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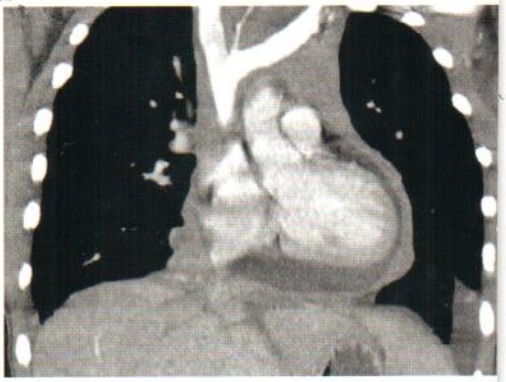

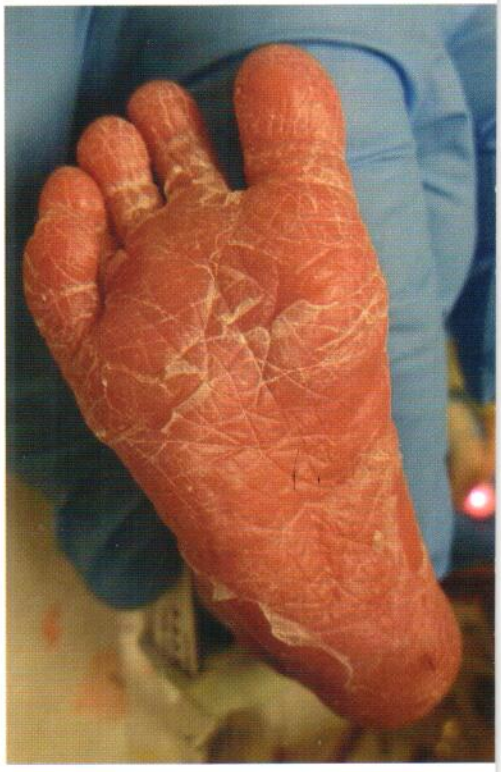

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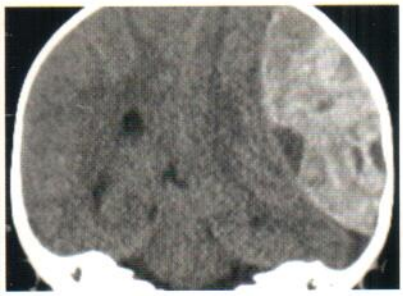

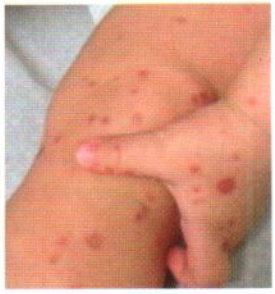

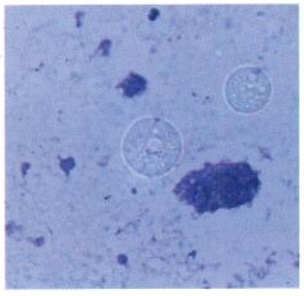

Also Inside

- A Teen Whose Bruises Won't Fade
- Can You Identify a Girl's Circular Lesions?
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A Girl With Concentric, Target-Shaped Lesions on Her Hands, Arms, and Legs

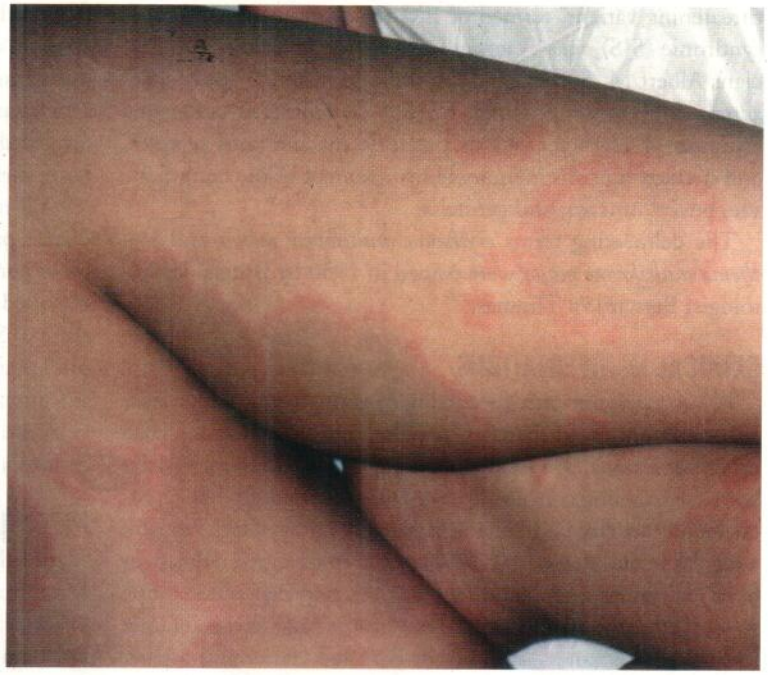
Germaine L. Defendi, MD, MS

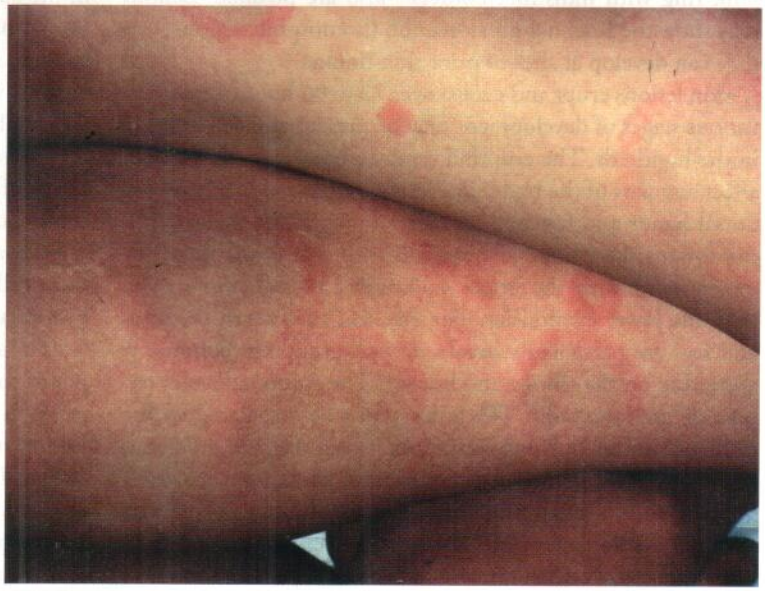
A 13-year-old girl presented to the pediatric urgent care clinic with concerns of headache, fever, pharyngitis, cough, and myalgia. She reported having had a fever (38.0°C) and a dry, hacking cough for the past 7 days. A mildly pruritic erythematous skin eruption had developed over the last 3 days, about which she was most concerned. She had no complaints of pain. She had no past history or family history of a similar skin eruption. Her past medical history was unremarkable. She had been taking acetaminophen to treat her symptoms. Her immunizations were up to date.

The patient appeared nontoxic and was well hydrated. She was in no respiratory distress. An intermittent cough was noted. Her oropharynx was slightly erythematous without purulence. Intermittent rales were heard in the lower lung fields on chest auscultation. A chest radiograph revealed peribronchial interstitial infiltrates.

A circular erythematous skin eruption, polymorphous in nature, was noted. Skin findings revealed target-like lesions on her hands, arms, and legs (**Figure**). Most lesions had well-defined borders and appeared as concentric rings of different skin colors—notably, dark centers surrounded by lighter rims and encircled by outermost erythema. Erythematous macules, papules, and wheals also were present. Some macules appeared papular and others appeared plaque-like within the center. Concentric rings of erythema were noted.

Sputum testing later confirmed the underlying cause of her cutaneous findings.





What explains the girl's skin lesions and other symptoms?

Answer: Erythema multiforme triggered by infection

Erythema multiforme (EM), also termed EM minor, is a relatively common skin disorder caused by a delayed hypersensitivity reaction. This dermatologic eruption often is triggered by infection or specific medications.[1,2]

EM was first described in the 1860s by Austrian dermatologist Ferdinand von Hebra as an acute, self-limited condition with characteristic erythematous papules and plaques.[3,4] A life-threatening variant, termed of EM major or Stevens-Johnson syndrome (SJS), was reported in 1922 by American pediatricians Albert M. Stevens and Frank C. Johnson.[5,6] SJS is characterized by prolonged high fever, a disseminated cutaneous eruption of erythematous or purpuric macules and plaques, and marked mucous membrane involvement of the conjunctivae, buccal mucosa, and genitalia.

The delineating terms *erythema multiforme minor* and *erythema multiforme major* were coined in 1950 by British dermatologist Bernard A. Thomas.[7,8]

CLINICAL MANIFESTATIONS

EM is a polymorphic (occurring in many forms, hence *multiforme*) eruption of macules, papules, and target lesions (also called iris lesions). Iris lesions appear as central bullae or vesicles with surrounding concentric erythema. These target-like circular lesions are fixed and characteristically have a sharp margin, a regular round shape, and 3 concentric color zones: a dusky or dark red center; a surrounding pale pink ring that is raised due to local edema; and a bright red outermost ring. Atypical target lesions with 2 zones—a dusky or dark red center and a paler pink ring with indistinct borders—also are possible. Lesions may show the Koebner phenomenon (isomorphic response), as they can develop at sites of prior skin trauma.

Skin lesions erupt and evolve over 72 to 96 hours. Lesions of various stages of development may be present and can number in the hundreds. The eruption tends to be symmetric and can affect any part of the body. The lesions appear suddenly on the dorsal hands and feet and spread centrally along the extremities toward the trunk. The body distribution favors the distal extremities,[9] with the upper extremities affected more often than the lower extremities. The extensor surfaces of the arms and legs are particularly involved, with groupings commonly appearing on the elbows and knees.[10] The palms and soles frequently are involved.[11] The lesions may be associated with a mild itching or burning sensation. There may be mild mucous membrane involvement, or none at all. In approximately 50% of cases, a small number of mucosal lesions (5 to 10 on average) develop a few days after skin lesions appear and most often affect the buccal mucosa and/or the lips.[11]

This acute and self-limiting eruption usually resolves without complications. Recurrence is possible.[12]

EPIDEMIOLOGY, ETIOLOGY, PATHOPHYSIOLOGY

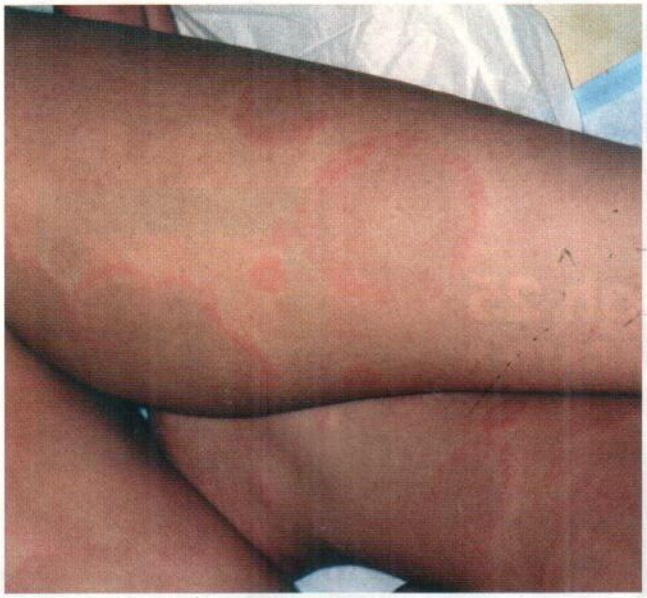
EM is diagnosed more often in boys and men than in girls and women, with a male to female ratio of 3 to 2. The condition affects all ages but is diagnosed more frequently in persons from 20 to 40 years old.[13,14] Approximately 20% of EM cases are diagnosed in children and adolescents.[15] There is no known racial or ethnic predilection.

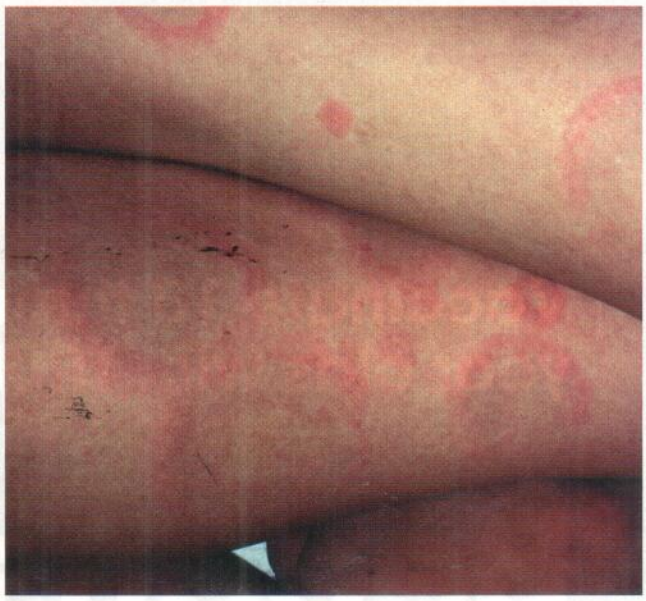
EM initially was thought to be part of a clinical spectrum of disease that included EM minor, EM major (SJS), and toxic epidermal necrolysis (TEN). Within this spectrum, EM minor was considered the mildest form and TEN the most severe.[16] In 1993, a useful clinical classification for EM, SJS, and TEN was proposed based on the skin lesion pattern and the amount of body surface area (BSA) that had detached from the epidermis (eg, blisters, denuded areas, erosions) at the worst stage of the disease process.[17] With little or no mucous membrane involvement and less than 10% BSA epidermal detachment, EM was classified as a distinct condition separate from the clinical spectrum of SJS and TEN.[9,17] In more than 50% of cases of EM, an underlying trigger is not identified, and the cause is unknown.[1] Infection (viral, bacterial, and fungal), medications, and vaccines are known triggers of EM. Infectious diseases and medications are the more common triggers in children.[10]

INFECTIOUS TRIGGERS

Infection is the trigger in approximately 90% of EM cases. More than 50% of these cases are triggered by herpes simplex virus (HSV) infection, with the culprit being HSV-1 more often than HSV-2.[9] In one study of the HSV genotypes found in the cutaneous lesions of patients with HSV-associated EM, 66.7% of cases were related to HSV-1, 27.8% to HSV-2, and 5.6% to coinfection with HSV-1 and HSV-2.[18] However, most persons with HSV infection do not develop EM. Those who develop HSV-associated EM may have clinically apparent HSV, may have an HSV reactivation, or may not have an apparent HSV infection at the time.[19,20] The HLA-DQw3 antigen type is more often found in patients with HSV-associated EM.

HSV-associated EM is a delayed-type hypersensitivity reaction (cell-mediated or type IV hypersensitivity), characterized by a delayed response of 48 to 72 hours after antigen exposure. This reaction occurs with the transport of HSV DNA fragments to distant skin sites by blood mononuclear cells. HSV genes in these viral fragments are expressed on keratinocytes, leading to the recruitment of HSV-specific $CD4^+$ T_H1 cells (helper T cells involved in cell-mediated immunity). $CD4^+$ cells respond to viral antigens by producing interferon-γ, initiating an inflammatory cascade.[15,21] HSV-associated EM appears 3 to 14 days after the viral infection.





Mycoplasma pneumoniae is the second most common trigger for this delayed hypersensitivity reaction and often is the etiology of EM in children.[9] Our patient's clinical history and presentation strongly suggested that her EM trigger was *M pneumoniae* infection. Results of polymerase chain reaction (PCR) testing of a sputum sample were positive for *M pneumoniae*, confirming it as the infectious trigger. PCR testing is the method of choice for the rapid direct detection of *M pneumoniae*, especially if the test is performed within 21 days of the onset of symptoms.[22] Sputum PCR testing has replaced hybridization and direct antigen detection owing to its higher sensitivity and early diagnostic advantage.[22] Immunoglobulin M and immunoglobulin G testing with enzyme-linked immunosorbent assay and complement fixation require acute and convalescent samples and are not as sensitive early on in the disease process. Bacterial culture testing for *M pneumoniae* is not recommended due to the organism's fastidious nature, the length of time (approximately 4 weeks) required for growth to occur, and the test's relatively high cost.[23]

Other infectious triggers of EM are Epstein-Barr virus, parapoxvirus, varicella-zoster virus, adenovirus, hepatitis C virus, HIV, and cytomegalovirus. Infections with *Yersinia*, *Treponema pallidum*, *Histoplasma*, and *Mycobacterium* also have been associated with EM.[1]

MEDICATIONS AND VACCINES

Various medications have been reported as EM triggers, with sulfonamides as the most common; other medication triggers are barbiturates, hydantoins, nonsteroidal anti-inflammatory drugs, penicillins, tetracyclines, and phenothiazines.[2] Specific medications such as metformin, adalimumab, bupropion, and ciprofloxacin also have been implicated.

Drug-associated EM lesions test positive for tumor necrosis factor α, but not interferon-γ as in HSV-associated EM, suggesting a different pathologic mechanism.[24] Medication-triggered EM accounts for approximately 10% of cases; thus, it is important to expand the differential diagnosis to other dermatologic diagnoses such as SJS, TEN, generalized fixed drug eruption, exanthematous (morbilliform) drug eruption, or urticaria. Iris lesions with dusky or purpuric centers may appear similar to the lesions of pityriasis rosea, lupus erythematosus, urticaria, or urticarial vasculitis. EM must be differentiated from the autoimmune bullous diseases if bullous lesions are present.[9,25] Vaccine-triggered EM has been reported in association with the tetanus-diphtheria, hepatitis B, and smallpox vaccines.[9,26]

DIAGNOSIS AND MANAGEMENT

EM is a clinical diagnosis. Dermatologic target lesions support the diagnosis. Care involves attempting to determine the underlying trigger. If a trigger is identified, clinical management is directed at either treating the suspected infection or discontinuing the causal drug. In most cases, no treatment is required, since the rash usually resolves by itself over several weeks without complications.

Supportive and symptomatic treatment may be necessary. Moist compresses, oral antihistamines, and topical corticosteroids can relieve EM symptoms. Acetaminophen is recommended to reduce pain and fever. Oral prednisone dosed at 0.5 to 1 mg/kg/day (maximum 80 mg/d) for 4 to 5 days may be used in patients whose clinical presentation involves the oral mucosa. However, the use of oral corticosteroids for the treatment of EM remains controversial, since no controlled studies have shown a true benefit. Immunosuppression induced

by oral corticosteroid use may harm patients with underlying HSV-triggered EM.[9,11]

Skin biopsy to help exclude other dermatologic conditions may be indicated if the clinical picture is unclear.[9] EM has a characteristic histology, but it is not considered diagnostic. The histology varies with the age of the lesion, its appearance, and which part of the target lesion is biopsied. Laboratory testing may be performed to identify pathogens commonly associated with EM. Oral antiviral therapy (eg, acyclovir) for HSV infection or antibiotic therapy (eg, erythromycin/macrolide) for *M pneumoniae* may be required.

EM can recur with multiple episodes per year for many years. In most recurrent cases, the trigger is infection with HSV-1 or HSV-2. Although antivirals do not effectively treat an existing rash, recurrent EM can be treated with daily oral acyclovir dosed at 5 mg/kg/day twice daily (maximum 400 mg twice daily). Placebo-controlled double-blind studies have illustrated acyclovir's effectiveness in suppressing recurrent EM.[27] For patients who have a poor response to acyclovir, a regimen of valacyclovir or famciclovir may be tried.[28] Once a patient has been free of recurrence for 4 months, the daily antiviral dose can be reduced with the goal of eventual discontinuation. Recurrent EM also is associated with certain HLA antigen types: HLA-B15, B35, A33, DR53, and DQB1*0301, and specifically for recurrent HSV-triggered EM, DQw3.[19]

Other treatments used to suppress recurrent EM are dapsone,[29] hydroxychloroquine,[13] azathioprine, thalidomide, cyclosporine, mycophenolate mofetil, and photochemotherapy with psoralen and ultraviolet light.[9] These treatment approaches require the care of a dermatologist. Evidence supporting their use is limited, but they have shown clinical benefit for some patients.

EM typically resolves spontaneously without residual scarring over 1 to 4 weeks, with an average of 3 weeks. EM does not progress to SJS or TEN.[11] In some cases, hyperpigmented areas of skin discoloration may remain. ■

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Which Halloween Health Hazards Are Factual? Which Are Just Scary Stories?

Bryce M. J. Harvey, MD, and Linda S. Nield, MD—Series Editor

A PARENT ASKS

This year my 10-year-old son plans to go trick-or-treating with his friends without my supervision. What Halloween health hazards have been reported? What are some Halloween safety tips?

THE PARENT COACH ADVISES

Participating in Halloween is popular among children of all ages. In 2013, the estimated number of potential trick-or-treaters—children aged 5 to 14 years—in the United States was 41.2 million.[1] The age at which it is safe for a child to trick-or-treat without supervision should be determined on a case-by-case basis based on the child's maturity level and the maturity level of the other children in the group. A 2011 national survey found that 12% of children under the age of 5 trick-or-treated without adult chaperones, and only 35% of parents talked to their children annually about Halloween safety concerns.[2]

REPORTED HALLOWEEN HEALTH HAZARDS

Pedestrian Injury. While many parents worry about strangers and candy that has been tampered with, the true danger during trick-or-treating is pedestrian injury. Halloween is ranked as the No. 1 day of the year for child-pedestrian accidents and fatalities. One analysis of data from the National Highway Traffic Safety Administration's Fatality Analysis Reporting System[3] found that 115 child-pedestrian fatalities occurred on Halloween from 1990 to 2010. This average of 5.5 pediatric fatalities per year on Halloween is more than double the average of 2.6 pediatric fatalities on all other days of the year. The group at highest risk is 12- to 18-year-olds, who accounted for 32% of the fatalities, followed by the 5- to 8-year-old age group at 23%. The majority of these fatalities (60%) occurred during the peak trick-or-treating hours between 5 PM and 9 PM. The deadliest hour of trick-or-treating was from 6 PM to 7 PM. The drivers of the vehicles involved in one-third of the accidents were young adults between the ages of 15 and 25 years.

Tampered Candy. The fear of candy that has been tampered with is media-driven and is a common concern among parents nationwide. The results of a 2011 Harris Interactive poll showed that 24% of parents with children under the age of 12 years worry about poisoned treats.[2] To date, five reported deaths have been linked to Halloween candy: two in the 1970s that eventually were attributed to the direct actions of family members rather than strangers, one when a child ingested heroin that a relative had stashed among the candy, and one when a father murdered his son with cyanide-laced Pixy Stix candy in order to collect on a life insurance policy. Other fatalities in 1978 and 1990 later were determined to be associated with preexisting cardiac disease and natural causes. The 2001

Table. Halloween Health Tips[2,13,14]

- Provide adult supervision for children under the age of 12 years.
- If trick-or-treating in a car, provide transportation for adolescents, or take the time to educate them about increased vigilance on Halloween night.
- Avoid the use of cell phones and other electronic devices that might distract children from walking and/or driving safely.
- Choose costumes that are easily seen, or place reflective tape on outfits and bags.
- Attempt to complete trick-or-treating before the darkest part of night.
- Carry glow sticks and flashlights.
- Avoid clothing, masks, and costume props that can obscure vision or walking.
- Trick-or-treat at known households in communities with sidewalks or paths.
- Check treats for signs of tampering (although this occurs rarely), and eat only treats that are sealed in the original packaging.
- Continue healthy snacking
- Avoid treats that will stick to teeth, and continue proper dental hygiene practices.
- Set a limit to the amount of candy to be consumed each day.

case of a 4-year-old in Vancouver, Canada, who died a day after ingesting trick-or-treat candy was widely reported in the news media. After police ordered children and families across the area to dispose of their Halloween candy, it eventually was determined that the cause of death was streptococcal infection unrelated to Halloween candy.

Nevertheless, some incidents of Halloween candy tampering have been reported. In 2000, trick-or-treaters in Hercules, California, found marijuana-packed Snickers bars in their Halloween candy.[5] A police investigation found that a worker in the Hercules post office had found the candy bars among the undeliverable mail and took them home to distribute to trick-or-treaters. The tainted candy was the result of an unknown person's failed attempt to mail 5 ounces of marijuana to San Francisco. Still, while the threat of encountering poisoned Halloween candy is a possibility, the likelihood remains very low.

Another fear is the placing of sharp objects such as needles, glass, or razors into Halloween treats. Approximately 80 such cases have been reported since 1959.[6] The large majority of these cases have been determined to be hoaxes. A needle stick injury was reported in 2000 when a teenager bit into a tainted candy bar; however, no long-term complications were associated with the incident. A 49-year-old man eventually was criminally charged after investigations found that he had hidden needles in chocolate bars and had given them to trick-or-treaters.

Physical Assaults. Parents also may fear that their children may be vulnerable to physical harm from other children or adults while trick-or-treating without supervision. In 2009, a group of researchers studied the threat of strangers and child sex-crime rates associated with Halloween activity.[7] The authors concluded that there was no increase in the rates of sex crimes against children aged 12 years and younger around Halloween.

Other Health Hazards. In the United States, more than $2.4 billion is spent on Halloween confections each year, exceeding the number of sweets purchased for Easter, Christmas, and Valentine's Day.[8] Excessive sugar consumption can contribute to obesity and dental decay. Increased hospital visits related to abdominal pain and/or diarrhea secondary to ingestion of large amounts of sorbitol on Halloween also have been reported.[9]

HALLOWEEN HEALTH TIPS

The accompanying **Table** lists other practical Halloween health tips compiled from various resources.

Halloween can be an enjoyable way for families to spend time together and for pediatricians to promote healthy lifestyle practices. This yearly celebration is another opportunity to discuss the importance of proper food choices and dental hygiene. Although the suggested recommended age to allow children to trick-or-treat without adult accompaniment varies depending on the children's maturity level, parents should consider supervising children younger than 12 years of age.

Some parents might ask about the value of X-raying their children's Halloween candy. A study involving three hospitals that offered candy X-raying found that the 394 radiographs cost $1,625, with no positive results. The authors estimated that nationwide, Halloween candy X-raying could cost as much as $1.4 million and provide no proven benefit. ■

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For a fascinating look at the history of Halloween and trick-or-treating by Bryce M. J. Harvey, MD, and Linda S. Nield, MD, visit the online version of this article at **PediatricsConsultant360.com**.

Is Urinalysis Reliable After All for UTI Diagnosis in Young Infants?

Jessica Tomaszewski, MD

Schroeder AR, Chang PW, Shen MW, Biondi EA, Greenhow TL. Diagnostic accuracy of the urinalysis for urinary tract infection in infants <3 months of age. *Pediatrics.* 2015;135(6):965-971.

Urine culture is the gold standard for the diagnosis of urinary tract infections (UTIs) in children because of the reported suboptimal sensitivity of urinalysis (UA). The 2011 clinical practice guideline on UTIs from the American Academy of Pediatrics (AAP) suggests that the diagnosis of a UTI should include an abnormal UA result and a positive culture result.[1] This approach does not apply to infants younger than 2 months of age; therefore, infants with positive culture and negative UA results still are treated for a presumed infection, and the results are not seen as contamination of the sample or asymptomatic bacteriuria.

With these discrepancies in mind, Schroeder and colleagues sought to calculate the sensitivity of the UA for infants younger than 3 months of age who had bacteremic UTI, since an infection in blood and urine made contamination or asymptomatic bacteriuria less likely. The specificity of UA also was examined.

To accomplish this, a multicenter database of infants younger than 3 months of age (276 infants at 20 hospitals) with bacteremic UTI was analyzed. Infants who had major comorbidities, who had central access, or who received care in an intensive care unit were not included. Data on specific components of the UA were collected, and a uniform collecting system was established to account for variation in the categories used at various institutions. To calculate UA specificity, a sample group of 115 infants with negative urine cultures was analyzed.

When the various components of the UA were reviewed, leukocyte esterase (LE) had the highest sensitivity at 97.6% (95% confidence interval [CI], 94.5%-99.2%), and nitrites had the highest specificity at 100% (95% CI, 96.8%-100%). If a positive UA was defined by pyuria (>3 white blood cells/high-power field [WBC/HPF]) and/or any LE, the sensitivity (99.5%; 95% CI, 98.5%-100%) and specificity (87.8%; 95% CI, 80.4%-93.2%) were both higher than those calculated when using the AAP guideline's definition of a positive UA (ie, pyuria >3 WBC/HPF or bacteriuria), where sensitivity was 98.3% (95% CI, 95.2%-99.7%) but specificity was only 63.5% (95% CI, 54%-72.3%). A definition that included any positive UA component was highly sensitive (99.4% in infants with complete UAs; 98.4% in infants with incomplete UAs), but was less specific (60%, 95% CI 50.4%-69%) than the aggregate of pyuria and/or LE.

These results show that if the definition of a positive UA included pyuria and/or positive LE, it was highly sensitive and specific. A negative LE and the absence of WBCs in the urine were notably specific (87.8%) in infants with negative urine cultures. UA bacteria demonstrated poor specificity, especially compared with pyuria or LE. The near perfect sensitivity of UA may be explained by spectrum bias, or that the current gold standard (urine culture) is flawed.

The implications of this study are noteworthy. UTIs are the most common serious bacterial infection in febrile infants, and they lead to interventions such as prolonged hospitalizations, antibiotic therapy, and imaging. Requiring a positive UA for diagnosis may lead to overtreatment, creating a risk of harm and increasing health care costs. Further studies are needed to investigate the role of UA in the definition and diagnosis of a UTI in young infants. ■

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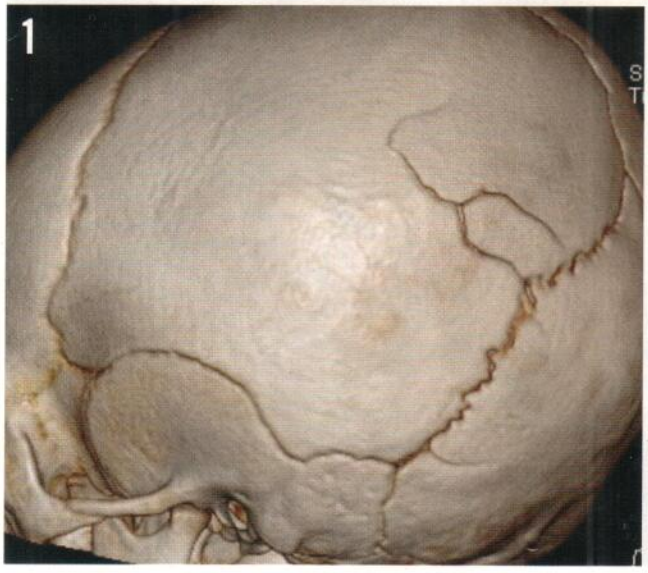
Epidural Hematoma in an Infant: Abuse or Accident?

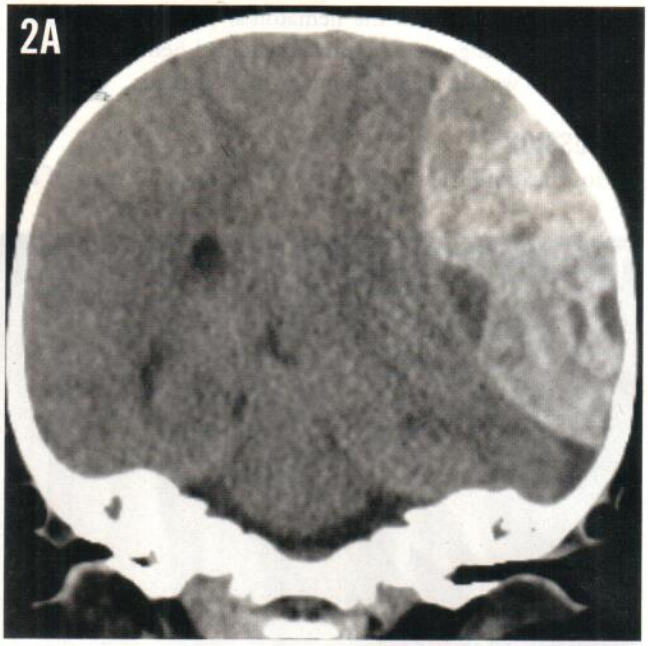
Dana M. Kaplan, MD; Amy Goldberg, MD; and Christine Barron, MD

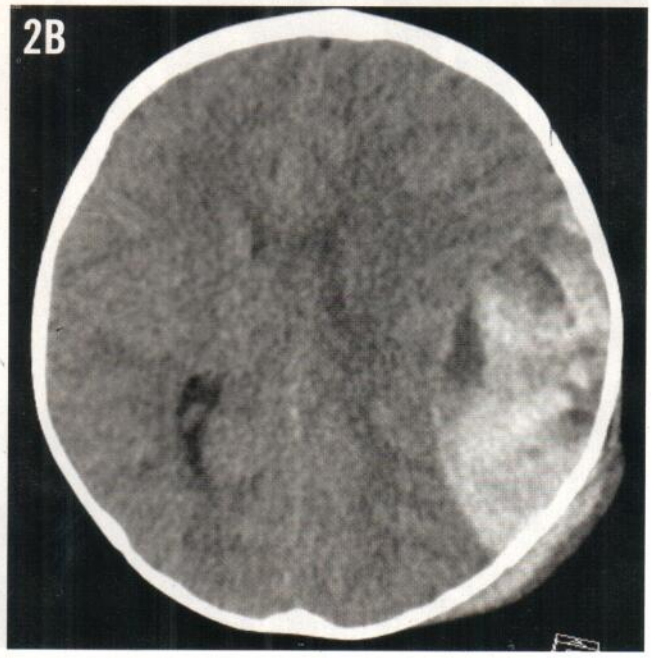
The parents of an 8-month-old boy brought him to the emergency department (ED) after a change in his mental status. The parents reported that the father and the patient had been sitting on a hardwood floor. The father was facing the child, supporting him from a seated position, when the boy suddenly swung his head backward, hitting it on the floor. The boy cried, fell asleep briefly, and then awoke at his neurologic baseline. He was given a bottle and fell asleep again. Approximately 1 hour later, he awoke vomiting, and then lost consciousness.

In the ED, the patient was obtunded, with a Glasgow Coma Scale score of 3, and was intubated. Physical examination findings were significant for a boggy area on his left scalp; no other injuries were identified. A noncontrast computed tomography (CT) scan of the head was performed the results of which are shown in **Figures 1 and 2**.

Do you suspect that the infant's injuries are accidental trauma, or signs of abuse?







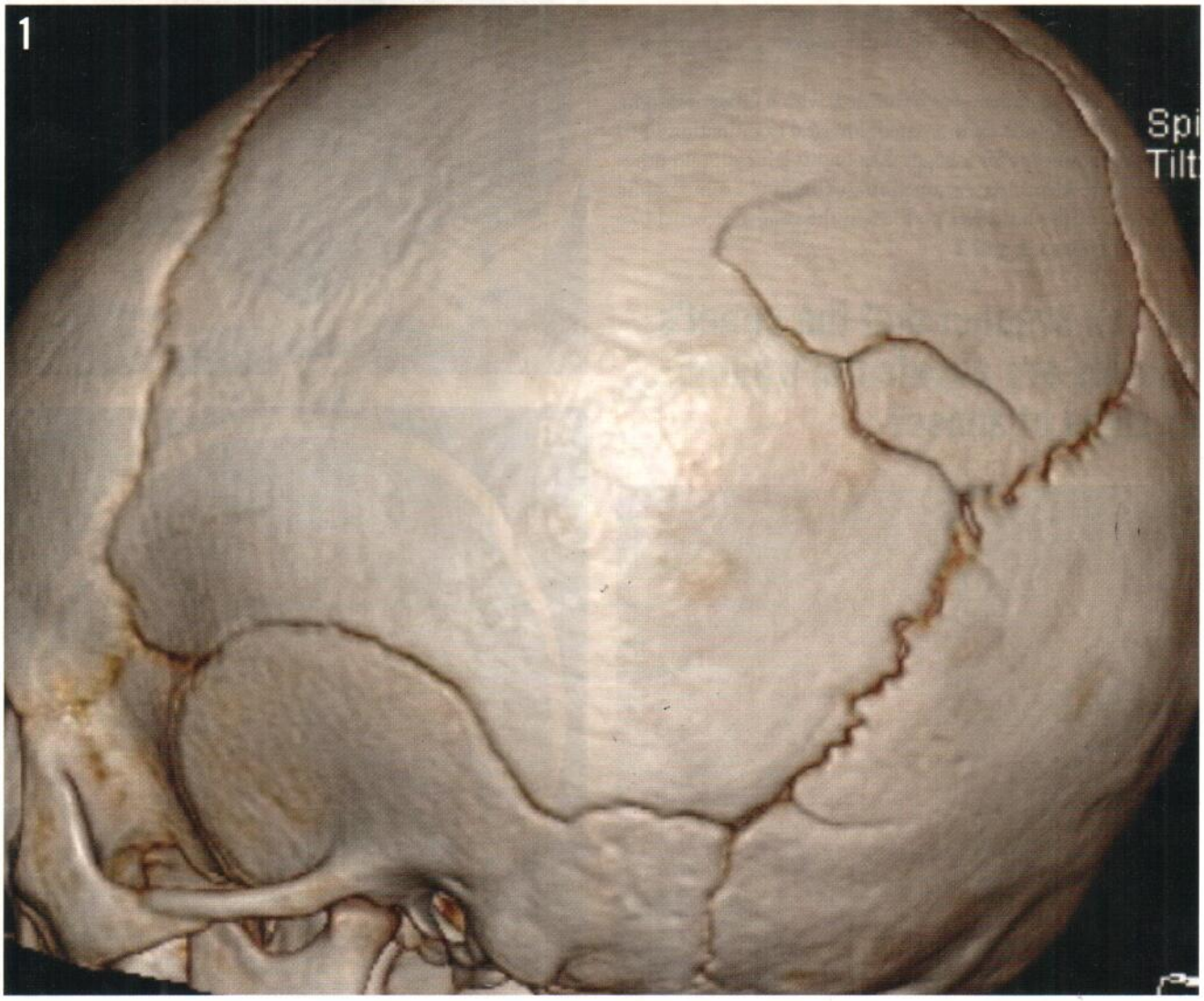
Answer: The infant's head injury was accidental

CT scan results (**Figures 1 and 2**) showed a left parietal skull fracture, along with an underlying massive epidural hematoma causing an 11-mm left-to-right midline shift, left lateral ventricular effacement, early uncal herniation, and left hemispheric cerebral edema with evolving infarcts in the left temporal and occipital lobes.

The boy was taken to the operating room, where a neurosurgical team evacuated the hematoma. Ophthalmologic examination findings were negative for retinal hemorrhages. Ultimately, he was discharged home with his parents.

DISCUSSION

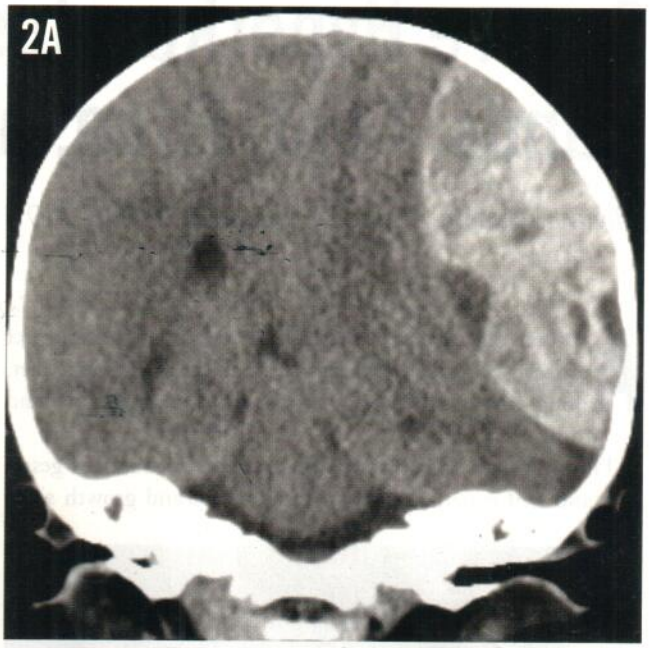
Most head injuries in children younger than 2 years of age result from falls. Such injuries are both age- and mechanism-dependent.[1] The mechanism is determined by the type of applied forces. In cases of falls, this correlates to fall height. Falls from low heights (1 m or less) primarily produce linear or translational impact forces,[1] where the head predominately moves in a straight line. Linear or translational impact forces are associated with contact injuries such as scalp swelling and bruising, skull fractures, epidural hematomas, and contact subdural hemorrhages. Rotational forces, where the head rotates around the neck, are associated with injuries such as noncontact subdural hemorrhages, diffuse axonal or sheer injury, and contusional white matter tears.[2] When retinal hemorrhages are numerous, involve multiple retinal layers, and extend beyond the posterior pole to the peripheral retina, they are similarly associated with rotational forces that have been applied to the brain.

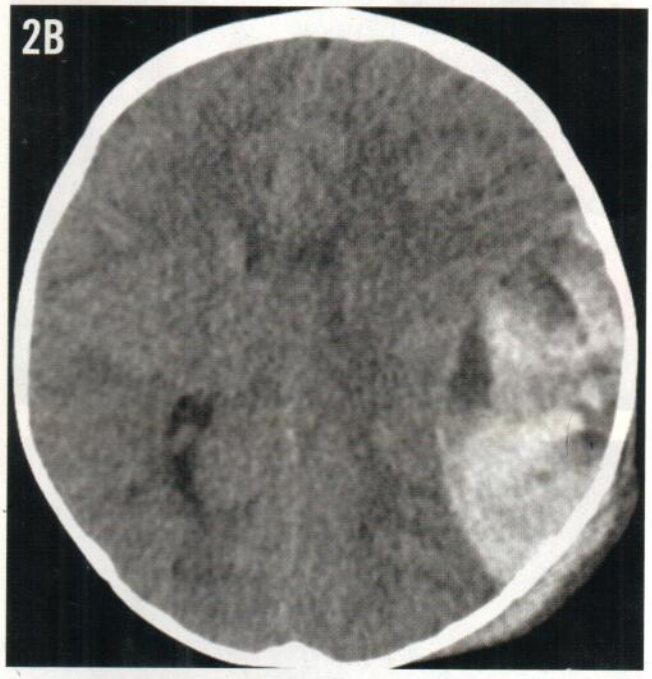


Contact subdural hemorrhages occur in cases of direct cranial impact that usually result in a skull fracture with an associated small underlying subdural hemorrhage.[3] In contrast, falcotentorial (interhemispheric or tentorial) subdural hemorrhages and subdural hemorrhages along a convexity (frontal, temporal, parietal, occipital) usually are the result of rotational forces and are highly concerning for child physical abuse in the absence of a history of a significant mechanism (eg, motor vehicle accident).[3]

Epidural hematomas typically arise from a direct impact that disrupts the middle meningeal arterial branches or dural venous sinuses. An overlying fracture also may be present.[4]

A history of a short fall often is provided by a caretaker to explain a child's injuries in cases of physical abuse.[5] The crucial distinction is that in cases of abuse, the explanation is inconsistent with the pattern, age, or severity of the injuries.[6] In the case described here, it might appear as though physical abuse was the cause of the child's injury, given the minimal mechanism described, the severe and acute change in the child's mental status, and the large intracranial hematoma. The critical point in differentiating accidental from nonaccidental trauma is the clinical history paired with a solid understanding of mechanisms of injury. The described history of a short fall and a direct impact onto a hard surface with an initial loss of consciousness, a lucid interval, and an abrupt onset of coma is consistent with the boy's skull fracture and epidural hematoma and, therefore, is consistent with an accident. ■





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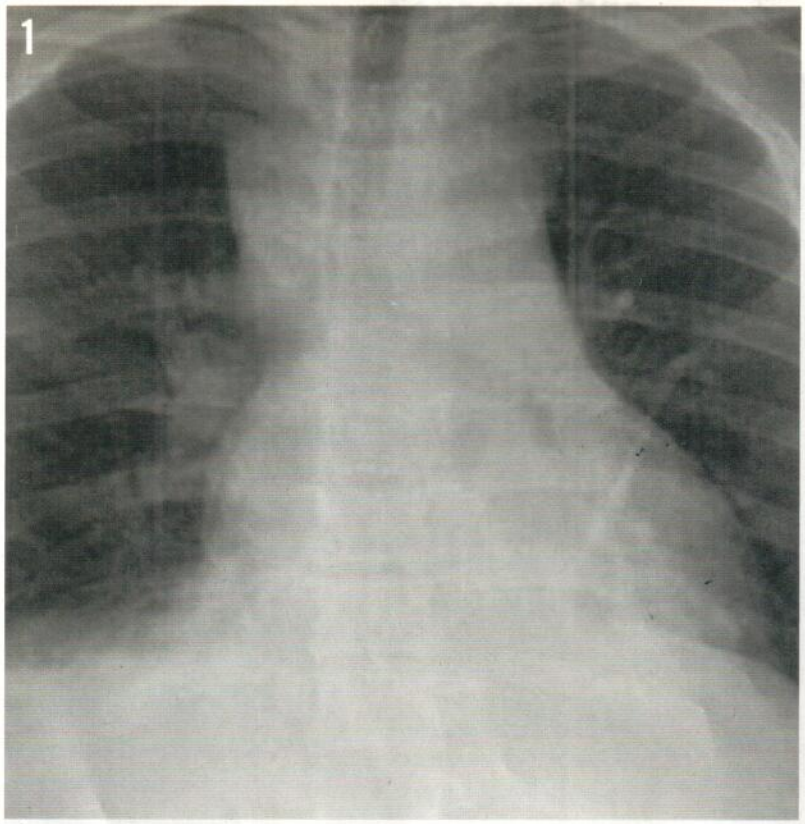
Answer: Mediastinal mass

A chest radiograph showed a widened mediastinum with a prominent cardiac silhouette (**Figure 1**). Further diagnostic workup was obtained; a computed tomography scan of the chest showed an extensive infiltrating mediastinal mass (**Figure 2**), and an echocardiogram exhibited early diastolic collapse of the right ventricle and atrium, mitral valve flow variation, and a pericardial effusion indicating tamponade physiology.

Results of cardiac enzymes tests and urinalysis were normal, but his white blood cell count was 28,900/μL (reference range, 4,000-10,000/μL). The peripheral blood smear showed anisopoikilocytosis, polychromasia, thrombocytopenia, and leukocytosis with 44% blasts; no Auer rods were seen. The lactate dehydrogenase level was 1,555 U/L (reference range, 105-333 U/L). Results of flow cytometry testing were consistent with a diagnosis of T-cell acute lymphoblastic leukemia.

Chest pain and syncope are common pediatric symptoms. The most common causes are benign and rarely life threatening, and they include musculoskeletal, pulmonary, psychogenic, and gastrointestinal etiologies.[1,2] Cardiac disease accounts for only 1% of cases of pediatric chest pain and syncope.[1-3]



A chest radiograph of a 17-year-old with chest pain and syncope showed a wide mediastinum and prominent cardiac silhouette.

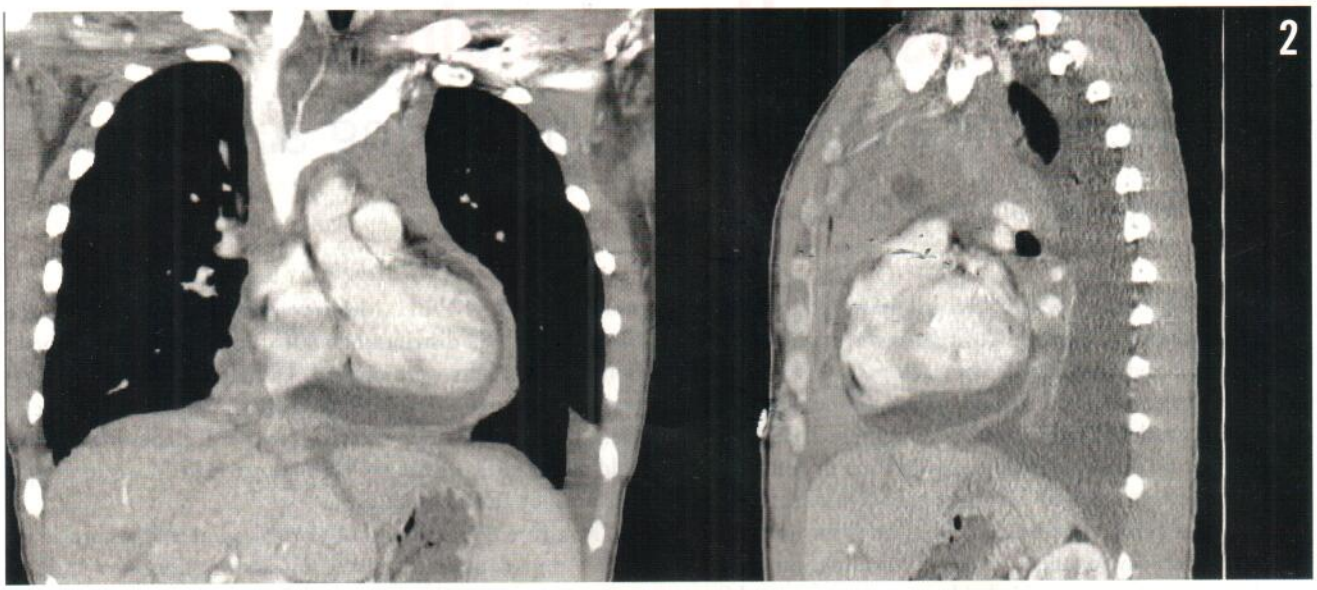
Although malignant etiologies of chest pain and syncope can be present in adults,[4-7] they are rare in children. In a study of 3,700 children with chest pain, for example, only 1.3% of cases were associated with syncope.[2] In adults, few published cases describe syncope without chest pain as the presenting symptom in a malignancy-associated cardiac mass.[8,9] A report of a case in an adolescent describes exercise-induced syncope with superior vena cava syndrome secondary to a mediastinal mass, but the patient had no chest pain or respiratory symptoms.[10]

Mediastinal masses usually present with nonspecific symptoms such as cough, chest pain, fever, and dyspnea.[11] Because most serious causes of pediatric chest pain and syncope are associated with cardiac conditions, current guidelines place little emphasis on malignant etiologies.

Given the frequency of chest pain and syncope in the pediatric population, standardized clinical assessment and management plans (SCAMPs) are available in many states to help reduce unnecessary testing.[1,2,12-15] According to SCAMPs for pediatric syncope and chest pain, only an abnormal cardiac history and examination findings, along with an abnormal ECG result, warrant further testing and referral to a specialist. Otherwise, reassurance and supportive treatment are recommended. Furthermore, abnormal lymphadenopathy on physical examination is not included in SCAMP criteria for further testing, and chest imaging is not part of the recommended diagnostic workup. However, our patient's case demonstrates that benign cardiac

Table. Signs and Symptoms Suggesting a More Serious Cause of Pediatric Chest Pain and Syncope

- Constitutional symptoms (eg, unexplained weight loss, fever, night sweats)
- Recurrent, nonexertional syncope and presyncope
- Positional chest pain
- Asymptomatic lymphadenopathy
- Splenomegaly or hepatomegaly
- Dyspnea and shortness of breath
- Diaphoresis and lightheadedness
- Weight changes
- Personal or family history of malignancy



Coronal view (left) and sagittal view (right) computed tomography scans showed a soft tissue mass infiltrating the 17-year-old's mediastinum.

evaluation results and a normal ECG do not preclude the need for further testing.

It is important to differentiate benign causes of chest pain and syncope from serious causes. Because a delay in diagnosis can be fatal or result in a poor long-term prognosis, a thorough physical examination and a detailed patient history to uncover any signs of more serious etiologies should be a part of the initial evaluation (**Table**). Painless lymphadenopathy in the setting of chest pain and syncope may suggest malignancy; in such cases, chest imaging and timely evaluation are indicated. Mediastinal masses should be included in the differential diagnosis of chest pain and syncope in children, especially after cardiac causes are ruled out. Immediate evaluation is needed when a mediastinal mass is detected. ■

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Entamoeba histolytica Infection

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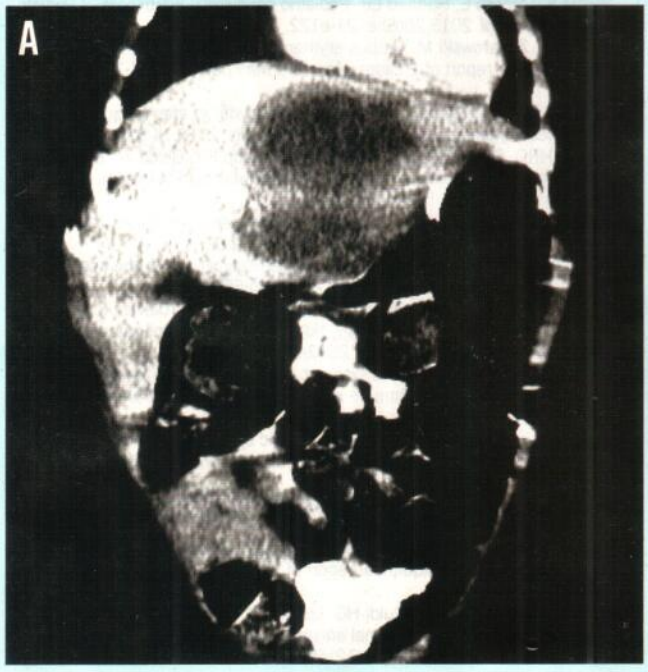
A 2½-year-old boy with no significant medical history presented to the emergency department with a 1-month history of generalized abdominal pain. No position made the pain better or worse, and it had no relationship to meals. He also had had intermittent fevers up to 39.4°C over the past 2 months. There was no associated diarrhea, gross blood in the stool, vomiting, or weight loss.

The boy had been seen by his primary pediatrician 3 weeks ago and had received a diagnosis of constipation. One week prior to presentation, he had been evaluated while in Mexico and had received a diagnosis of sinusitis and acute otitis media, for which he had been prescribed a 10-day course of amoxicillin/clavulanic acid. He had been feeling much better until the day of presentation, when the abdominal pain returned, and he refused to eat.

Physical examination revealed an afebrile, mildly ill-appearing boy in no apparent pain. His weight was below the 5th percentile and his height was in the 29th percentile. His abdomen was soft, nondistended, and mildly tender to palpation in the right upper quadrant and epigastrium. Bowel sounds were normal. The liver edge was palpable 1.5 cm below the right costal margin. He had no guarding, rebound tenderness, scleral icterus, or jaundice. The rest of the examination findings were noncontributory.



Laboratory tests revealed a white blood cell count of 27,000/µL, with 82.6% neutrophils, 9.5% lymphocytes, 7.7% monocytes, and 0.1% eosinophils; a hemoglobin level of 9.4 g/dL; mean corpuscular volume of 82 μm^3; and a platelet count of 802,000/µL. A comprehensive metabolic panel disclosed normal serum electrolytes; aspartate aminotransferase, 41 U/L; alanine aminotransferase, 24 U/L; alkaline phosphatase, 360 U/L; and total bilirubin, 0.5 mg/dL. Results of a coagulation profile were normal. The erythrocyte sedimentation rate and C-reactive protein level were increased at 104 mm/h and 202 mg/L, respectively.

A computed tomography (CT) scan of the abdomen with oral and intravenous contrast was performed, revealing a 5.2 × 4.5 × 5.9-cm abscess occupying the entire left lateral segment of the liver (**A**). Giemsa stain of fluid aspirated from the liver lesion, viewed at ×40 magnification, showed multinucleated cysts consistent with *Entamoeba histolytica* (**B**).

The patient received a diagnosis of hepatic abscess and was admitted for intravenous piperacillin/tazobactam and metronidazole. On day 2, CT-guided percutaneous drainage under sedation yielded 60 mL of purulent fluid. A percutaneous pigtail catheter was secured and drained approximately 50 mL of fluid per day. Gram stain of the fluid revealed no organisms. Giemsa and Papanicolaou stains of smears, as well as hematoxylin-eosin stain and periodic acid-Schiff reaction testing of cell block sections showed amoebic trophozoites and/or cysts. The sample was sent to the Centers for Disease Control and Prevention, where real-time polymerase chain reaction (PCR) test results for *E histolytica* returned positive. On discharge, the boy's medications were transitioned to oral metronidazole followed by a 20-day course of iodoquinol.

After discharge, the boy had good weight gain with no residual symptoms. Follow-up stool study results were normal 2 weeks after hospital discharge, and results of abdominal ultrasonography 6 weeks out confirmed the complete resolution of the hepatic abscess. The boy's mother and father both had positive stool cultures for *E histolytica*, while his 6-month-old sibling's stool culture results were negative.

Amebiasis is a parasitic infection caused by the protozoan *E histolytica*, which inhabits the large intestinal lumen of humans and primates.[1] Although 80% to 90% of cases are asymptomatic, amebiasis rarely can give rise to intestinal disease and extraintestinal manifestations, including liver abscess (most common) and pulmonary, cardiac, and cerebral dissemination.[2]

Amebiasis occurs in 1% to 5% of the world's population, with prevalence rates highest in the tropics and in areas of crowding and poor sanitation.[1] In developed countries such as the United States, it is seen mainly in migrants from and travelers to endemic countries.[2] The infection route is fecal-oral, usually when cysts are ingested from contaminated water and

food. After exposure, symptoms may develop in a few days to as late as 1 year, if at all, but 2 to 4 weeks is most common.[3]

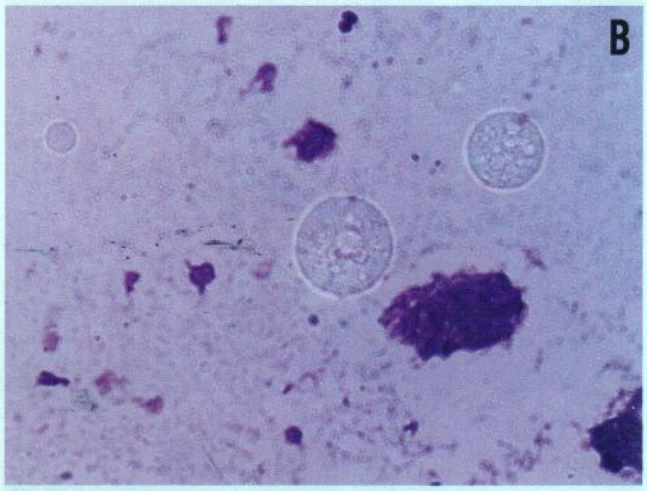
Symptoms depend on the site and extent of involvement and may include fever, malaise, abdominal pain, and anorexia, which may progress to liquid stools with flecks of blood or mucus.[1] Extraintestinal involvement primarily is due to hematogenous spread, with the liver as the most common site and occurring in less than 5% of cases.[1] Invasive amebiasis is a serious life-threatening disease in children, and delay in diagnosis significantly increases morbidity and mortality.

Identifying cysts or trophozoites in the feces is diagnostic of intestinal amebiasis. A single stool specimen evaluation has a yield of 50% to 60%, with higher sensitivity of approximately 95% with successive evaluations daily over 3 days, since cyst excretion is intermittent.[1,2] Endoscopic scrapings also can be examined.[1] A newer PCR test can differentiate *E histolytica* from the morphologically identical *Entamoeba dispar*—an important distinction, because infection with the latter does not require treatment.[3]

Liver abscess aspirates rarely have the classic "anchovy paste" appearance[4] and, when examined microscopically, seldom show trophozoites or leukocytes. If extraintestinal disease is suspected, serologic assays may be helpful in that they are more than 95% sensitive.[1] However, seropositivity also can indicate prior infection and may be delayed or absent in very young patients.[3]

The choice of amebicide is based on the location and severity of disease. Asymptomatic intestinal disease can be treated with paromomycin, iodoquinol, or diloxanide furoate. Therapy duration ranges from 7 to 20 days depending on the agent. The choice can be guided by expense, availability, and adherence factors. Extraintestinal disease is treated with 5 days of tinidazole or 7 to 10 days of metronidazole, followed by the same course as that of asymptomatic amebiasis.[5] Some authors promote needle aspiration as an additional routine therapy for the treatment of uncomplicated liver abscess.[6] Relapses are rare, but examination of feces should be repeated monthly for 4 to 6 months after therapy. Household contacts also should be examined for asymptomatic infection and treated if test results are positive.

Amebiasis is uncommon, especially outside endemic areas.



Its presentation is extremely variable and unpredictable. Diagnosis requires a high degree of suspicion, especially in children.

Our case underlines the importance of keeping amebic liver abscess in the differential diagnosis in a child with prolonged abdominal pain with fever and elevated inflammatory markers. Our patient's symptoms preceded his travel to an endemic area (Mexico); thus, we suspect he likely was infected via contamination from his parents, who were asymptomatic carriers. ■

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Gianotti-Crosti Syndrome

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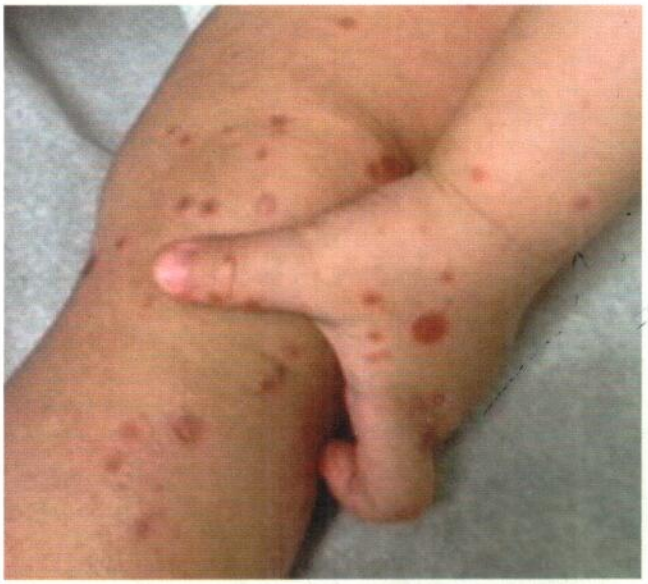
A 10-month-old infant presented with a 3- to 4-day history of a rash on her arms, legs, and ankles bilaterally. Her mother reported that the girl had a reduced appetite 1 week prior and had a fever for 2 days just before the rash broke out. The girl's mother also mentioned that the rash had begun with a small red dot, then had become a blister and had spread to the arms and legs. She denied any other symptoms in the girl, and there was no history of travel.

The infant did not seem to be bothered by the rash, and there had been no scratching, no medications given, and no sick contacts. Her vaccinations were up-to-date, and there



was no family history of similar lesions.

Physical examination revealed an afebrile, well-appearing, and comfortable infant with multiple erythematous and hemorrhagic maculopapular lesions on her elbows, hands, knees, legs, and ankles bilaterally. The girl received a clinical diagnosis of Gianotti-Crosti syndrome (GCS).

GCS is a rare, self-limited eruption that usually is associated with viral infections. In the 1950s, Gianotti and Crosti first described GCS as an exanthem associated with hepatitis B virus, which they termed papular acrodermatitis of childhood.[1-3]

Because it is benign and self-limited, most cases of GCS go unreported, making the overall incidence unknown.[4] It has been suggested that the incidence of GCS parallels that of precipitating infections in specific geographic regions.[4]

GCS can affect children between 3 months and 15 years of age but occurs primarily in children between 1 and 6 years old. Boys and girls are affected with equal frequency, but adult cases have been reported almost exclusively among women, suggesting that hormones influence the occurrence of GCS. There is no racial or ethnic predilection.

Because of routine universal hepatitis B vaccination during infancy, Epstein-Barr virus (EBV) infection is a more common cause worldwide; in the United States, EBV is responsible for as many as three-quarters of cases.[5-8] Other associated infectious agents include adenovirus, rotavirus, hepatitis A and C, parainfluenza, coxsackievirus, respiratory syncytial virus, cytomegalovirus, parvovirus, mumps virus, human herpesvirus 6, HIV, group A streptococcus, *Mycoplasma pneumoniae*, and *Bartonella*. Administration of certain vaccines also has been associated with cases of GCS.

Our patient's case of GCS eventually was found to have been caused by coxsackievirus serotypes B1 and B3.

GCS often is preceded by an upper respiratory infection. The condition usually begins with mild systemic symptoms such as, malaise, low-grade fever, lymphadenopathy, hepatosplenomegaly, and diarrhea. The lesions of GCS most often occur on the face, buttocks, and extremities. Although the lesions of GCS typically are nonpruritic, there are some reports of pruritus in the later stages of the rash. The only significant morbidity involves the underlying infectious process, particularly hepatitis B virus infection.

GCS is a clinical diagnosis, and no laboratory studies are necessary. The condition requires no treatment other than supportive therapies such as topical agents or systemic antihistamines for relief of pruritus.

Our patient's rash disappeared without any treatment after a week. ■

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