreign-body insertion and are symptom free. However, one quarter are discovered incidentally, with no preceding history by the caregiver. Common symptoms include mucopurulent discharge, foul odor, epistaxis, pain, and nasal obstruction. Objects most often include inorganic material (beads or small toys) and food. Unilateral foul-smelling discharge suggests organic or porous material (paper, sponge, foam rubber) or the long-standing presence of a foreign body leading to a localized inflammatory reaction.

On examination, foreign bodies are typically lodged anteriorly on the floor of the nasal passage under the inferior turbinate or superiorly in front of the middle turbinate. Diagnosis is through direct visualization, though high foreign bodies may preclude visual diagnosis even with a head lamp or otoscope. Most are radiolucent, and unless you suspect a concomitant airway or GI foreign body, plain films are unnecessary. Button batteries and paired magnets can be especially dangerous due to the risk of local tissue compromise and should be removed promptly. Otherwise, removal can be an elective procedure. Techniques for removal include the use of positive pressure through the mouth with simultaneous occlusion of the unaffected nares ("mother's kiss maneuver"), removal with alligator forceps, a day hook (right-angle hook), or via a balloon catheter device threaded past the foreign body with subsequent balloon inflation and device retraction (Katz extractor). Children should be adequately prepared and properly immobilized. The value of an adept holder cannot be underestimated. The differential diagnosis includes allergic rhinitis, upper respiratory infection, acute sinusitis, cerebrospinal fluid leak, and nasopharyngeal polyp or mass.

Management and Disposition

Most foreign bodies can be successfully removed in the emergency department. After removal, always inspect for trauma and the presence of additional foreign bodies. Otolaryngology consultation or referral is warranted for poorly visualized posterior foreign bodies, chronic or impacted objects with significant inflammation, button batteries, penetrating or hooked foreign bodies, or any foreign body that cannot be removed due to poor cooperation or limited resources. After successful removal, patients can be safely discharged with instructions to return with any persistent, unilateral nasal discharge, recalcitrant epistaxis, or new fevers. Patients with minor bleeding after removal can be treated with a vasoconstricting nasal spray like oxymetazoline for no longer than 3 days.

Pearls

1. Organic materials that may expand with exposure to moisture (such as dried beans) require urgent removal.
2. A button battery can lead to septal perforation in as little as 4 hours!
3. There is little concern for migration of inert foreign bodies into the airway in children with intact protective reflexes.

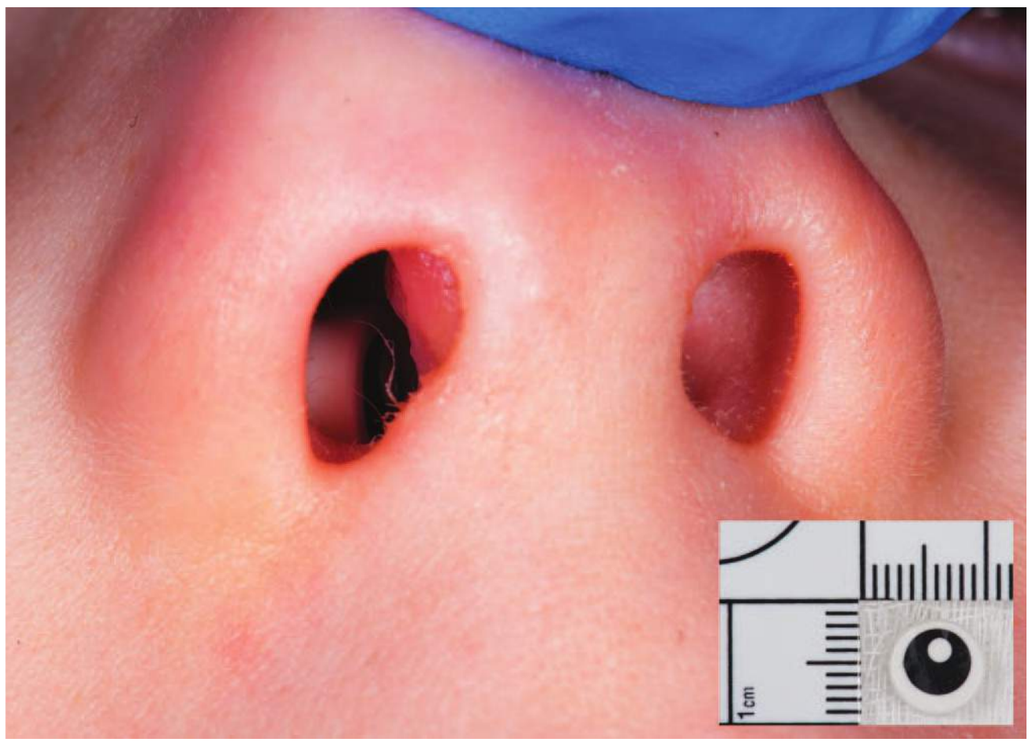


FIGURE 14.88 ■ **Nasal Foreign Body.** A round circular bead (insert) is embedded in the right nostril. (Photo contributor: Lawrence B. Stack, MD.)

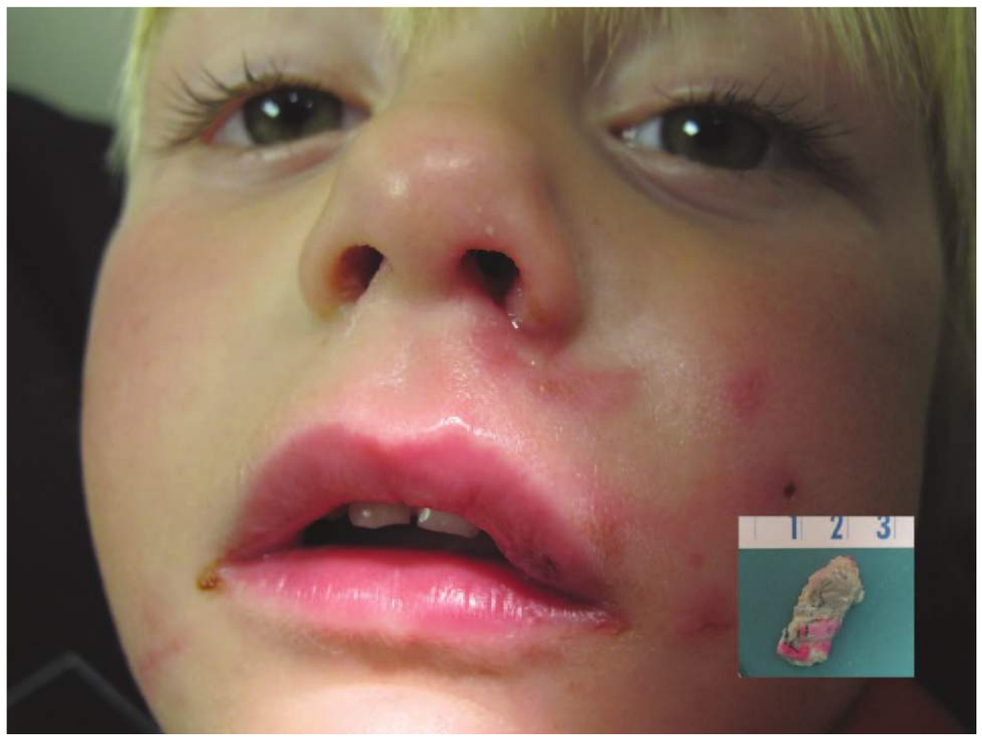


FIGURE 14.89 ■ **Nasal Foreign Body.** Purulent drainage from the left nostril due to a candy wrapper which had been in place for 2 weeks. (Photo contributor: Larry B. Mellick, MD, MS, FAAP, FACEP.)

PHYSEAL FRACTURES: SALTER-HARRIS CLASSIFICATION

Clinical Summary

There are several fracture patterns that are unique to children. Physeal fractures, torus (buckle) fractures, greenstick fractures, and bowing fractures or deformities all occur secondary to the physiologic differences between immature and mature bone. Physis (growth plate) fractures are common because of the relative weakness of the growth plate compared to the cortical lamellar bone. This weakness is related to the relatively large ratio of cells to matrix in the physis. The Salter-Harris (SH) classification was designed to describe each type of physeal fracture, its prognosis, and its treatment.

SH type I: Fracture that extends only through the growth plate. It is a difficult radiologic diagnosis since the fracture line is not visible unless the fracture is displaced. It is usually a clinical diagnosis marked by pain at the location of the physis, but occasionally a physeal widening is observed on the x-ray.

SH type II: Fracture along the growth plate with an oblique extension through a piece of the metaphysis. This is the most common growth plate fracture.

SH type III: Fracture through the growth plate that extends into the epiphysis and articular cartilage into the joint space.

SH type IV: Fracture through the growth plate that extends into both the metaphysis and the epiphysis and into the joint space.

SH type V: Crushing injury resulting in compression of the growth plate.

Management and Disposition

Immobilization by splinting or casting is the treatment of choice in types I and II (minimum of 3 weeks). In these cases, reduction is easy to achieve and maintain. Growth is generally unimpaired. Types III and IV may require open reduction to avoid later traumatic arthritis and, in some cases, growth arrest. Type V fractures are rare and require very close orthopedic follow-up because of arrest of growth caused by the death of the germinal cells. Types III, IV, and V require immediate orthopedic consultation.

Pearls

1. After initial assessment and evaluation, always suspect SH type I if there is evidence of tenderness and swelling around the growth plate area despite negative x-rays.
2. Comparison views of the unaffected side may be useful in diagnosing SH types I and V.
3. Types I and II generally possess a substantial potential for remodeling and have a very good prognosis. Types III and IV threaten growth potential and articular integrity and require anatomic (often surgical) reduction. Type V is very high risk for growth arrest.

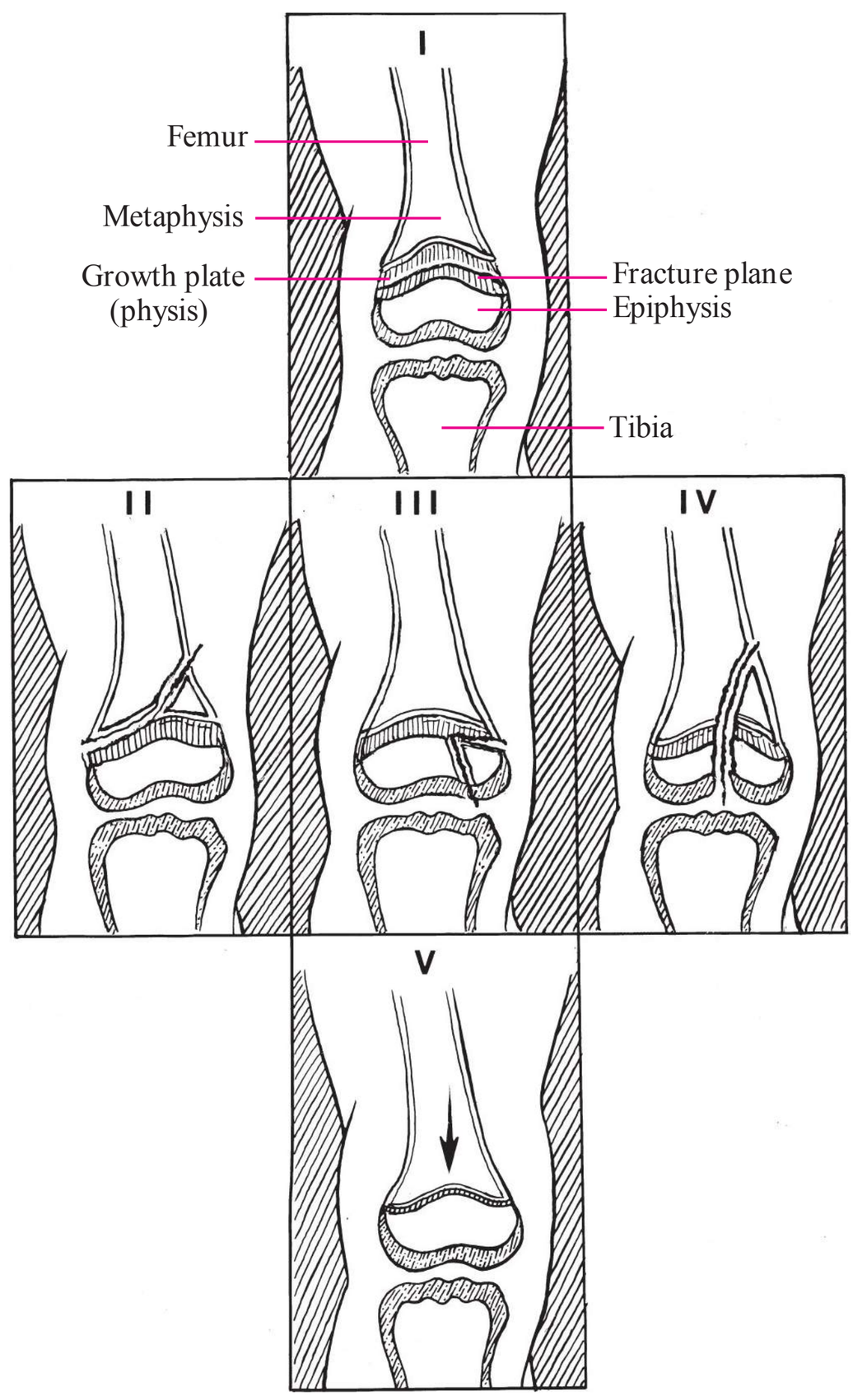


FIGURE 14.90 ■ **Salter-Harris Fracture Classification.** Salter-Harris classification for physeal (growth plate) fractures.

Clinical Summary

The toddler's fracture is a nondisplaced oblique or "spiral" fracture of the mid to distal tibial in a child who has just begun to bear weight. The typical age range is 9 months to 3 years. The clinical history is often nondescript and may include a fall or injury with a twisting mechanism of the lower extremity. Many children will limp or outright refuse to bear weight on the affected leg. Point tenderness over the distal third of the tibia can often be elicited with a careful exam.

Management and Disposition

Obtain anteroposterior and lateral films of the tibia and fibula. The child with a toddler's fracture should be immobilized in a long leg posterior splint or cast. If clinical suspicion remains high for toddler's fracture based on history (refusal to bear weight) or exam (point tenderness over the distal third of the tibia), a splint should be applied even if the initial radiographs are negative. X-rays obtained 10 to 14 days after the initial evaluation may show periosteal reaction or sclerosis. Most toddler's fractures heal without secondary sequelae. The differential diagnosis includes other lower extremity fractures including those associated with nonaccidental trauma, toxic synovitis, septic hip, bony tumors, and osteomyelitis.

Pearls

1. A child who outright refuses to bear weight despite a "negative" radiograph should be treated presumptively for a fracture. A faint hairline fracture may not be apparent on initial x-rays.
2. A toddler's fracture would not be expected if the child cannot yet support their own weight in a standing posture. Spiral fractures of any lower extremity long bone in a nonambulatory child should raise concern for child abuse.
3. Children often walk or crawl on splints leading to an increased risk of pressure sores. Be sure to pad the splint liberally and impress the importance of non–weight bearing on the splint and early orthopedic follow-up for casting.

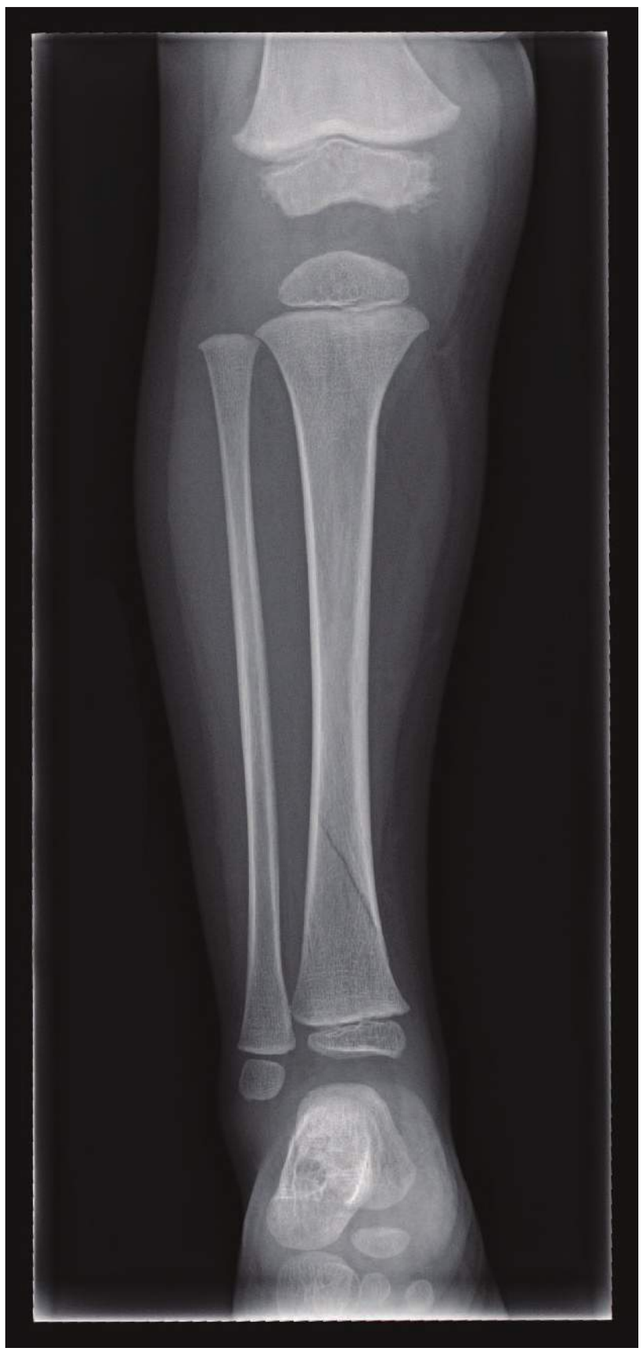


FIGURE 14.91 ■ **Toddler's Fracture.** Anteroposterior radiograph of the tibia and fibula shows an oblique (spiral) fracture lucency in the distal tibial diaphysis. (Photo contributor: Alexander Towbin, MD.)

DACTYLITIS (HAND-FOOT SYNDROME)

Clinical Summary

This painful vaso-occlusive condition is commonly the first clinical manifestation of sickle cell disease in infants. It usually presents in children younger than 5 years of age. Patients are acutely ill with fever, leukocytosis, and swollen hands and/or feet that are exquisitely painful due to bone infarction despite initially normal x-rays. It is not until 1 to 2 weeks later that subperiosteal new bone, cortical thickening, and even complete bone destruction can be seen. The differential diagnosis includes osteomyelitis, trauma, cold injuries, acute rheumatic fever, juvenile rheumatoid arthritis, and leukemia.

Management and Disposition

Treatment of vaso-occlusive crises in sickle cell disease centers around providing adequate fluid balance, oxygenation, and analgesia. Therapy should be individualized. Codeine, hydromorphone, morphine, and ketorolac are analgesic agents commonly used in the treatment of children with painful sickle crises. If fever is present, bacterial infection should be assumed until proven otherwise. CBC, reticulocyte count, and blood cultures should be obtained from all febrile sickle cell patients. If the patient is afebrile, you can omit the blood culture. Empiric broad-spectrum antibiotic coverage should be instituted immediately (third-generation cephalosporin). In cases of dactylitis, very close follow-up is necessary not only for the management of sickle cell disease but also to reevaluate the radiologic changes in the small bones of the hands and feet. In most instances, the previously described changes disappear; however, rarely, shortening of the fingers and toes may occur due to significant infarct.

Pearls

1. Most clinical manifestations of sickle cell disease occur after the first 5 to 6 months of life. Hemolytic anemia gradually develops over the first 2 to 4 months (changes that follow the replacement of fetal hemoglobin by hemoglobin S) and leads to the clinical syndromes associated with an increased percentage of abnormal SS hemoglobin.
2. Sickle cell patients are at high risk for infection from encapsulated organisms due to their functional asplenia. The most common organisms causing osteomyelitis in sickle cell patients are Salmonella sp, S aureus, and S pneumoniae.
3. Dactylitis can often be differentiated from osteomyelitis based on the symmetrical involvement of multiple bones and a negative blood culture.
4. Dactylitis, severe anemia, and leukocytosis within the first 2 years of life increase the risk for adverse sickle cell–related outcomes by age 10 years (death, stroke, frequent pain crises, or acute chest syndrome).

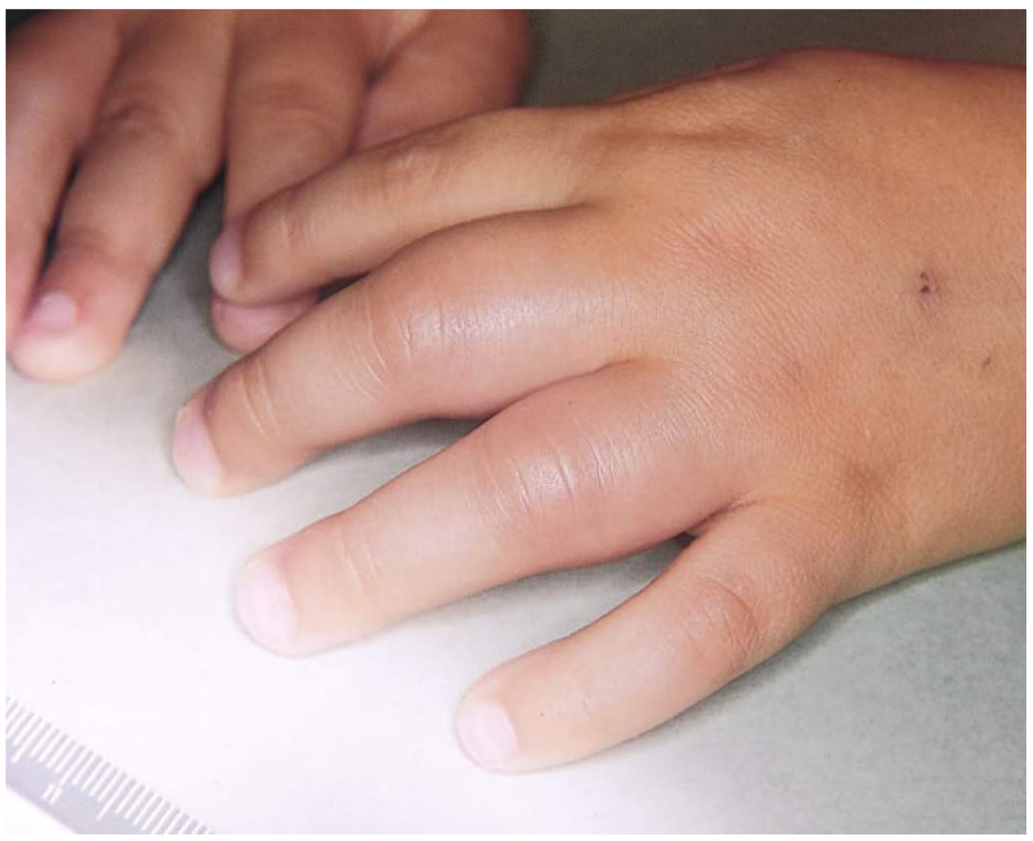


FIGURE 14.92 ■ **Acute Sickle Dactylitis.** Bilateral cylindrical swelling of soft tissue of the hands in sickle cell disease consistent with vaso-occlusive crisis or dactylitis. (Photo contributor: Donald L. Rucknagel, MD, PhD.)

Clinical Summary

A single strand of hair or thread may encircle a finger, toe, or the penis, leading to circumferential strangulation of the appendage. Children in their first year of life are particularly at risk from inadvertent encirclement by a parent's hair or loose thread especially within confining clothing. The affected digit appears edematous, erythematous, and painful. If not corrected, vascular compromise or infection can ensue. The differential diagnosis includes insect bites, trauma, cellulitis of the digit, osteomyelitis, or ainhum (dactylosis spontanea), a painful constriction in the base of the fifth toe of unknown etiology followed by spontaneous autoamputation months to years later.

Management and Disposition

Visualization of the constricting material may be difficult. Edema, erythema, and periarticular skin folds may hide the hair or thread. It is imperative to carefully retract the skin around the proximal aspect of the edematous appendage. A magnifying lens may be helpful in identifying the band. Since the removal can be painful, consider a digital or penile block prior to removal. A topical anesthetic may also be applied to assist in reducing local pain. Using a small hemostat, grasp a portion of the material, cut it with a surgical blade, and unwind it. Occasionally, chemical hair removers have been used to dissolve the hair. This is not advisable on deeply cut skin and will be ineffective for synthetic fibers. Elevation of the involved digit after removal of the constricting agent provides resolution of the edema and erythema within 2 to 3 days. In some cases, the digit's blood supply may have been irreversibly compromised. Subspecialty consultation should be considered whenever neurovascular integrity is in question or if the constricting band cannot be visualized and removed.

Pearls

1. In the vast majority of cases, a clear line of demarcation can be identified between the normal tissue and the affected area.
2. Fussiness or irritability may be the only presenting symptom. Examination for hair tourniquets should be included in the evaluation of any inconsolable infant.
3. There may be an insidious onset with reepithelialization over the hair tourniquet, leaving it difficult to visualize.

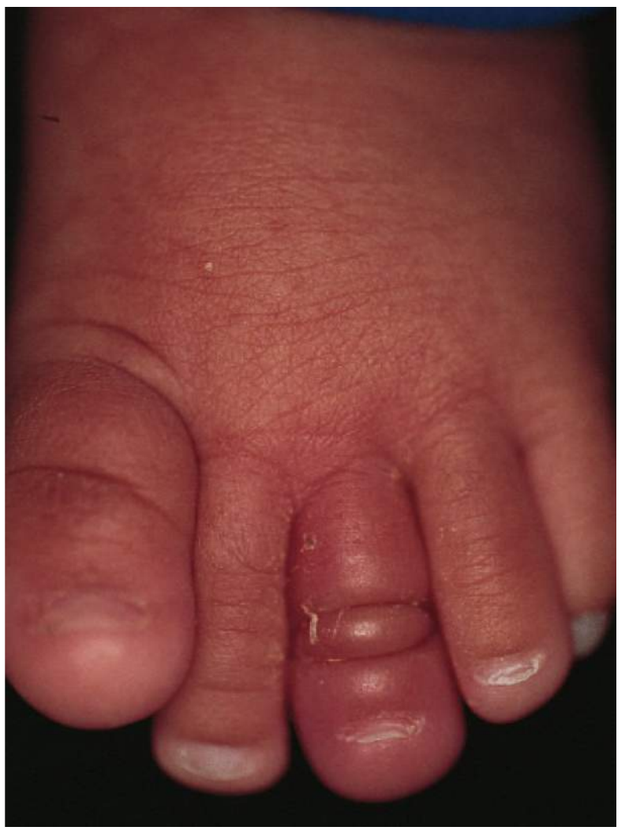


FIGURE 14.93 ■ **Hair Tourniquet.** A strand of hair has encircled the middle toe in two places, causing erythema and swelling. (Photo contributor: Kevin J. Knoop, MD, MS.)

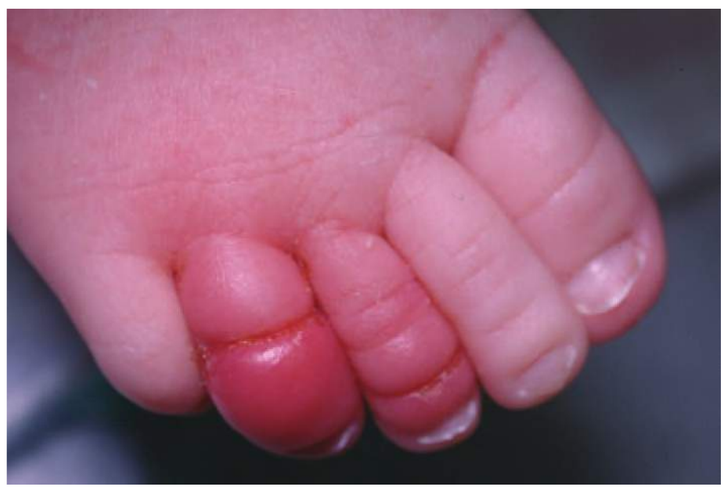


FIGURE 14.94 ■ **Hair Tourniquet.** Erythema distal to the hair tourniquet of the affected toes due to strangulation by the tourniquet. (Photo contributor: Robert W. Hickey, MD.)

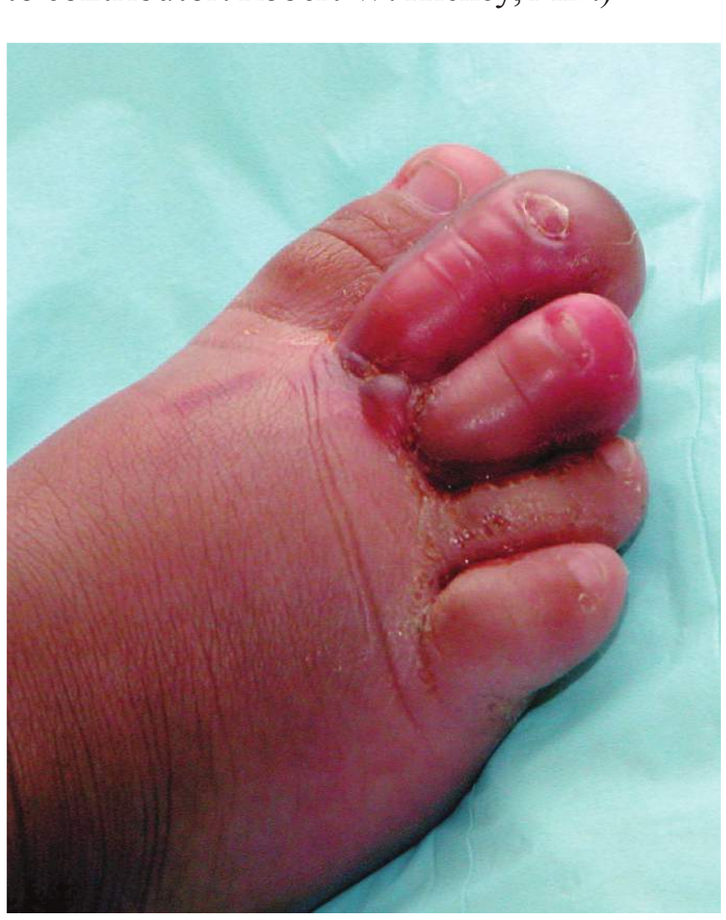


FIGURE 14.95 ■ **Hair Tourniquet.** Erythema and edema from ischemia due to a hair tourniquet. (Photo contributor: David Effron, MD.)

FAILURE TO THRIVE

Clinical Summary

Failure to thrive (FTT) is the inability to maintain a normal growth pattern in weight, stature, and occasionally in head growth. Definitions are varied and include a fall in weight below the second percentile relative to age or growth deceleration that crosses two major percentiles on a standardized growth chart. It is most common in infancy, and the condition is nonorganic (50%), organic (25%), or mixed (25%) in etiology. The diagnosis is made after complete history and physical examination with comparison of the measurements of length (supine in children <3 years of age), weight, and head circumference (maximal occipital-frontal circumference) to standard measurements. In cases of deficient caloric intake or malabsorption, the patient's head circumference is normal and the weight is reduced out of proportion to length/height. The differential diagnosis of FTT is lengthy. Nonorganic disorders include poor feeding technique, disturbed maternal-child interaction, emotional deprivation, inadequate caloric intake, and child neglect. Organic causes are numerous.

FIGURE 14.96 ■ **Failure to Thrive.** This infant has not been able to maintain a normal growth pattern. Note skin folds in upper extremities due to loss of subcutaneous fat. (Photo contributor: Lawrence B. Stack, MD.)

Management and Disposition

Depending on history, physical findings, the social situation, and ability to ensure close monitoring by a primary care physician, most cases can be managed as outpatients. The primary care provider can assist in determining whether outpatient management is indicated. If the diagnosis of FTT is made in the emergency department, admission is suggested to complete the evaluation. That the child presents to the emergency department could be the only indicator of a poor social environment or inadequate access to medical care. Simple testing including CBC, urinalysis, electrolytes, BUN, creatinine, and lead testing is appropriate. More specific testing should be directed by findings in the history and physical examination. Early involvement of social services may facilitate the evaluation and follow-up. Treatment will vary according to the underlying disorder and often involves a team approach, employing the assistance of nutritionists, social services, and home health workers.

Pearls

1. FTT in neglected children is accompanied by signs of developmental delays, emotional deprivation, apathy, poor hygiene, withdrawn behavior, and poor eye contact.
2. The major contributor to FTT is caloric inadequacy. Dietary history should include details of formula preparation, volume consumed, and, in toddlers, the volume of juice consumed.
3. In severely malnourished children, consider assessing for rickets by measuring calcium, phosphorous, albumin, and alkaline phosphatase.

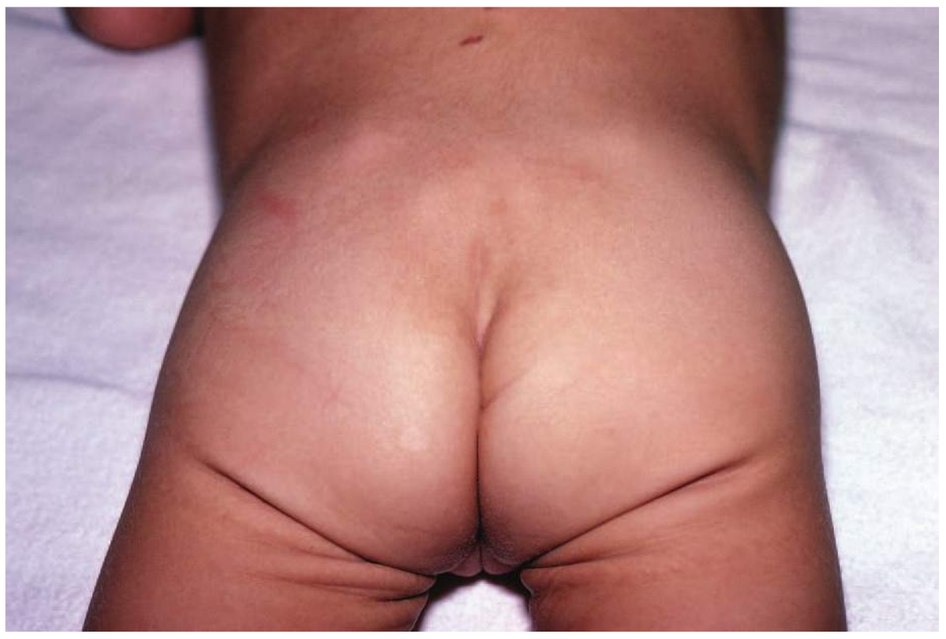


FIGURE 14.97 ■ **Failure to Thrive.** Accentuation of the gluteal folds secondary to loss of subcutaneous fat in an infant with FTT. (Photo contributor: Andrew H. Urbach, MD.)

Clinical Summary

The etiology of dental caries is multifactorial with an interplay between microflora (plaque colonized with Streptococcus mutans), substrate (fermentable carbohydrates from breast milk, formula, or juice), and host (saliva and teeth). Nursing or milk bottle caries result from prolonged and frequent night time breastfeeding or sleeping with a bottle containing milk or sugar-containing juices. The sugars are fermented by the bacteria in plaque, lowering the pH in the mouth and resulting in demineralization of the tooth enamel. The condition generally occurs before 18 months of age and is more prevalent in medically underserved children. Upper central incisors are most commonly involved. Outpatient dental referral is indicated.

Management and Disposition

Parental education and timely referral to a dentist is necessary to prevent complications. If untreated, the caries may destroy the teeth and spread to contiguous tissues. These patients have a high risk for microbial invasion of the pulp and alveolar bone with the subsequent development of a dental abscess and facial cellulitis. In these cases, aggressive treatment with antibiotics (amoxicillin) and pain control, with prompt dental referral for definitive care, is necessary.

Pearls

1. The role of the emergency department physician is to recognize this pattern of dental decay (upper incisors most commonly) and initiate dental referral and parental education.
2. Nursing or milk bottle caries tends to spare the lower front teeth because of the shielding of the lip and tongue and the increased exposure to saliva from the sublingual glands that washes away cariogenic substrates.
3. The newborn mouth is generally devoid of microorganisms. Newborns and infants are colonized with S mutans from parents and family members. Education on avoidance of sharing utensils and cups may help delay colonization of infants.

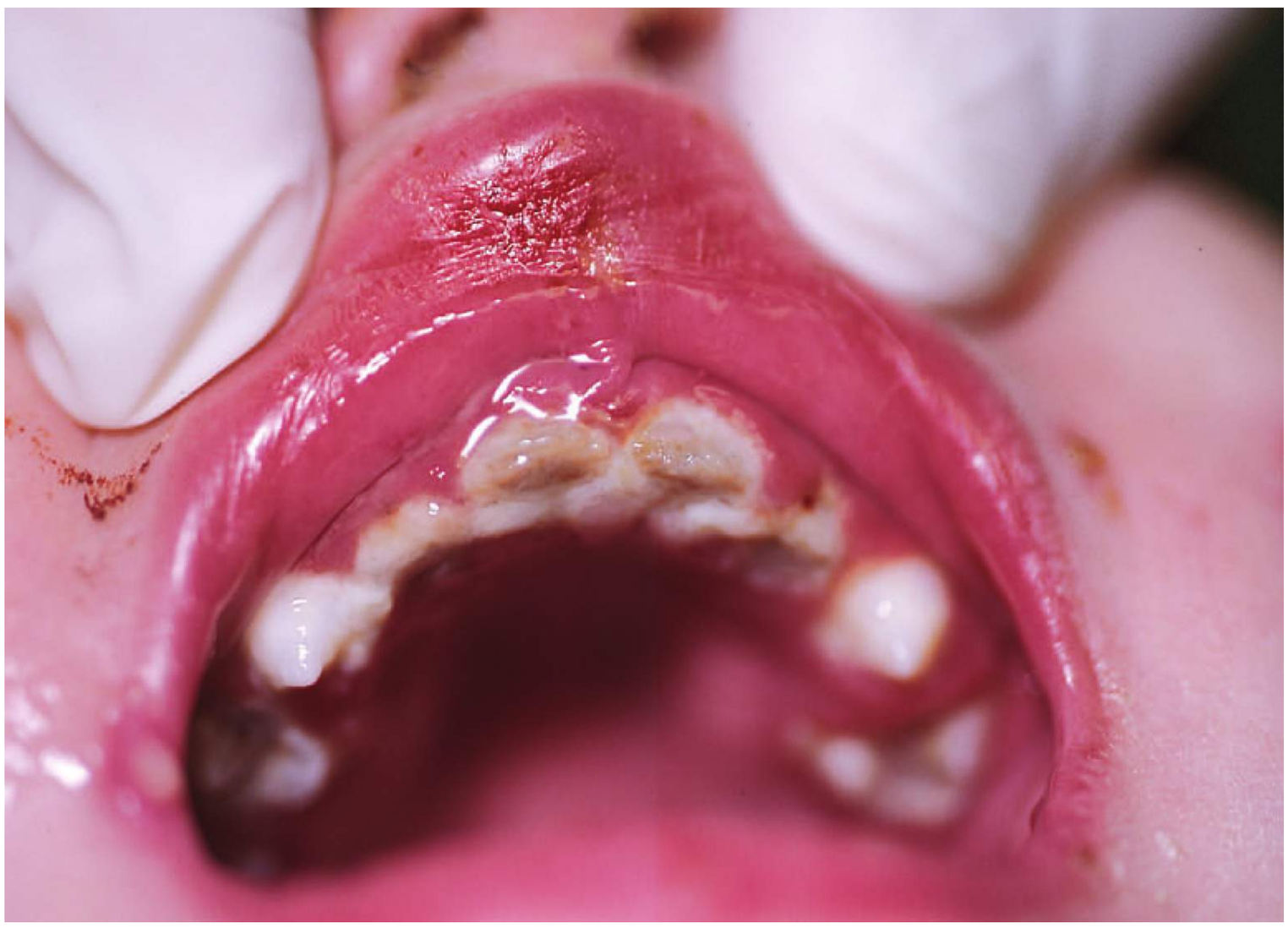


FIGURE 14.98 ■ **Nursing Bottle Caries.** Extensive tooth decay from sleeping with bottle containing milk or sugar-containing juices. (Photo contributor: Lawrence B. Stack, MD.)

NURSEMAID'S ELBOW (RADIAL HEAD SUBLUXATION)

Clinical Summary

Nursemaid's elbow is a condition that occurs commonly in children younger than 6 years of age who are usually picked up or pulled by the extended, pronated arm. The peak incidence is 2 to 3 years of age. These children present unwilling to supinate or pronate the hand on the affected side. Generally they hold the affected arm close to their side in a passive pronation with partial flexion at the elbow. Radiographic studies should be considered only in patients with an unusual mechanism of injury, significant bony tenderness, or those who do not become rapidly asymptomatic after the reduction maneuver. The differential diagnosis includes radial head fracture or complete dislocation, posterior elbow dislocation, condylar and supracondylar fractures of the distal humerus, or buckle fracture of the radius or ulna.

Management and Disposition

Carefully palpate all points of the affected arm for tenderness. There should be minimal to no pain with palpation. Orthopedic consultation is generally not indicated unless an underlying fracture is diagnosed. Reduction is usually achieved by one of two maneuvers:

1. Flexion/supination. Beginning with the elbow flexed at 90 degrees, the wrist of the affected arm is grasped with one hand, while the other stabilizes the elbow. The forearm is then gently supinated while the arm is flexed until the palm approaches the anterior shoulder.
2. Hyperpronation. While holding the elbow in extension, hyperpronation of the forearm is maintained until reduction is achieved.

In either maneuver, a palpable click over the radial head is evident upon successful reduction. Studies have indicated that hyperpronation is more likely to be successful when used as the initial reduction maneuver. The patient usually begins using the arm normally within minutes. When the injury has been present for several hours, reduction may be difficult, and it may take several hours to recover full function of the elbow.

Pearls

1. Radiographs of radial head subluxation typically appear normal.
2. Immobilization after reduction is not necessary.
3. If the patient remains symptomatic after reduction attempts, obtain x-rays to assess for fractures.
4. Up to one-third of children will have recurrence, but rarely beyond age 4 to 5 years.

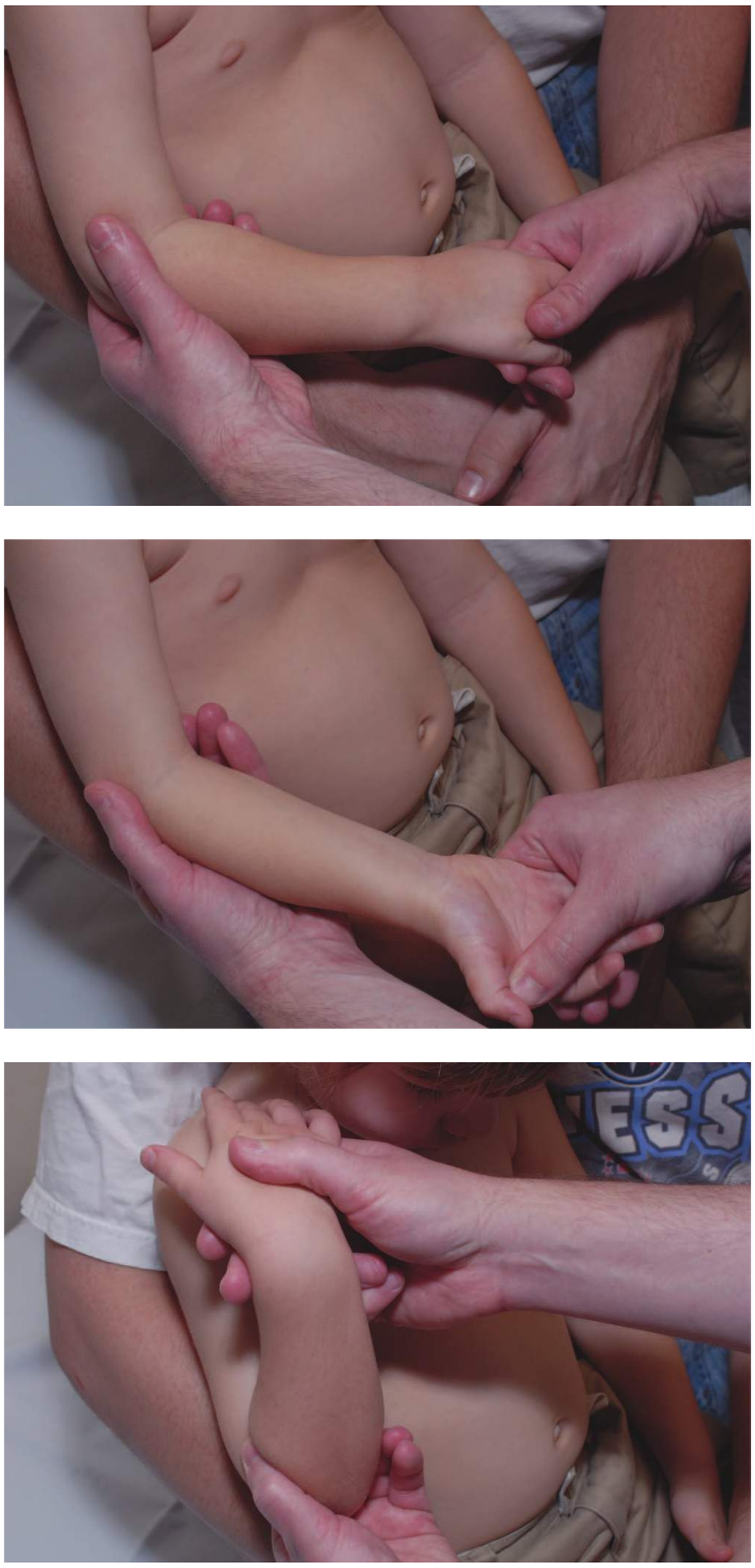


FIGURE 14.99 ■ **Nursemaid's Elbow Reduction.** Simultaneous supination with elbow flexion technique for reduction of nursemaid's elbow. (Photo contributor: R. Jason Thurman, MD.)

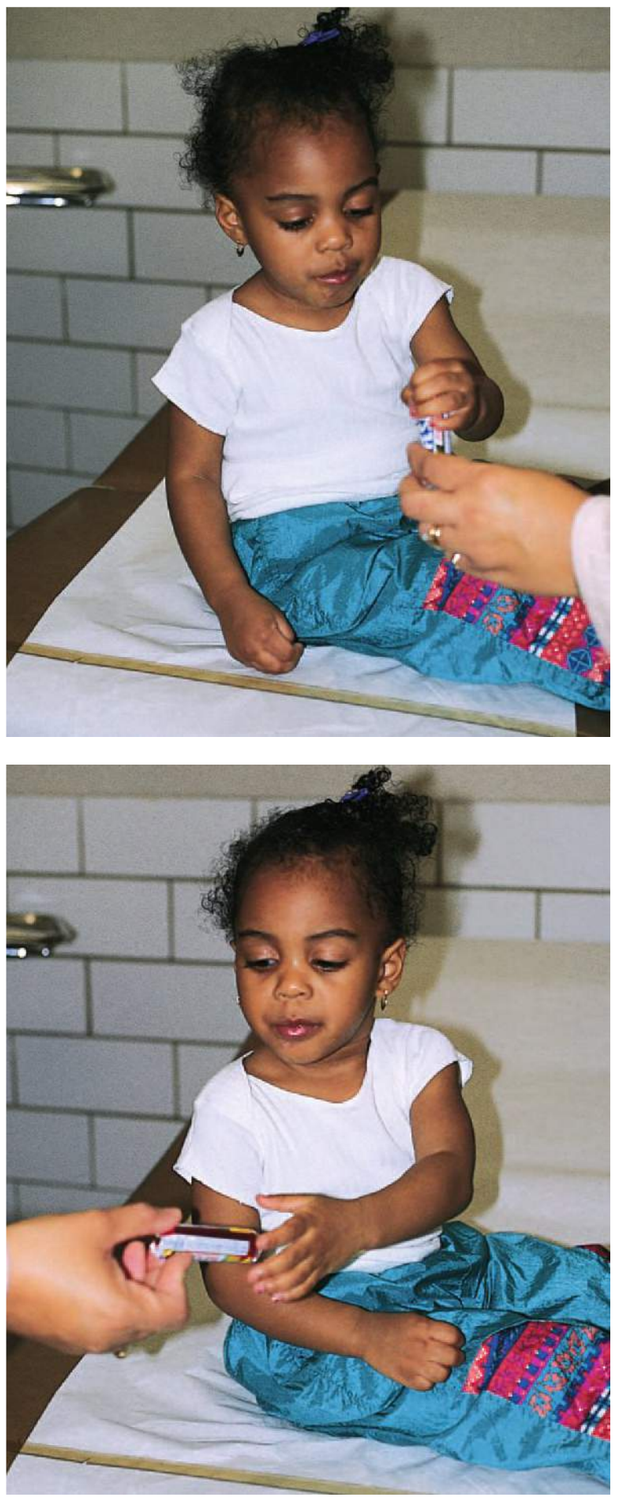
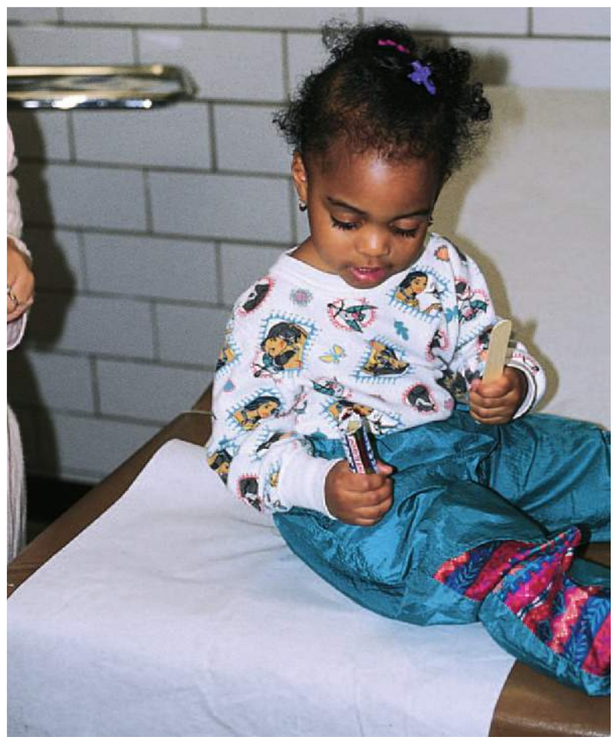


FIGURE 14.100 ■ **Nursemaid's Elbow.** This child presents with pseudoparalysis of the right arm after a pulling injury. Note how she avoids use of the affected arm and preferentially uses the other arm. (Photo contributor: Kevin J. Knoop, MD, MS.)



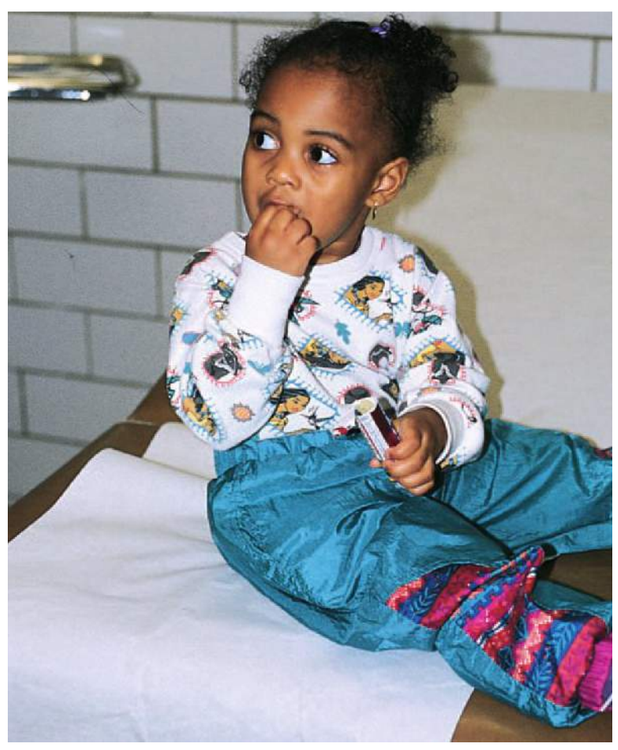


FIGURE 14.101 ■ **Nursemaid's Elbow—Reduced.** After reduction, there is initial reluctance to use the injured arm. With distraction and encouragement, the patient demonstrates successful use of the extremity. (Photo contributor: Kevin J. Knoop, MD, MS.)

ILEOCOLIC INTUSSUSCEPTION

Clinical Summary

Intussusception occurs when one segment of bowel invaginates into itself, most commonly at the ileocecal junction. It is the most common abdominal emergency and cause of intestinal obstruction in children less than 2 years of age. The precise cause is unknown in 75% of cases, though the incidence increases during seasonal viral gastroenteritis outbreaks. Adenovirus, bacterial enteric infections, Meckel diverticula, tumors, hematomas, and vascular malformations are thought to be potential lead points.

Patients typically present with sudden onset of intermittent severe episodes of cramping abdominal pain with inconsolable crying and drawing the legs up to the abdomen. Episodes usually occur at 20-minute intervals, but become more frequent as the illness progresses. Vomiting may follow painful episodes and can eventually become bilious. Initially, children appear normal between episodes, but progressive lethargy eventually develops. Bloody stools (occult or gross), a sign of intestinal ischemia, are seen in 70% of patients. A sausage-shaped mass may be palpated in the right upper quadrant. The differential diagnosis includes viral gastroenteritis, constipation, HPS, intestinal malrotation with volvulus, appendicitis, and meningoencephalitis.

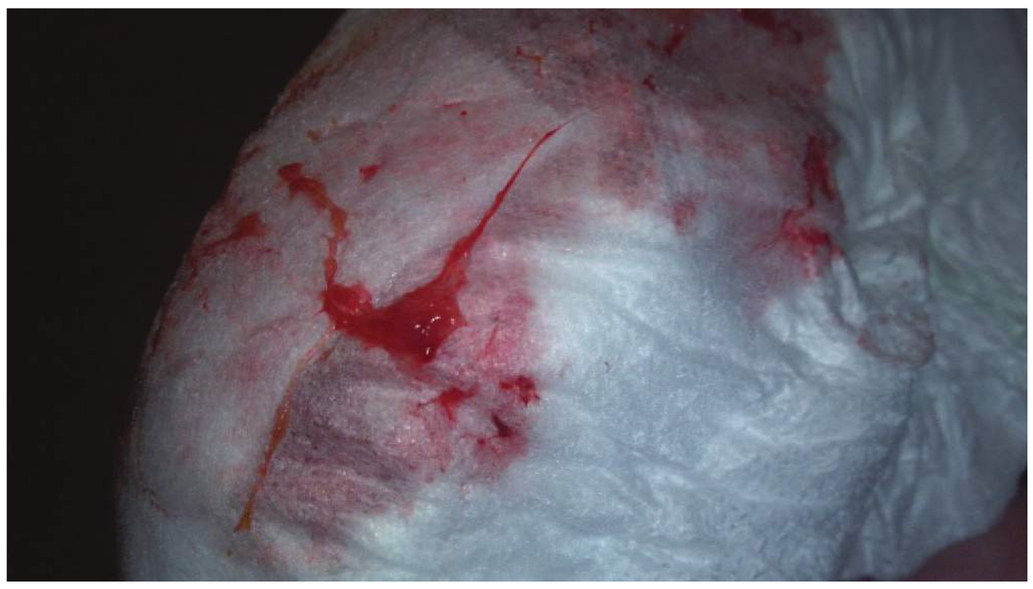


FIGURE 14.102 ■ **Ileocolic Intussusception.** Bloody "currant jelly" stools in a hypersomnolent 3-month-old female infant with transverse colon intussusception reduced by air enema. (Photo contributor: Donald H. Arnold, MD, MPH.)

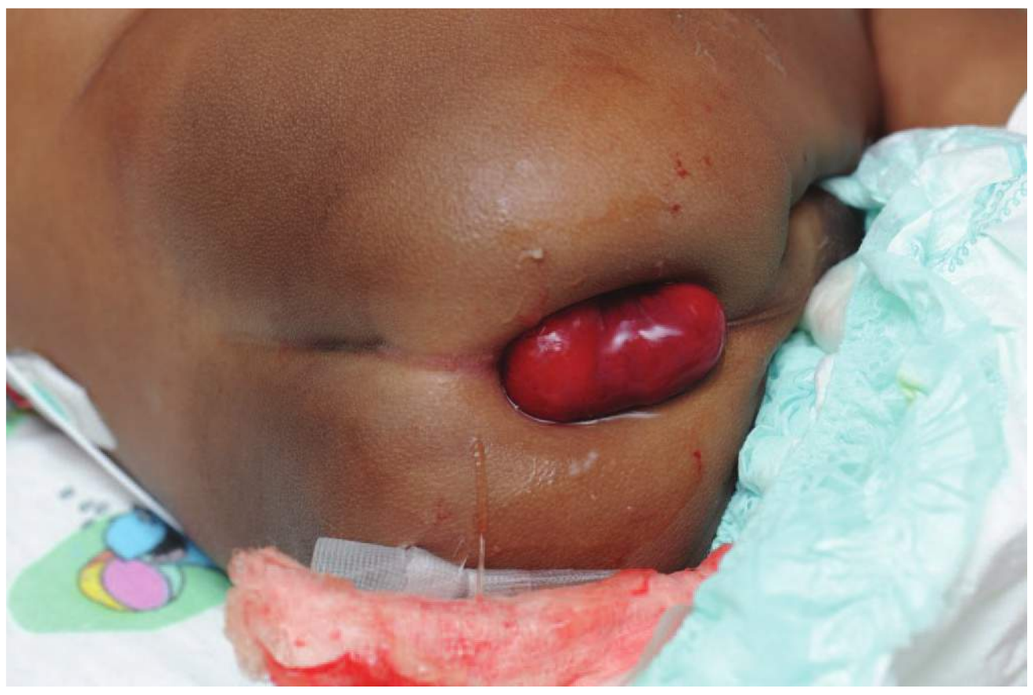


FIGURE 14.104 ■ **Rectal Prolapse.** Protrusion of the sigmoid colon is seen in this infant. (Photo contributor: Lawrence B. Stack, MD.)

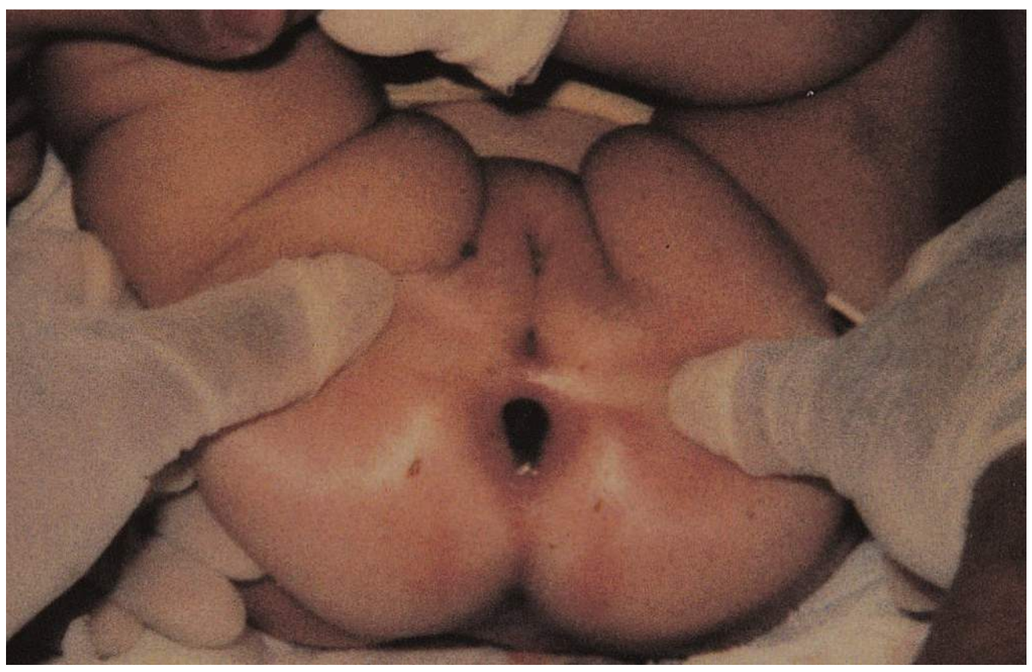


FIGURE 14.103 ■ **Intussusception.** The apex of the intussusception may extend through the anus mimicking rectal prolapse. It is distinguished from rectal prolapse by the separation between the protruding intestine and the rectal wall. (Photo contributor: Binita R. Shah, MD. Used with permission from Shah B, Lucchesi M, Amodio J, Silverberg M. Atlas of Pediatric Emergency Medicine. 2nd ed. New York, NY: McGraw-Hill; 2013: Fig. 10-3, p. 379.)

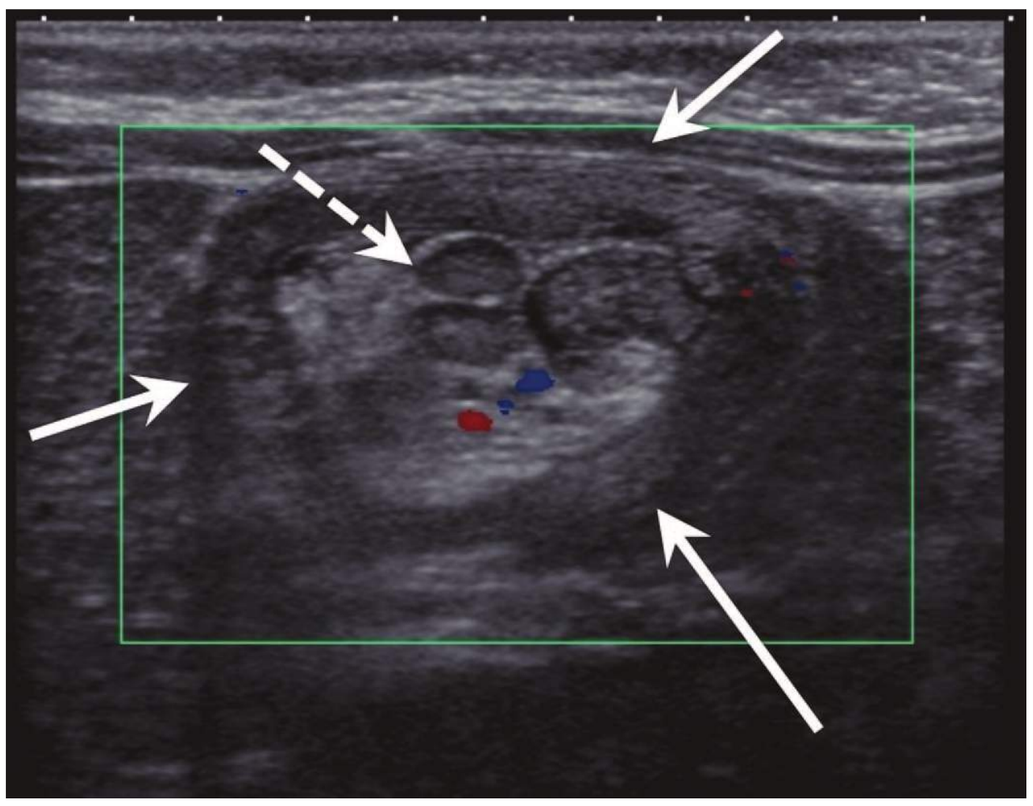


FIGURE 14.105 ■ **Ileocolic Intussusception.** Ultrasound showing intussuscepiens (arrows) containing echogenic fat and multiple lymph nodes (dashed arrow). (Photo contributor: Alexander Towbin, MD.)

Management and Disposition

If the diagnosis is uncertain, ultrasound is the imaging test of choice often demonstrating the classic "coiled spring" or "bull's eye" lesion. Plain x-rays obtained early in undifferentiated cases of abdominal pain may show a target or crescent sign, along with a paucity of cecal gas. Treatment begins with nonoperative reduction, using air enema under fluoroscopic or sonographic guidance utilizing pneumatic pressure to reduce the intussusception. The success rate is 80% to 95%. The risk of perforation is <1%. Observe patients for 12 to 24 hours following reduction. Seek surgical consultation, as laparoscopy is indicated when nonoperative reduction is unsuccessful or incomplete.

Pearls

1. Currant jelly stools are seen in only 20% of patients and are a combination of blood and mucous.
2. The classic triad of pain, palpable mass, and bloody stools is seen in less than 15%.
3. Infants can present with profound lethargy alone, with one-third having no apparent abdominal pain. Consider intussusception in any infant with altered mental status.

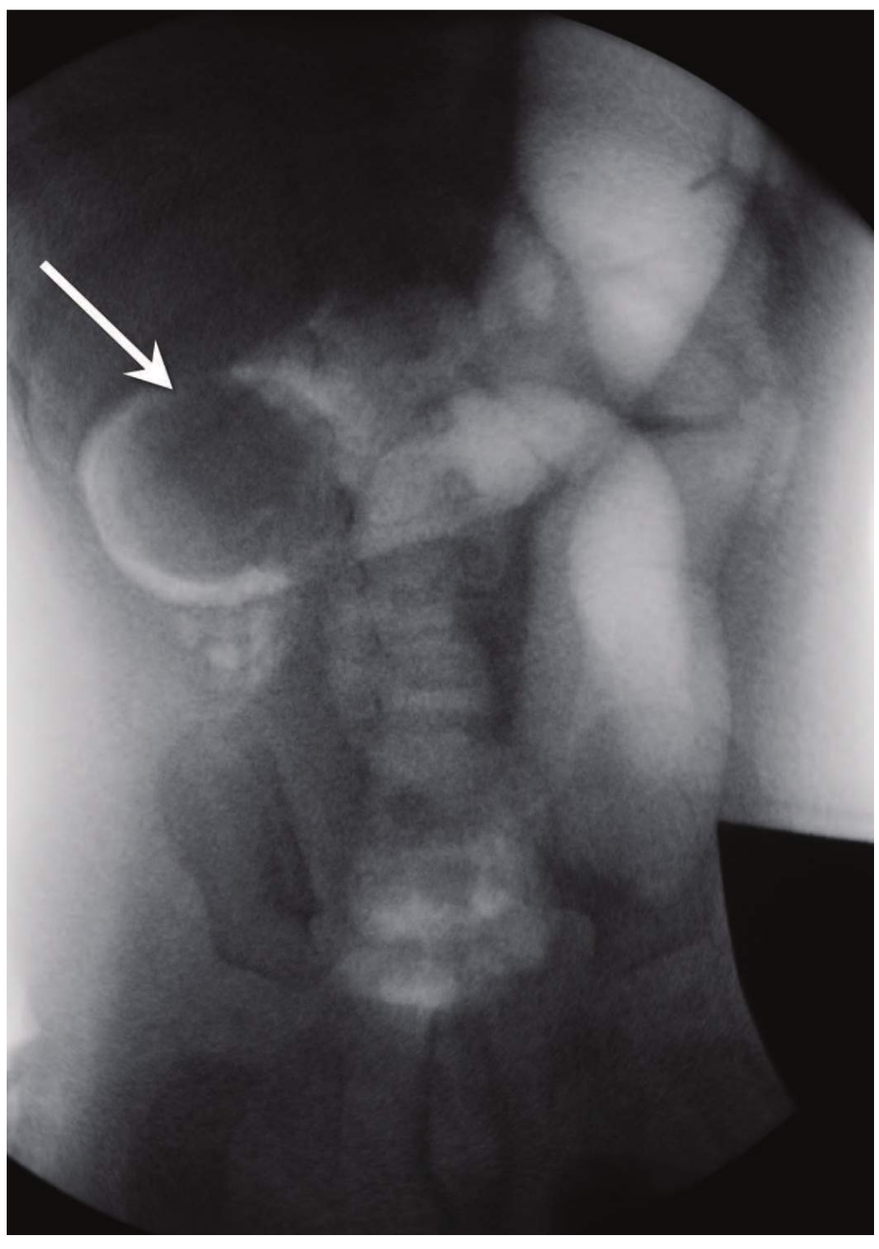


FIGURE 14.106 ■ **Intussusception reduction via air contrast enema part 1.** Air distending the rectum and colon just before reduction. The intussusception mass (arrow) is in the right upper quadrant near the hepatic flexure. (Photo contributor: Alexander Towbin, MD.)

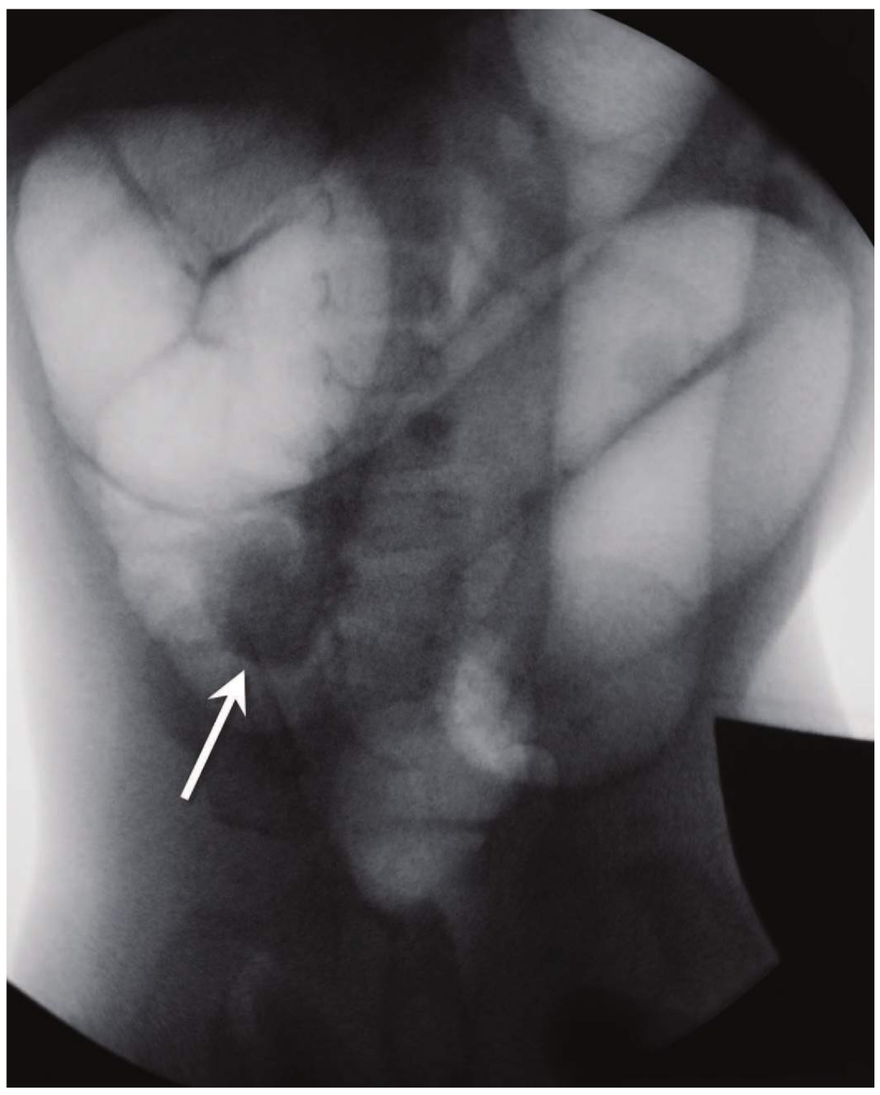


FIGURE 14.107 ■ **Intussusception reduction via air contrast enema part 2.** Reduction of the intussusception (arrow) to the level of the ileocecal valve. (Photo contributor: Alexander Towbin, MD.)

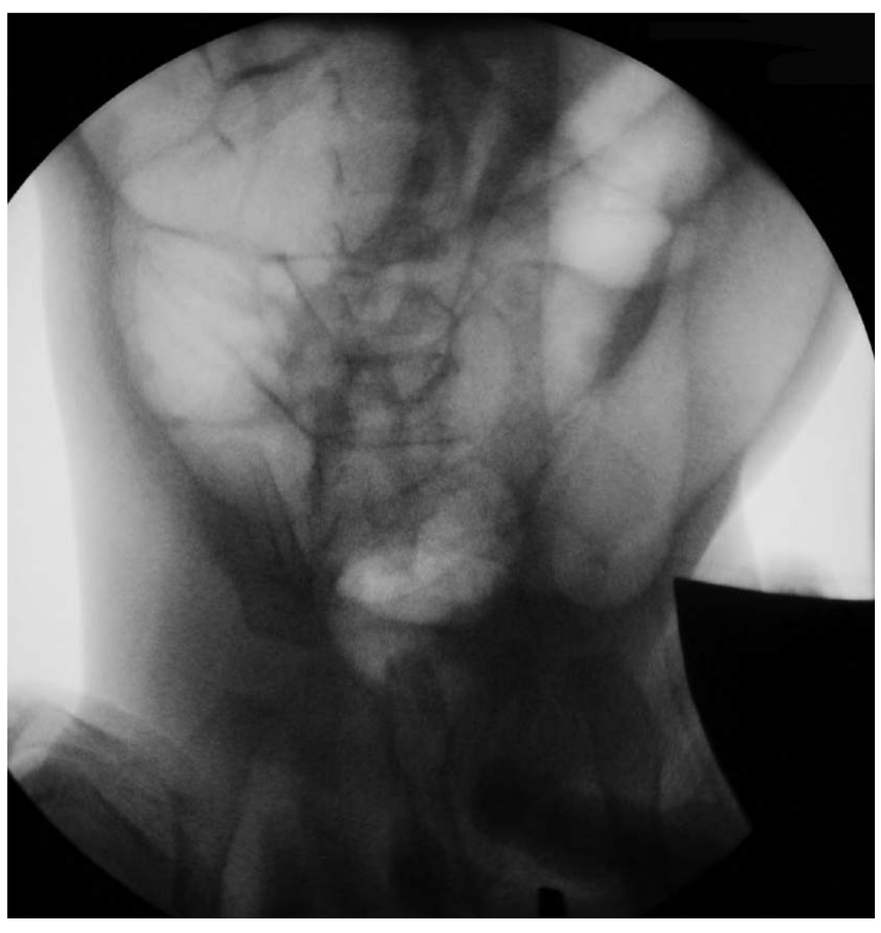


FIGURE 14.108 ■ **Intussusception reduction via air contrast enema part 3.** The intussusception has been reduced. There is free reflux of air into the small bowel. (Photo contributor: Alexander Towbin, MD.)

Clinical Summary

Inguinal hernias are common in childhood with an incidence as high as 5%. Premature infants have an even higher incidence, and boys are approximately 10 times more likely than girls to develop an inguinal hernia. There are two types of inguinal hernias: indirect (common) and direct (rare). Indirect inguinal hernias result from failure of the processus vaginalis to obliterate toward the end of fetal development. With a patent processus vaginalis, the intra-abdominal viscera can protrude through the internal inguinal ring. Indirect inguinal hernias are more common on the right and present as a bulge in the groin by parental history or on physical examination. Maneuvers that increase intra-abdominal pressure, such as crying in an infant or blowing bubbles in an older child, may make the hernia easier to visualize. Associated symptoms such as vomiting, abdominal distention, constipation, blood in the stool, lethargy, or irritability suggest incarceration or strangulation of the hernia. Incarceration is most common in the first year of life. The differential diagnosis includes hydrocele, inguinal lymphadenopathy, testicular torsion, torsion of the appendix testis/epididymis, epididymitis/orchitis, and a retractile testicle.

Management and Disposition

With the history of scrotal swelling but a normal physical examination, refer to a pediatric surgeon for timely evaluation and repair. If a hernia is palpable, ensure that it can be easily reduced manually. Reduction of an incarcerated inguinal

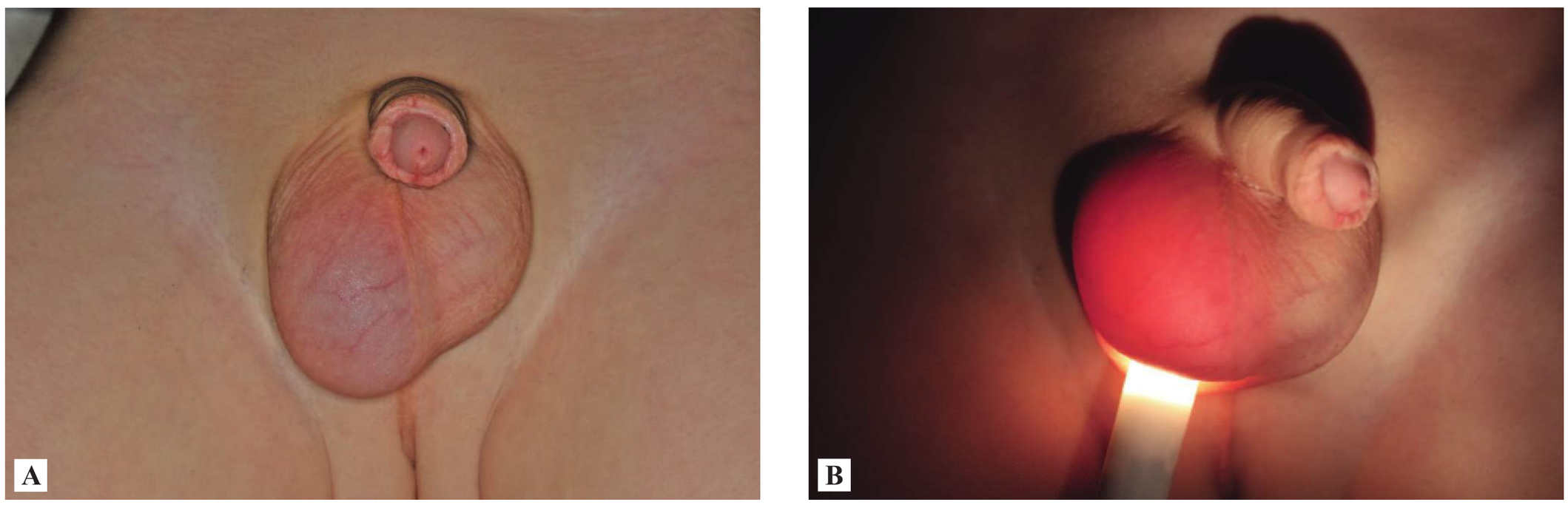


FIGURE 14.109 ■ **Hydrocele.** Painless scrotal swelling (**A**) which transilluminates (**B**). This patient had an inguinal hernia which was repaired electively. (Photo contributor Kevin J. Knoop, MD, MS.)

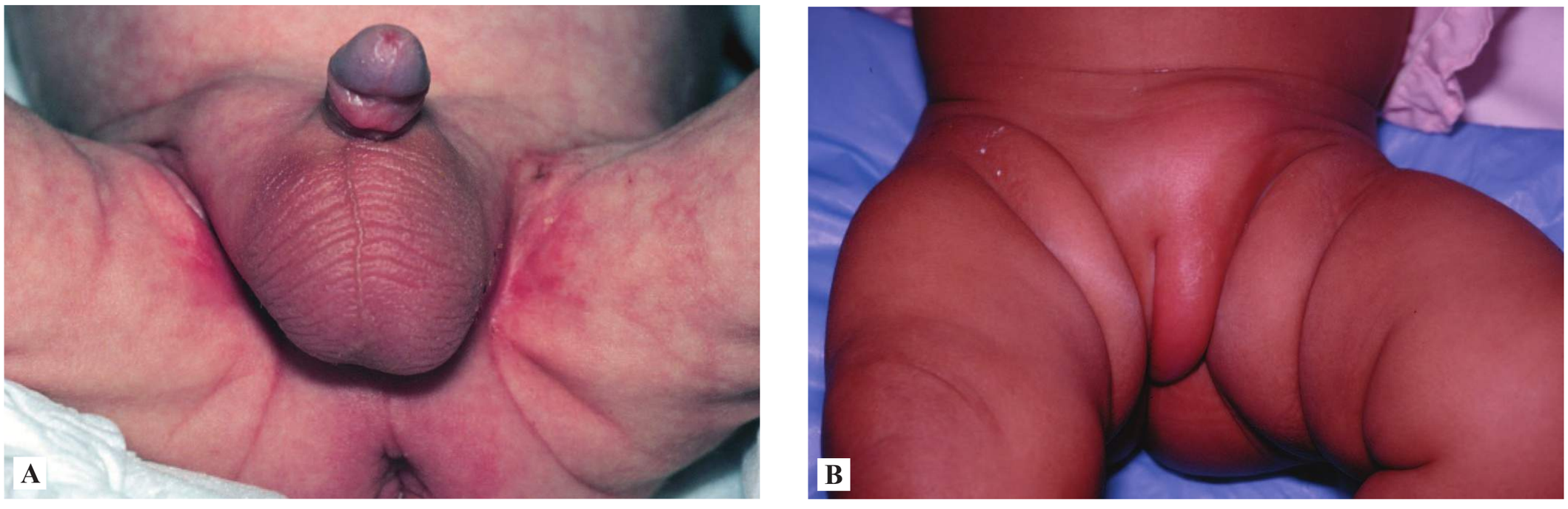


FIGURE 14.110 ■ **Inguinal Hernia.** (**A**)Right inguinal hernia in a male infant (**B**) Left inguinal hernia in a female infant. (Photo contributor: Lawrence B. Stack, MD.)

hernia may be facilitated by adequate pain control and sedation (80% successful). The reduction technique consists of gentle traction inferiorly on the hernia sac with pressure from above to straighten the inguinal canal. There is a high rate of early recurrence of incarceration. Admission to the hospital with repair in 24 to 48 hours after the associated edema has subsided is advisable. Immediate surgical consultation for operative repair is indicated for any incarcerated hernia that cannot be manually reduced.

Pearls

1. Transilluminate the scrotum. Hydroceles will transilluminate, whereas hernias will not.
2. The Trendelenburg position may aid in hernia reduction.
3. Palpate both testicles in the scrotum prior to diagnosing an inguinal hernia.
4. Plain abdominal radiographs are unlikely to be helpful in cases of scrotal swelling where the diagnosis is not apparent. Scrotal ultrasound has a diagnostic accuracy of over 90%.

PINWORM INFECTION (ENTEROBIASIS)

Clinical Summary

Enterobius vermicularis is a threadlike white worm that infects the colon and causes intense nocturnal perianal pruritus when the gravid adult female migrates to deposit eggs at night. Female worms measure 8 to 13 mm in length and can be observed moving about the perianal area at night. On rare occasions, this nematode can lead to vulvovaginitis. The diagnosis can be made by direct visualization of the nematode by the parents or by using a piece of transparent adhesive tape and touching it to the perianal area upon awakening in the morning. This tape is then applied to a glass slide for microscopic examination under low power to look for eggs. The differential diagnosis includes perianal irritation, cellulitis, fissures, hemorrhoids, and contact dermatitis.

Management and Disposition

The treatment of choice is the anthelmintic agent, albendazole. The initial single dose is repeated in 2 weeks to treat secondary Enterobius hatchings. Because of the high frequency of reinfection, families should be treated as a group.

Pearls

1. Reinfection from other infected individuals (daycare cohorts) or autoinfection is necessary to maintain enterobiasis in the individual, since these nematodes usually die after depositing their eggs in the perianal region. Frequent hand washing may reduce chances of infection as transmission occurs by the fecal-oral route.
2. E vermicularis is the most common intestinal nematode in the United States, affecting 5% to 15% of the population. Many infections are asymptomatic.
3. Suspect pinworm infection in children who present with nocturnal restlessness. These patients are often evaluated for urinary tract infection because the scratching of the perineal area is misinterpreted by the parents and treating clinicians as painful urination.

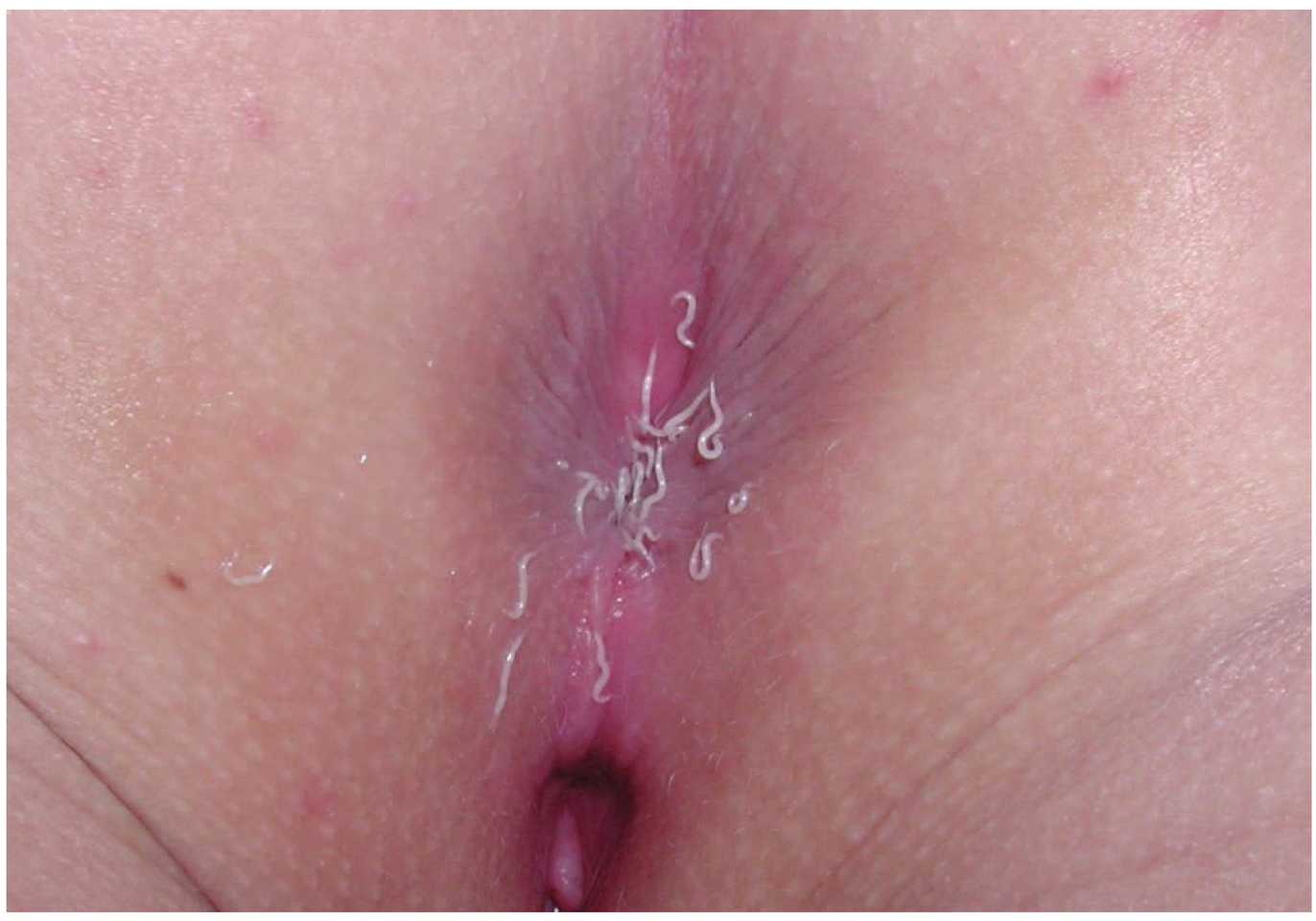


FIGURE 14.111 ■ **Pinworms.** Multiple tiny pearly-white worms are seen at the anus. (Photo contributor: Lawrence E. Heiskell, MD.)

Clinical Summary

There are three varieties of lice specifically parasitic to humans: Pediculus humanus capitis (head louse), which infests the hair and scalp, Pthirus pubis (crab louse), which infests the pubic hair, and Pediculus humanus corporis (body louse). Affected patients may be asymptomatic or may have pruritus. The diagnosis is made via visual examination whereby crawling lice (nymphs and adults) and eggs (nits) are found on hair shafts. The treatment is topical pediculicides (including permethrin, malathion, benzyl alcohol, spinosad, and topical ivermectin) and wet combing to remove lice and nits. Lindane has an association with neurotoxicity, and its use should be avoided in children. Of note, nits may persist for several weeks following therapy. The differential diagnosis includes pseudonits (2-7 mm hair casts), psoriasis, cutaneous fungal infections, scabies, seborrheic dermatitis, and dandruff.

Management and Disposition

The diagnosis is clinical. Patients should be treated with topical pediculicides and educated on signs of resolution. Treatment failure is associated with noncompliance, reinfestation, and drug resistance. Close contacts and those who share bedding should be treated prophylactically. Head lice generally do not survive for more than 2 days when off of a person. The nits die within 1 week if the temperature close to the scalp is not maintained. Clothing and items (eg, pillows, bedding, or stuffed animals) that cannot be washed should be dry-cleaned or sealed for 2 weeks in a plastic bag.

Pearls

1. Children with head lice do not necessarily need to be excluded from school as many have had lice for several weeks and been attending school prior to diagnosis.
2. Combs and brushes can be disinfested by soaking in hot water (130°F or greater) for 5 to 10 minutes.
3. Though shaving hair is anecdotally an effective treatment, it can have adverse psychological effects.

FIGURE 14.112 ■ **Nits.** Several small white cylindrical nits (louse eggs) are seen clinging to dark hair. (Photo contributor: Larry B. Mellick, MD.)

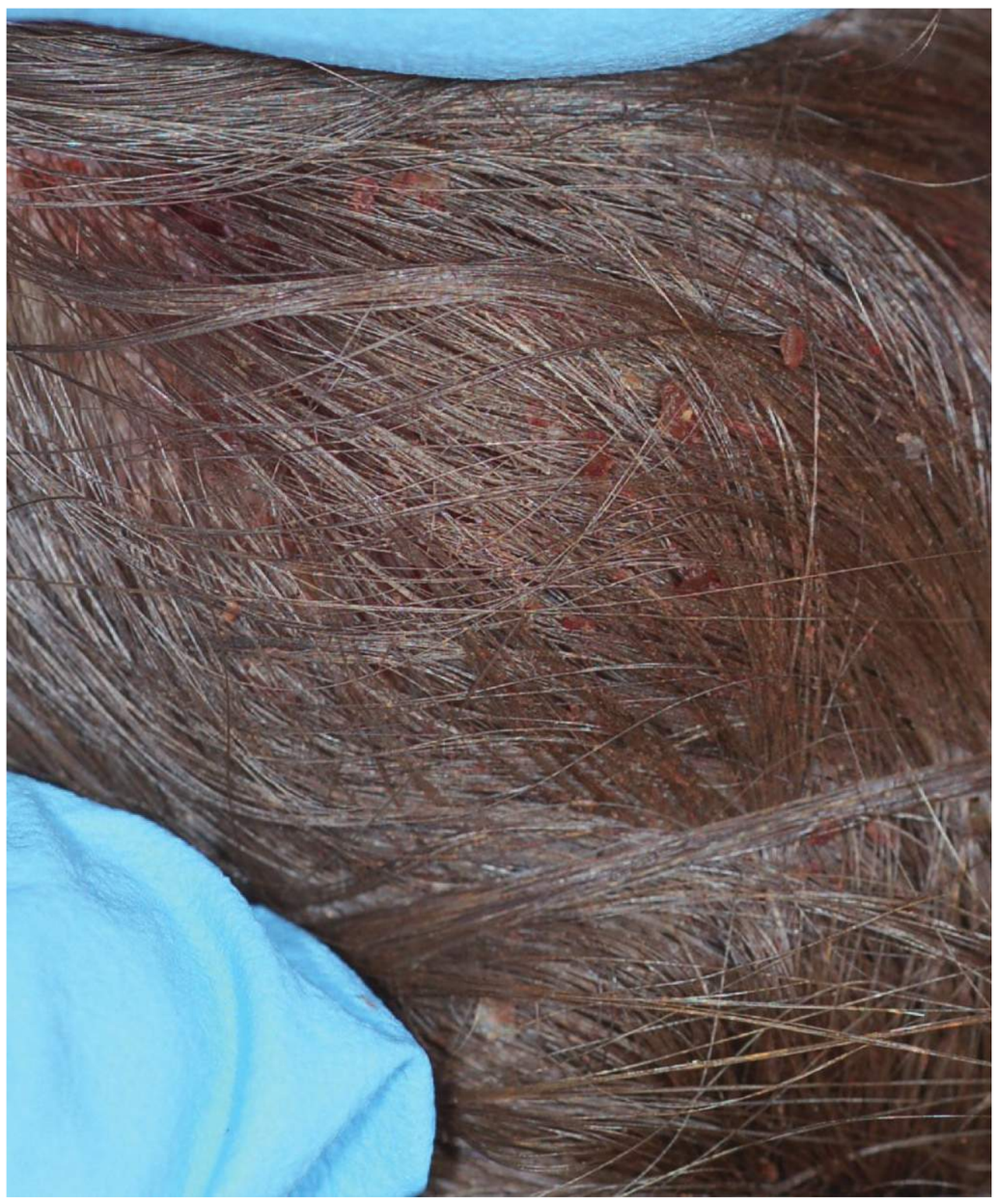


FIGURE 14.113 ■ **Lice Infestation.** Multiple lice are seen in the hair of this patient. (Photo contributor: David Effron, MD.)

TRICHOTILLOMANIA

Clinical Summary

Trichotillomania is an impulse control disorder, which results in compulsive hair pulling causing noticeable hair loss. Hair is repeatedly twisted around the finger then pulled out, or rubbed until it breaks off. There is an irregular-angled border with short hair of different lengths in the affected area. The eyebrows and eyelashes may also be targeted. True alopecia (complete loss of hair) is not present. Young children, adolescents, and women more commonly are affected. The course is chronic with frequent remissions. It is highly distressing to most patients, causing significant psychological duress.

Management and Disposition

Ask if the patient manipulates the hair to confirm the behavior pattern. Perform a Wood lamp exam to rule out noninflammatory tinea capitis. Refer to primary care for initial uncomplicated presentation. Consider referral to dermatology (for biopsy) or mental health in questionable or refractory cases. Additional information for sufferers is at the Trichotillomania Learning Center at www.trich.org.

Pearls

1. Hair density is greatly reduced at the affected site, but it is never bald.
2. There may be an associated mood or anxiety disorder.

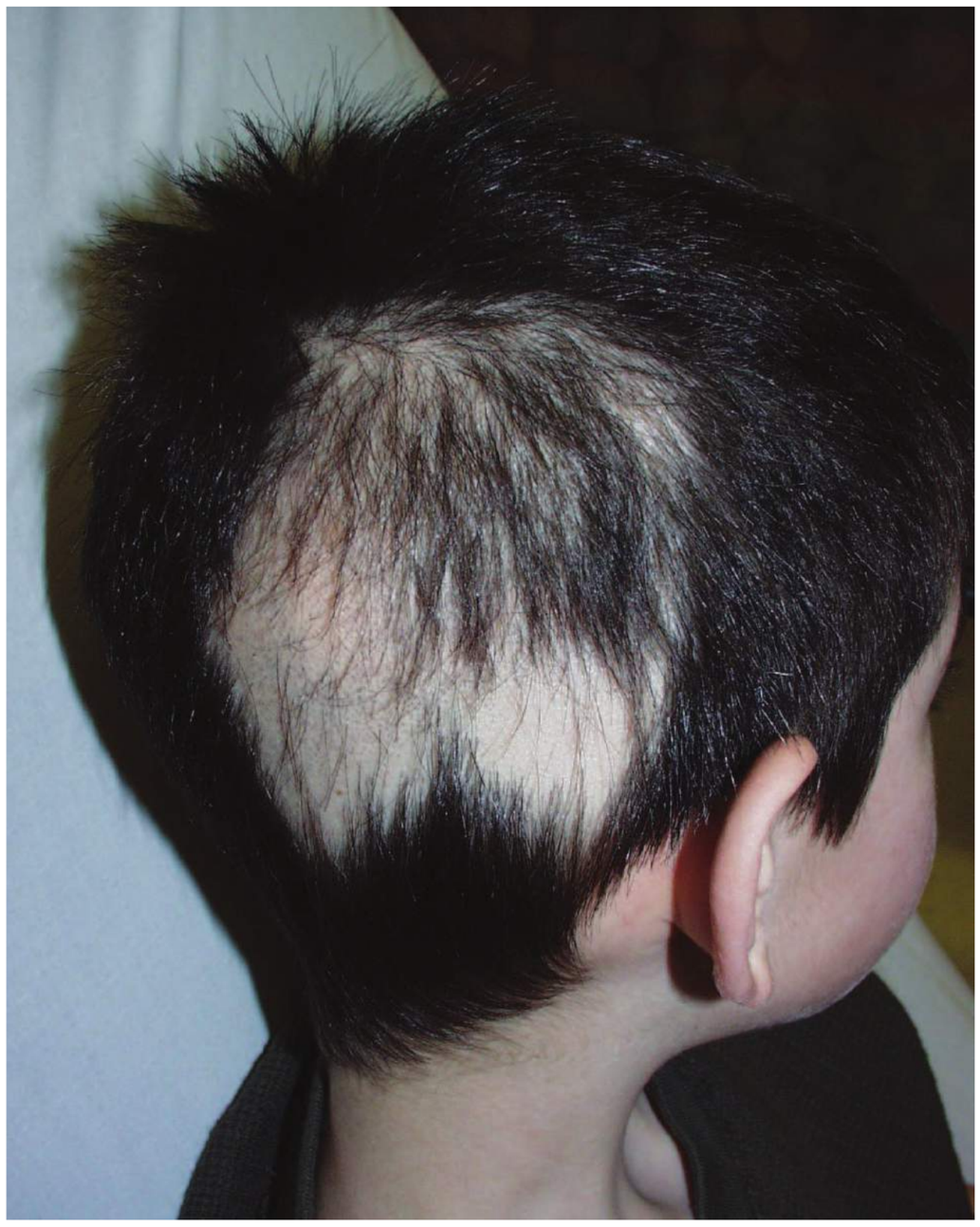


FIGURE 14.114 ■ **Trichotillomania.** Patchy hair loss seen in a young child with a history of self-induced hair pulling. Short hair of different lengths are randomly distributed. (Photo contributor: Timothy D. McGuirk, DO.)

Clinical Summary

Oculogyric crisis (OGC) is the most common of the ocular dystonic reactions. It includes blepharospasm, periorbital twitches, and protracted staring episodes. It usually occurs as a side effect of neuroleptic drug treatment. OGC represents approximately 5% of the dystonic reactions. The onset of a crisis may be paroxysmal or stuttering over several hours. Initial symptoms include restlessness, agitation, malaise, or a fixed stare followed by the more characteristically described maximal sustained upward deviation of both eyes. The eyes may also converge, deviate upward and laterally, or deviate downward. The most frequently reported associated findings are backward and lateral flexion of the neck, widely opened mouth, tongue protrusion, and ocular pain. A wave of exhaustion follows some episodes. Other features noted during attacks include eye blinking, lacrimation, pupil dilation, drooling, facial flushing, vertigo, anxiety, and agitation. Several medications have been associated with the occurrence of OGC: cetirizine, neuroleptics, amantadine, benzodiazepines, carbamazepine, chloroquine, levodopa, lithium, metoclopramide, and nifedipine. Careful history and physical examination should exclude the possibility of focal seizures, meningitis, encephalitis, head injury, conversion reaction, Parinaud syndrome, and other types of movement disorders.

Management and Disposition

Treatment in the acute phase in children involves reassurance, discontinuation of the causative agent, and diphenhydramine at a dosage of 1.25 mg/kg initially; this may be repeated if there is no effect. For moderate to severe cases, give the initial dose parenterally. Occasionally, doses up to 5 mg/kg are required. The treatment with diphenhydramine should be continued every 6 hours for 1 to 2 days. Benztropine can also be used; however, it is not approved for children below 3 years of age. Close monitoring is important during treatment as dystonic reactions are occasionally accompanied by fluctuations in blood pressure and arrhythmias.

Pearls

1. The abrupt termination of the symptoms at the conclusion of the crisis or after the use of diphenhydramine is diagnostic and most striking.
2. In infants presenting with "seizures," unusual behavior, eye deviation, and a history of reflux treated with metoclopramide, the possibility of OGC should be considered. Although the overall incidence of extrapyramidal effects associated with metoclopramide is 0.2%, pediatric and geriatric patients are affected more commonly, with an incidence as high as 10%. These side effects usually occur within a few days of initiation of the medication and are more common at higher doses.

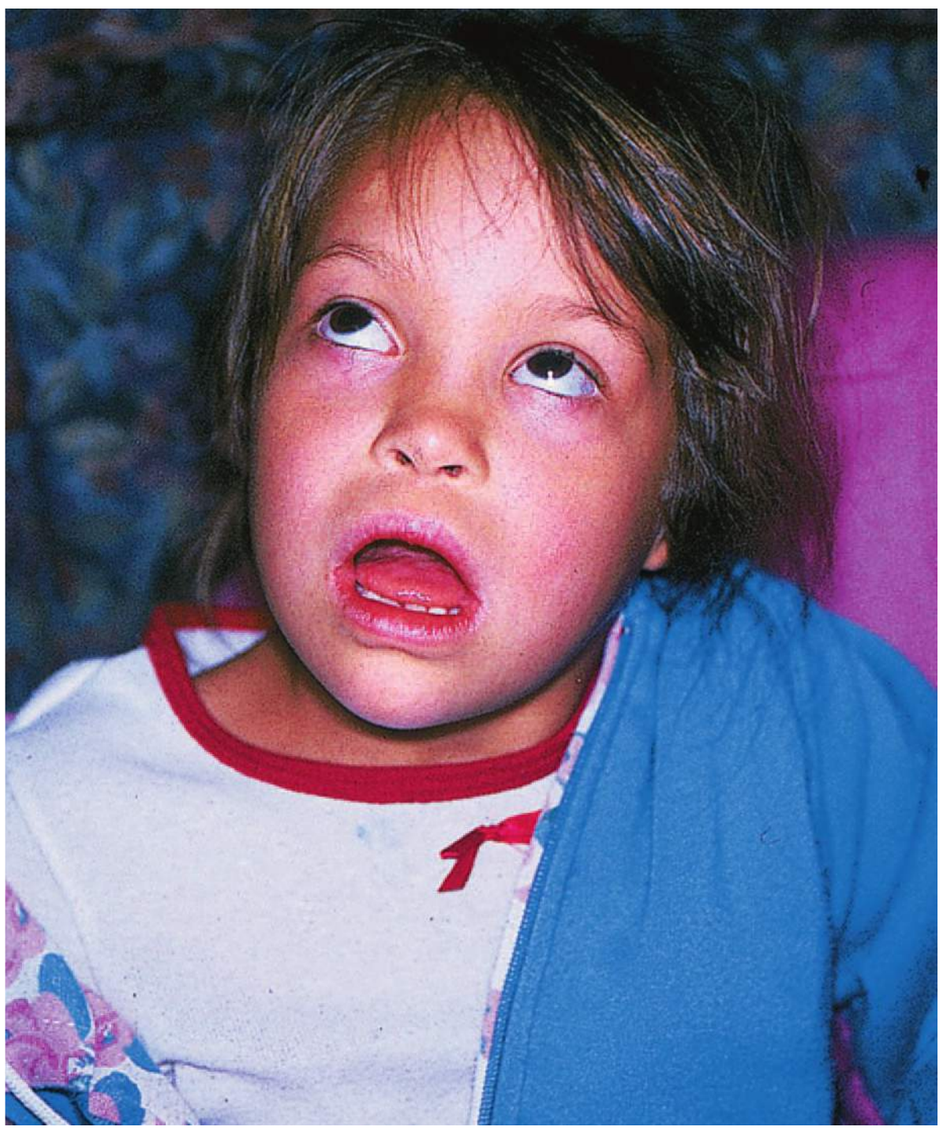


FIGURE 14.115 ■ **Oculogyric Crisis.** This 6-year-old boy developed extrapyramidal symptoms, including opisthotonos and oculogyric crisis, after his dosage of risperidone was increased. Benadryl 12.5 mg PO given at home resolved the opisthotonos. Persistent oculogyric crisis (upward gaze deviation) and hypertonia resolved completely after Benadryl 25 mg IM. (Photo contributor: Mark Ralston, MD.)

SETTING-SUN PHENOMENON (SUNDOWNING)

Clinical Summary

The setting-sun phenomenon also known as "sundowning" is a concerning sign that may represent a pathologic increase in intracranial pressure in the young infant. The clinical presentation consists of an upward-gaze paresis with eyes that appear to be driven downward. There is usually sclera showing between the upper eyelid and the iris. Retraction of the upper eyelids, sometimes accompanied by raising of the brow, may be seen. Although this sign is commonly seen in children with obstructive hydrocephalus, it may also be seen as a result of intracranial hypertension of other causes (trauma or ventriculoperitoneal shunt dysfunction) and occasionally in normal infants. It is thought to result from compression of the periaqueductal structures.

The phenomena could be an important sign in early detection of elevated intracranial pressure, appearing sooner than enlarged head circumference, full fontanelle, separation of sutures, irritability, or vomiting.

Management and Disposition

Suspect increased intracranial pressure and obtain neuroimaging urgently. Consult neurosurgery for guidance in initial management and for definitive treatment in the operating room.

Pearls

1. This sign is a valuable cue for obtaining prompt neuroimaging and urgent surgical intervention.
2. When persistent, this sign is a frequent marker of elevated intracranial pressure, appearing in 40% of children with hydrocephalus (of any cause) and in 13% of patients with shunt dysfunction.
3. Ask parents to share photographs of their child taken since birth to assess for changes to head size and for the presence of sundowning.

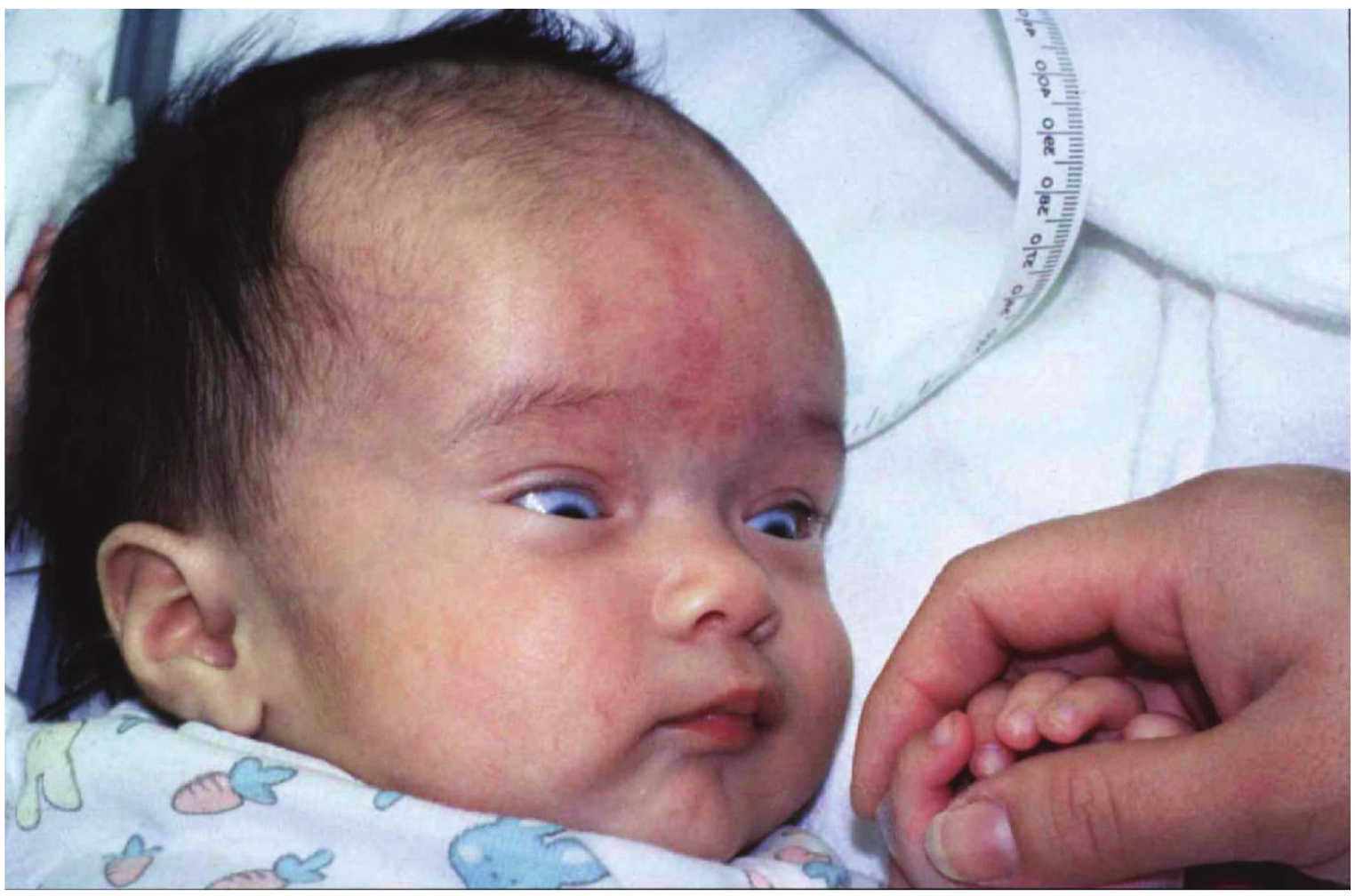


FIGURE 14.116 ■ **Setting-Sun Phenomenon (Sundowning).** The eyes appear driven downward in this infant with hydrocephalus. (Photo contributor: Stephen W. Corbett, MD.)

Clinical Summary

Kawasaki disease (KD), also known as mucocutaneous lymph node syndrome, is an acute self-limited medium to small vessel vasculitis of unknown etiology occurring most commonly in young children with a peak incidence between 1 and 2 years of age. The classic diagnosis is made clinically and is based on the presence of more than 5 days of fever and at least four of the following five principal clinical features:

1. Generalized polymorphous rash usually appearing within 5 days of the onset of fever and most commonly is a nonspecific, diffuse maculopapular eruption. Early desquamation may occur in the perineal region, especially in infants.
2. Bilateral bulbar conjunctivitis sparing the limbus and nonexudative.
3. Changes to the lips and oral mucosa including dry, red, cracked lips, strawberry tongue, and diffuse erythema of the oropharyngeal mucosa.
4. Peripheral extremity changes: acute erythema and induration of the palms and soles followed in 2 to 3 weeks by desquamation of the fingers and toes beginning in the periungual region. One to two months after the onset of the fever, deep transverse grooves across the nails (Beau lines) may appear.
5. Cervical lymphadenopathy that is usually unilateral, confined to the anterior cervical triangle, and is greater than or equal to 1.5 cm in diameter.

The fever of KD is typically spiking and unremitting with peak temperatures often greater than 40°C (104°F). Incomplete (atypical) KD, which is more common in infants, does not meet all of the classic criteria but should be suspected in any child with unexplained fever for more than 7 days or unexplained fever for more than 5 days with less than three KD-associated clinical criteria. Laboratory evaluation is necessary to support the diagnosis of incomplete KD and includes elevated ESR (≥ 40 mm/h) or CRP (≥ 3 mg/dL), and ≥3 supplemental laboratory criteria (albumin ≤ 3 g/dL, anemia for age, elevation of alanine aminotransferase, platelet count after 7 days ≥ 450,000/mm^3, white blood cell count ≥ 15,000/mm^3, and urine ≥ 10 white blood cells/high-power field). An abnormal echocardiogram may also aid in the diagnosis of incomplete KD.

Coronary artery aneurysms or ectasias are the most important complication of KD and develop in 15% to 25% of untreated children and may lead to ischemic heart disease, myocardial infarction, or sudden death. KD may also cause aseptic meningitis, urethritis, myocarditis, arthritis, liver

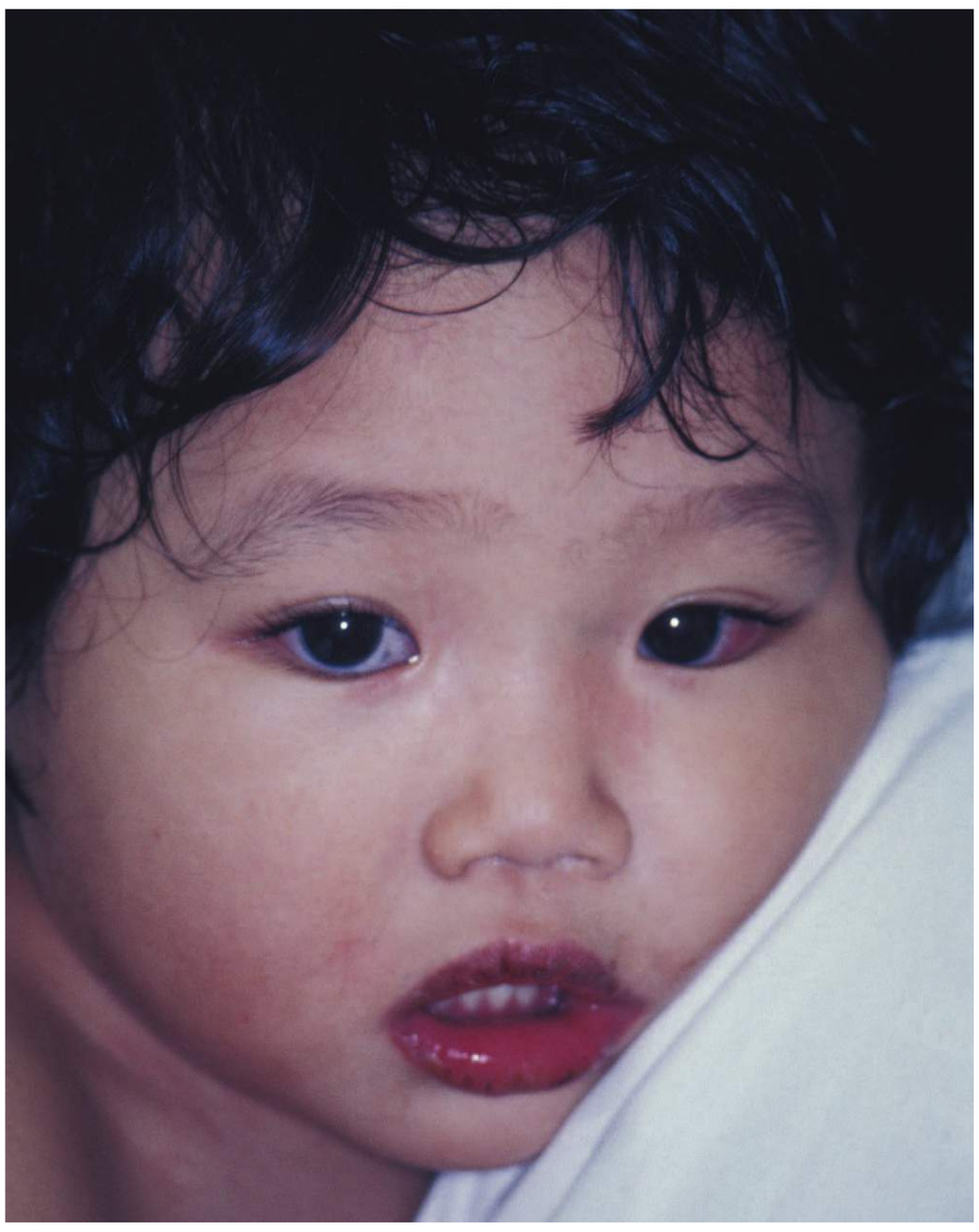


FIGURE 14.117 ■ **Kawasaki Disease.** Irritability in an infant with Kawasaki disease. Note also the conjunctivitis, and red, cracked lips. (Photo contributor: Tomisaku Kawasaki, MD.)

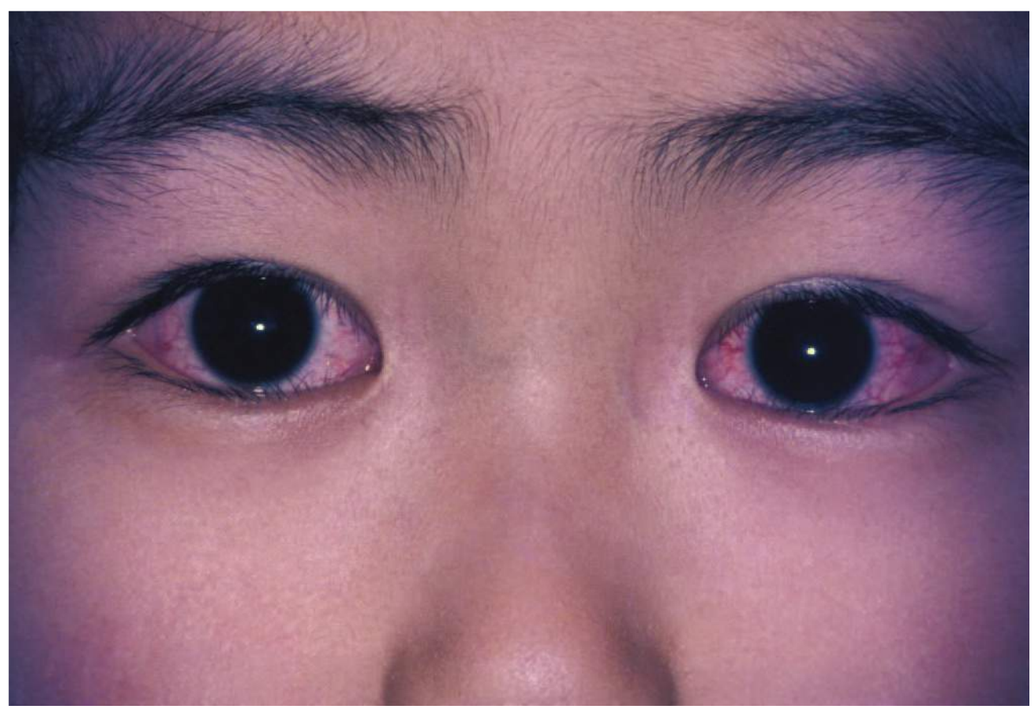


FIGURE 14.118 ■ **Conjunctivitis.** Note the intense redness of the bulbar conjunctiva seen in this patient with Kawasaki disease. (Photo contributor: Tomisaku Kawasaki, MD.)

dysfunction, abdominal pain, and gallbladder hydrops. The differential diagnosis of KD includes scarlet fever, staphylococcal scalded skin syndrome, toxic shock syndrome, viral infections (measles, adenovirus, enterovirus, Epstein-Barr virus), bacterial cervical lymphadenitis, drug hypersensitivity reaction, Stevens-Johnson syndrome, and acrodynia.

Management and Disposition

If suspected, consultation with a cardiologist or other specialist in KD is highly recommended for confirmation of diagnosis as well as treatment. A baseline echocardiogram should be obtained to document the status of the coronary arteries. Treatment includes hospitalization, administration of IVIG 2 g/kg infused over 8 to 10 hours, and initiation of high-dose aspirin 80 to 100 mg/kg/day in four divided doses. Approximately 90% of patients will defervesce with this regimen. Failure to respond is usually defined as persistent or recurrent fever greater than or equal to 36 hours after completion of the initial IVIG infusion. Retreatment with IVIG 2 g/kg is recommended. With IVIG treatment in the acute phase of the disease, the risk of coronary artery abnormalities is decreased to less than 5%.

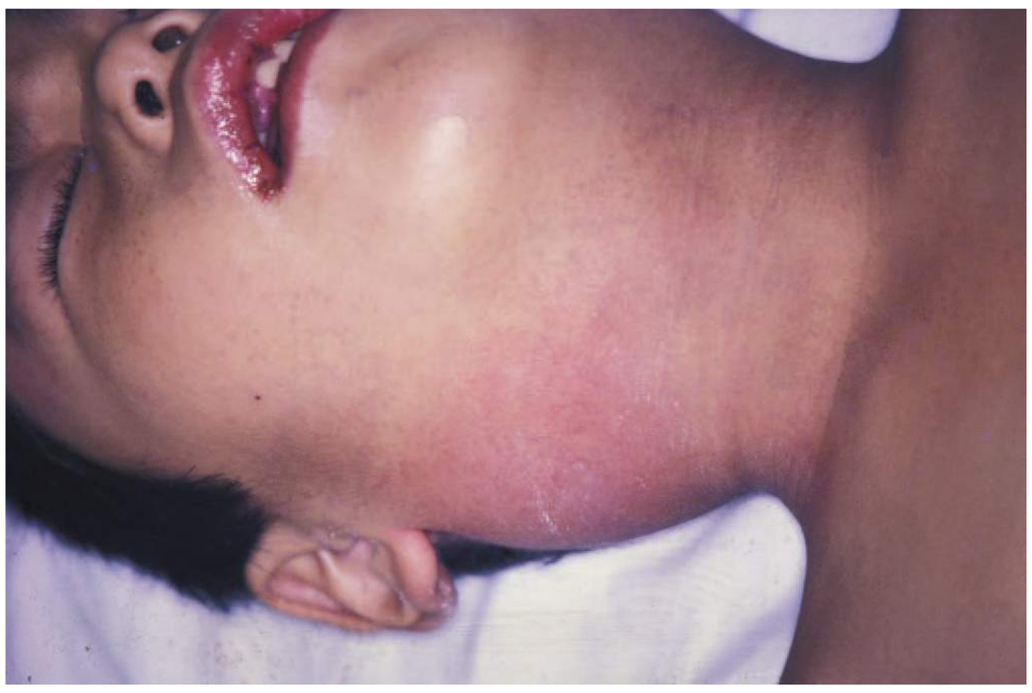


FIGURE 14.119 ■ **Lymphadenopathy.** Visible cervical lymphadenopathy is seen in this child with Kawasaki disease. (Photo contributor: Tomisaku Kawasaki, MD.)

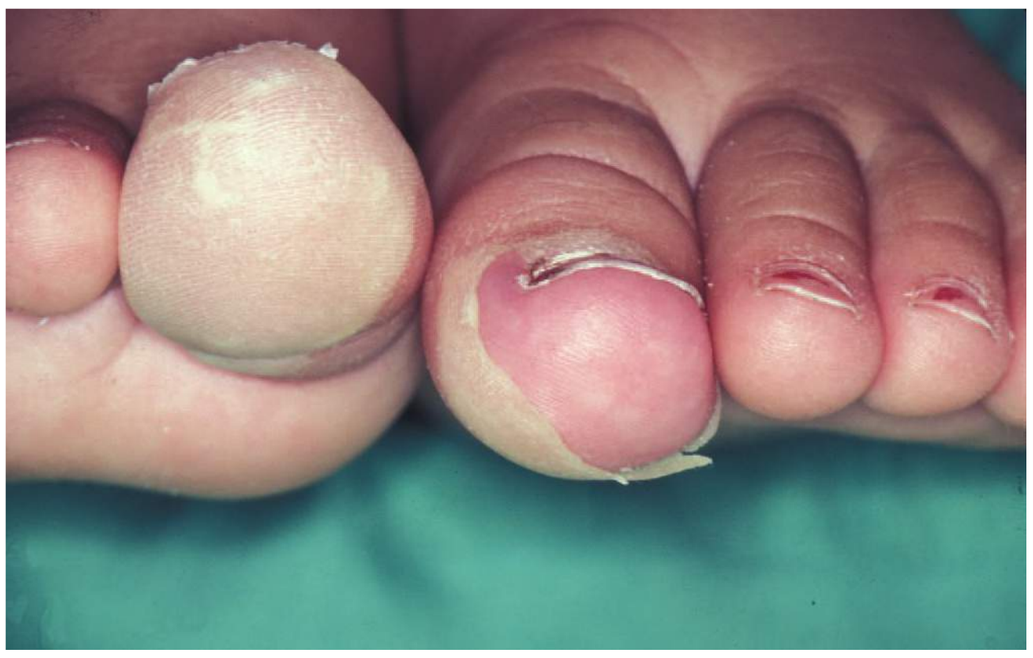


FIGURE 14.121 ■ **Periungual Desquamation.** This finding typically begins 2 to 3 weeks after the onset of Kawasaki disease, in contrast to perineal desquamation that occurs during the early course of the disease in infants. (Photo contributor: Tomisaku Kawasaki, MD.)

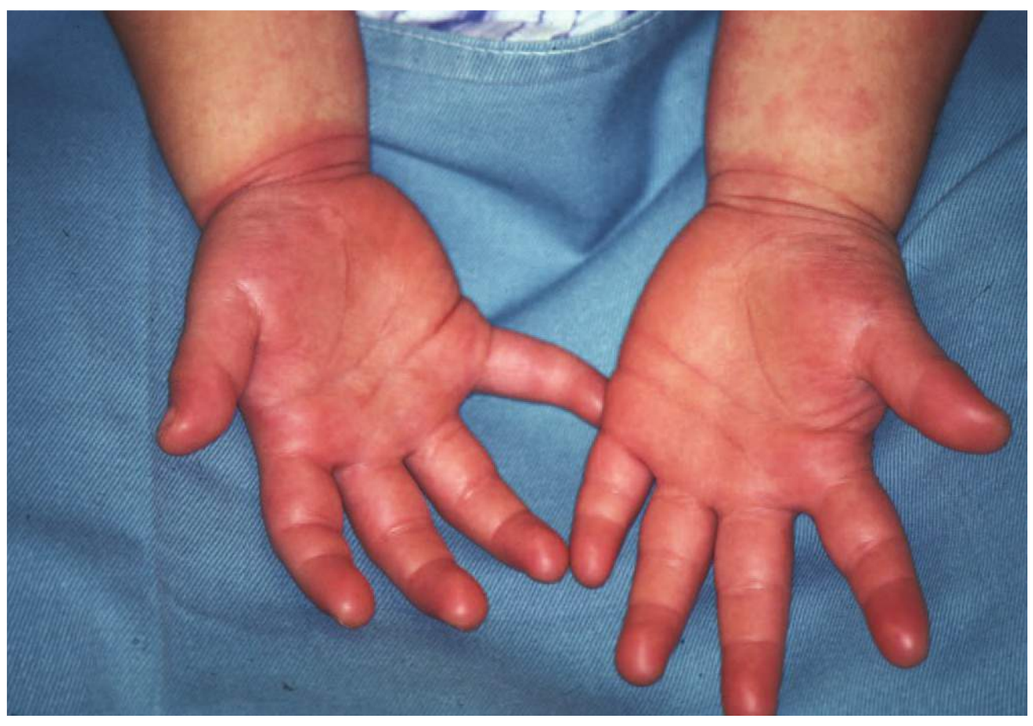


FIGURE 14.120 ■ **Extremity Findings.** Red swollen hands are present in this patient with Kawasaki disease. (Photo contributor: Tomisaku Kawasaki, MD.)

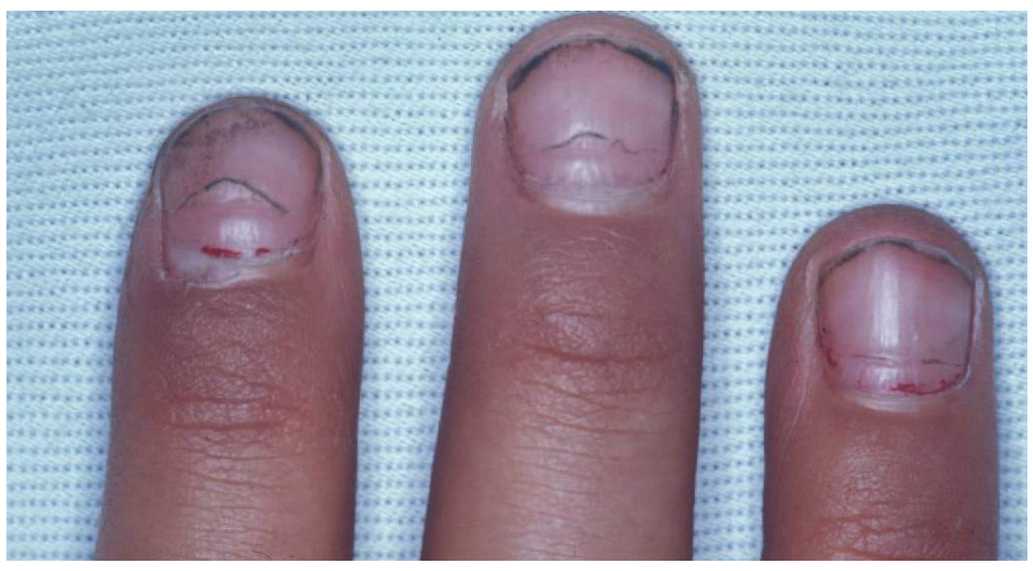


FIGURE 14.122 ■ **Beau Lines.** Deep transverse grooves across the nails may appear 1 to 2 months after the onset of fever. (Photo contributor: Tomisaku Kawasaki, MD.)

Pearls

1. The diagnosis of typical KD is clinical, but laboratory studies may be helpful in cases of suspected incomplete KD.
2. Children with KD are commonly irritable and difficult to console.
3. KD is the leading cause of acquired heart disease in children in the United States. Echocardiography should be considered in any infant less than 6 months of age with fever of greater than or equal to 7 days duration, laboratory evidence of inflammation, and no other explanation for the febrile illness.
4. Even when treated with high-dose IVIG regimens within the first 10 days of illness, approximately 5% of children with KD develop at least transient coronary artery dilation.
5. Vaccination against influenza should be provided to all patients receiving aspirin therapy to avoid Reye syndrome.

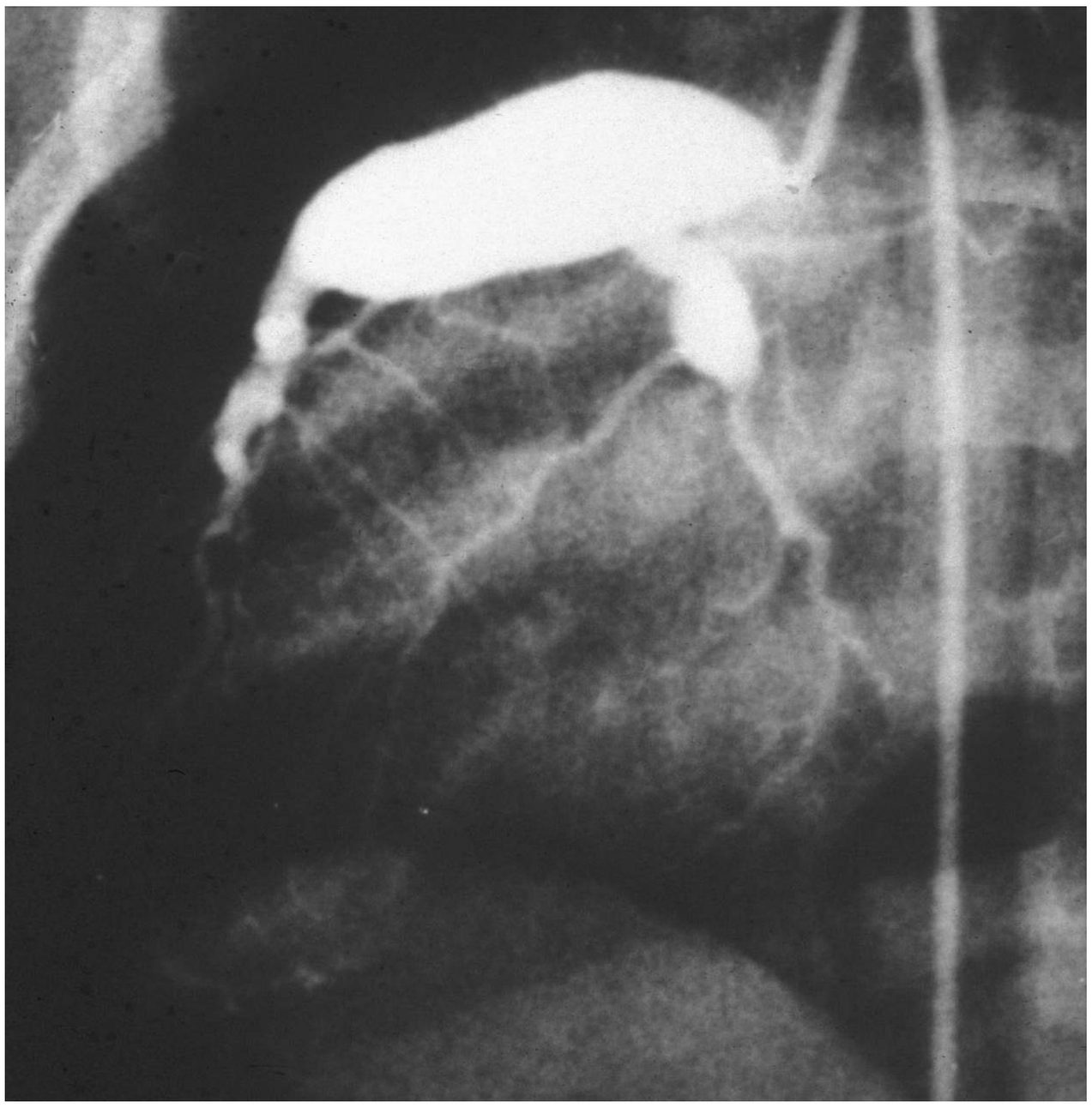


FIGURE 14.123 ■ **Coronary Artery Aneurysms.** A large coronary artery aneurysm is seen in this patient with Kawasaki disease. (Photo contributor: Tomisaku Kawasaki, MD.)