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Recognizable and Suspected Group A Beta-Hemolytic Streptococcal Syndromes

Streptococcus pyogenes *is an aerobic, gram-positive coccus, forming either short or long chains. These streptococci produce clear (beta) hemolysis on agar plates containing blood from a variety of mammalian species. They are known as group A beta-hemolytic streptococci (GABHS). These ubiquitous organisms are responsible for a wide array of human illnesses and GABHS are the pathogenic bacteria identified most frequently in man. GABHS is spread from carriers who have subclinical colonization or individuals symptomatic with active infection to others. Specific mechanism of spread from one person to another varies; however, close contact is required for transmission. Spread occurs via direct projection of oral secretions, transferred large airborne droplets, direct contact of cutaneous lesions, and to a lesser extent, through contact with inanimate, contaminated surfaces (fomites) or contaminated foods. This article discusses the wide variety of syndromes associated with GABHS.*

— The Editor

Introduction

GABHS infection occurs throughout all ages, with the pediatric population an overrepresented group. From the earliest depictions in the literature, GABHS has been a common pathogen throughout all of childhood. The incidence of pediatric infection of benign disease involving the skin and upper respiratory tract has been relatively constant over the millennium. The geographic and seasonal cluster spikes of benign disease expressions have been noted but are of lesser consequence than more virulent disease expressions. In the 1930s and 40s, there was a significant spike in morbidity and mortality from suppurative and nonsuppurative complications of oropharyngeal disease. For the first four to five decades of the post-antibiotic era, GABHS infection in the pediatric population was not a significant public health problem. During this timeframe, there were only sporadic case reports of invasive GABHS infections in children.[1] A dramatic rise in the incidence of invasive disease during childhood was first noted in the mid 1980s. Subsequently, there have been multiple depictions within the literature of invasive events such as streptococcal toxic shock syndrome and serious bacterial infections including bacteremia, septicemia, osteomyelitis, pyarthrosis, necrotizing fasciitis, pyomyositis and bacterial meningitis.[2]

Worldwide, GABHS infection may be a common reason for emergency department visits.[3] Pediatric patients may be brought for evaluation of delayed, nonsuppurative sequelae, such as glomerulonephritis, rheumatic fever or neuropsychiatric manifestations. Alternatively, patients may be brought for acute suppurative disease. Emergency physicians will evaluate patients with GABHS

Executive Summary

- Worldwide, group A beta-hemolytic streptococci (GABHS) infections are a common reason for emergency department visits.
- GABHS are spread via direct projection of oral secretions, transferred large airborne droplets, or through direct contact with oral secretions, transferred large airborne droplets, direct contact of cutaneous lesions, and through contact with inanimate, contaminated surfaces, or contaminated foods.
- Host defense factors and immunologic response to tissue contamination play an important role in limiting GABHS.
- GABHS has more than 125 different serologically distinct surface proteins under genetic control that render the organism to various degrees of invasiveness; however, only five genes are responsible for half of the invasive disease.

infection without manifestations of systemic illness (superficial syndromes) and those who are systemically ill (invasive syndromes).

In specific circumstances of GABHS infection, the clinician should diagnose GABHS as the offending pathogen after a history and physical examination. Alternately, the clinician will retreat from the bedside and contemplate GABHS as the most likely pathogen, but acknowledge other infectious agents are possibilities. This article highlights first the superficial syndromes, then the invasive syndromes of GABHS that are reliably diagnosed on clinical grounds alone. Lastly, the article discusses the circumstances of suspected GABHS infection.

Recognizable Superficial Syndromes

Since the earliest depiction in the medical literature, children have developed noninvasive infections with GABHS. The oropharynx and skin are the typical sites of infection; no complete explanation exists for the predilection of these sites for benign disease. Host defense factors and immunologic response to tissue contamination play an important role in limiting disease. However, virulence factors of the bacteria are major contributors to the clinical expression. GABHS has more than 125 different serologically distinct surface proteins (M proteins) under genetic control (emm gene) that render the organism to various degrees of invasiveness. Despite the large number of genes that encode the M protein, only five are responsible for half of the invasive disease.[4] Certain M types are more frequently associated with uncomplicated infections.[5] GABHS organisms also produce and release a large number of biologically active extracellular products that bear different degrees of toxicity to man. The complex interplay of host and virulence factors leads to several pediatric syndromes of throat and skin infection that are readily diagnosed as GABHS infection.

Scarlet Fever. Scarlet fever (scarlatina) refers to the clinical features of mucous membrane changes and rash associated with an exotoxin-releasing strain of *Streptococcus pyogenes.* Those with scarlet fever have either a symptomatic site of infection or a relatively silent colonization. In both cases, it is now known that the causative organism possesses a gene controlling exotoxin release. By current nomenclature, the erythrogenic substances are called streptococcal pyrogenic exotoxins (SPEs). Of the five known exotoxins, SPE A is most commonly responsible for scarlet fever. Scarlet fever is rare in children younger than 2 years. The affliction is most common in children between 4-8 years. In the past few decades, there has been a decline in scarlet fever in parallel with a decrease in the number of group A streptococcal strains producing SPE A.[6] The nasopharynx is the most common portal of entry. Scarlet fever also may follow streptococcal infection of disrupted skin from conditions such as bites, stings, atopic dermatitis or varicella. Incisional wounds can lead to surgical scarlet fever. The uterus is the least common portal of entry (puerperal scarlet fever).[7]

The typical patient with scarlet fever presents within 3-7 days of a defined streptococcal infection. Occasionally, there are no recognizable prodromal events. Those with oropharyngeal involvement may complain of rhinitis, sore throat, or neck discomfort. Those with pyoderma or surgical scarlet fever may have minimal localized symptomatology. The majority of scarlet fever patients do experience associated symptoms of malaise, fever, headache, and abdominal pain, with or without vomiting. On examination, patients are not ill. If the primary site of infection is the throat, the patients may have petechiae and punctate, red macules on palatal surfaces or the uvula. There may be an accompanying exudative tonsillopharyngitis. A patient seen within several days of the onset of the illness will have a white-coated tongue with protruding prominent red papillae. Within several additional days segments of the white coating disappear (*see Figure 1*). Ultimately, there is a consistently beefy red tongue referred to as a red strawberry tongue. The exanthem of scarlet fever first appears sparingly on the base of the neck and face. The exanthem appears on the trunk and reaches the lower legs last, if at all; the palms and soles remain clear. At the time of an early encounter, the

Figure 1. Partially denuded 'white strawberry tongue' of scarlet fever

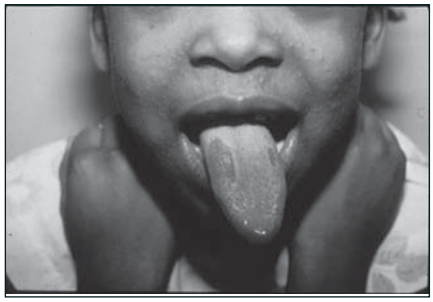


Figure 2. 'Milk sign' of angular chelitis during scarlet fever

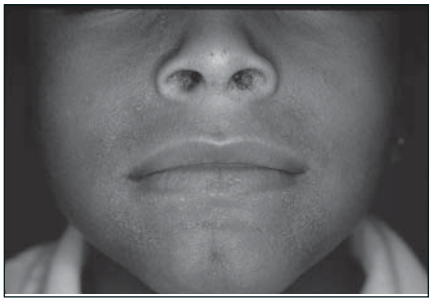
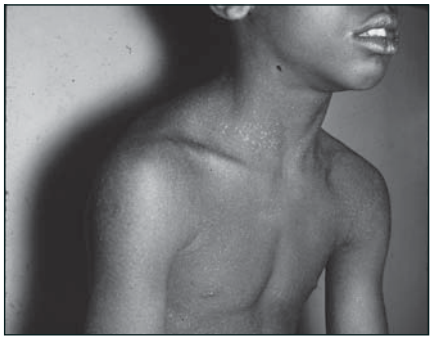


Figure 3. Fine, thin, flaking skin desquamation on the trunk in scarlet fever



rash is generally most intense and almost confluent at areas of pressure such as the axillae and inguinal folds. If the patient presents later, the exanthem becomes generalized. The exanthem spares the mouth creating circumoral pallor. The rash is finely papular with a texture of coarse sandpaper. In some patients, particularly darker-skinned individuals, the rash may be palpated more easily than it is seen.[8] If the patient is not evaluated within several days of the onset of the erythroderma, fine flakes of a whitish material appear to create an angular cheilitis, referred to as the "milk sign." (*see Figure 2*). At the end of a 1-week timeframe, desquamation occurs over the trunk (*see Figure 3*). Ultimately, desquamation may occur in the interdigital region.

The complications from GABHS infection are local tissue invasion and late inflammatory issues. These risks are not greater in those with scarlet fever when compared to those without toxin-mediated disease. However, treatment should be initiated to eradicate streptococci. A large number of oral penicillins, macrolides, and cephalosporins are approved for treatment of superficial GABHS (*see Table 1*).

Blistering Distal Dactylitis. Blistering distal dactylitis (BDD) is a superficial blistering lesion over the volar fat pad of the distal portion of a digit or digits. The disease most commonly affects children between 2 and 16 years but has also been described into adulthood.[9] The distinct clinical entity is rare. The mode of infection is unknown.

Clinical characteristics include a brief prodrome of mild pain and low-grade fever that is followed by distal digital swelling. The swelling occurs rapidly and the patient presents with a painful bulla with a clearly-defined, erythematous border (*see Figure 4*). The tense bulla is unilocular. The bulla may extend into the lateral and proximal nailfold. GABHS dactylitis most commonly affects a single digit with the thumb preferentially involved.[10] *Staphylococcus aureus* is increasingly reported as the pathogenic organism but is clinically distinguishable by the propensity for staphylococcal dactylitis to involve multiple finger pads.[11]

Treatment involves unroofing the blister with a needle puncture or a knife blade stab incision. The clear to white liquor within the bulla is cultured. Based on the gram stain and subsequent culture, a 10-day course of oral antibiotics known to be effective against the isolated species is curative. Recurrence is common.[12]

Perianal Dermatitis. Perianal dermatitis is an indolent infection of the superficial skin and subcutaneous perianal tissues. Perianal dermatitis was first described in 1966 in patients who had marked perirectal erythema.[13] The disease occurs most commonly in children between 6 months and 10 years of age. In the early contributions to the literature, one pediatrician reported one case for every 218 patient visits.[14]

Figure 4. Distal volar pad bulla of blistering dactylitis

The condition remains common but the signs and symptoms may be under-recognized or misinterpreted as other conditions, such as perianal sensitivity to contact agents, pinworm infestation, hemorrhoids, rectal fissure, poor rectal hygiene, and chronic constipation. Perianal dermatitis typically is associated with a primary GABHS site of involvement, such as nasopharynx or skin that is distant from the perineum. Autoinoculation results in perianal dermatitis.[14]

The mean duration of symptoms with perianal dermatitis is $\geq$ 6 months.[15] The dermatitis is unassociated with fever or constitutional manifestations. Patients first present with perianal pruritus. More than half of the patients then exhibit rectal pain that is exacerbated with

Table 1. Suitable Ambulatory Therapeutics for Superficial GABHS Infection

Penicillins & Macrolides			Cephalosporins		
Agent	**mg/kg/day**	**Divided**	**Agent**	**mg/kg/day**	**Divided**
Penicillin V	25-50	2-4	Cephalexin	25-50	2-4
Amoxicillin	40	2-3	Cefadroxil	30	1
Amox/clavulanate	43.8/6.2	2	Cefuroxime	20-30	2
			Cefpodoxime	10	2
Azithromycin	12	1	Cefprozil	20	2
Clarithromycin	15	2	Cefixime	8	1
E. estolate	20-40	2-4	Ceftibuten	9	1
E. ethylsuccinate	40	2-4	Cefdinir	14	2

Key: E = erythromycin

defecation. One-third of the patients report blood-streaked stooling or encounter drops of blood or pus on the toilet paper after wiping the perineum. Those severely affected become adverse to defecation; obstipation and constipation follow. On examination, a superficial, sharply marginated anal rash is noted. The area of involvement typically extends several centimeters from the anal verge but may extend into the vagina or foreskin.[16] The involved skin varies in color from pink to salmon to fiery red (*see Figure 5*). The discolored skin is flat and invariably tender to touch. The skin may be dry, or there may be a wet, white pseudomembrane.[13,14]

Clinical response, as defined by reduction in rectal symptoms, resolution of signs and negative perianal cultures for GABHS, is achieved in two-thirds of patients. Topical mupirocin is equally efficacious in the initial treatment as the FDA-approved oral antibiotic agents.[17] Recurrent disease may be treated with either oral clindamycin 20 mg/kg/day in 3 divided doses or a repeat course of oral penicillin or parenteral Bicillin CR combined with 4 days of rifampin.[18]

Impetigo. Impetigo contagiosa (nonbullous impetigo) is a superficial infection of the upper layer of the skin and the most frequently encountered infectious dermatitis throughout childhood. Endemic and epidemic impetigo occurs throughout all age groups. In the pediatric population, the problem primarily is seen in preschool-aged and school-aged children. The peak incidence is between the second and sixth years of life. It accounts for 50-60% of all bacterial skin infections and is the cause for 1-2% of all pediatric office visits.[19] Impetigo is a result of disruption of environmental factors that typically protect the skin against invasion. Following a break in the skin as a result of multifactors, which include abrasion, dermatitis, burn or bite, the resident skin flora invades the skin and proliferates.[3]

Fever and constitutional manifestations are absent throughout the entire course of impetigo, irrespective of the extent of infection. The first skin change is the development of a painless, occasionally pruritic, 1-3 mm erythematous, single or multi-clustered macule. The macules develop into papules as they elevate above the skin surface. The papules briefly fill with a clear liquid and lysis rapidly occurs. A serous discharge creates an oozing superficial lesion. The lesion itself is of varying size (*see Figure 6*). The lesions may range from a few millimeters to an excess of a centimeter. The central crusted lesion is honey colored. A discrete, nontender margin of erythema surrounds the honey-colored plaque. If the crust is manually removed, amber serous fluid exudes from the base. The fluid contains either a pure culture of GABHS, or GABHS can be recovered with methicillin-sensitive *Staphylococcus*

Figure 5. Superficial, sharply marginated perianal erythema of perianal cellulitis

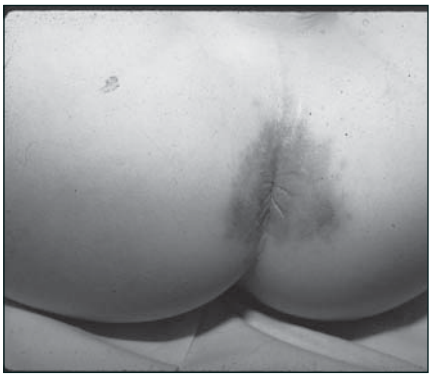
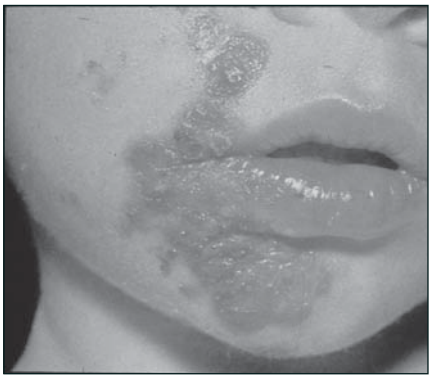


Figure 6. Honey-colored crusted pyoderma of impetigo contagiosa in perioral region



aureus. The staphylococci are presumed to be secondary invaders.[20] Despite its emergence as a frequent pathogen in folliculitis, cellulitis, furuncles, and abscesses, methicillin-resistant *Staphylococcus aureus* is an uncommon cause of non-bullous impetigo.[21,22]

Several labeled topical agents are effective for the management of impetigo. Mupirocin is effective against gram-positive bacteria, especially methicillin-sensitive *Staphylococcus aureus* (MSSA) and GABHS. As monotherapy for impetigo, mupirocin achieves pathogen eradication in 92% of cases.[3,23] Clinical cure rates are achieved in ≥ 90% of patients who have the 1% ointment applied 3 times a day for 7-10 days.[24] Retapamulin (Altabax™) is approved for patients 9 months of age and older. Retapamulin is active against MSSA, methicillin-resistant *Staphylococcus aureus* (MRSA) and GABHS. When the 1% ointment is applied twice a day for 5 days, microbial success rates of 92% and clinical cure rates of 86% are achieved.[25] Injectable Bicillin® C-R would be of utility for those with intolerance or refusal to take oral antibiotic agents.[26] Parenteral therapy also would be of utility in those with immunologic impairment or other comorbid conditions that would predispose to metastatic suppurative complications. Irrespective of the method chosen, the overwhelming majority of patients with superficial impetigo have a good outcome. Their lesions with effective therapy resolve over a 3-7 day time frame.

Intertrigo. Intertrigo refers to the superficial cellulitis localized in intertriginous folds. GABHS was highlighted as a causative pathogen in 1974.[27] The disease most commonly affects infants in the first six months of life.[28] Those younger than several months have deep neck skin folds as a result of big heads and decreased motor tone resulting in a flexed neck posture. Chubbiness in early infancy predisposes to trapping of moisture in redundant folds. Opposing skin surfaces in the axillae, leg folds, and inguinal folds encounter friction leading to maceration. This predisposes to secondary invasion by skin flora.[29]

Patients with GABHS intertrigo may display fussiness or experience a low-grade fever concurrent with the rash. The typical presentation is that of a recurrent eruption that has failed to respond to topical drying ointments, barrier creams, absorptive powders and/or anti-yeast preparations. Parents indicate vigorously washing the skin with soap and water. The skin is adequately dried, but within an hour moisture reappears. On examination, the deepest area of the affected skin fold has a uniformly fiery-red, denuded appearance. An erythematous, irregular border without satellite lesions will be seen on both sides of the fold (*see Figure 7*). When the fold is opened, a foul odor may be appreciated.

A 10-day course of oral penicillin or first-generation cephalosporin is curative. Recurrence is uncommon.[29]

Recognizable Invasive Syndromes

In the pre-antibiotic era, the majority of patients with GABHS infection had skin, pharyngeal, or soft tissue involvement. Uneventful recovery occurred in the majority of superficial infections. However, all patients were at risk for septic complications arising from direct extension of the streptococci from the primary site of infection. Those with local direct extension could indolently exhibit invasion of the blood (septic streptococcal infection). Alternately, physicians saw a hyperacute pattern of infection with onset to death within 24 hours. Those affected presented with marked fever, ≥ 41° C, sore throat, headache, delirium, and seizure activity. Because the majority evidenced a scarlatiniform eruption, this condition was referred to as malignant scarlet fever. In malignant scarlet fever, GABHS was not uniformly recovered from blood cultures.[30] These septic and malignant scarlet fever cases had all but disappeared in the first two to three decades of the antibiotic era.

In the mid-1970s, authors recognized the reemergence of serious GABHS infection.[31] Since the 1980s there have been multiple publications that have documented the emergence of severe, invasive disease.[32] Published incidence rates of severe invasive GABHS infections in North America have ranged from 1.5 to 7 cases per 100,000 persons annually.[33] These serious infections have been described worldwide and affect otherwise healthy children.[34] The invasive diseases seen include bacteremia without apparent focus of infection (occult bacteremia), overt septicemia, streptococcal toxic shock syndrome, erysipelas, and deep-seated infection within body regions where bacteremia is usually documented. Of these GABHS-invasive diseases, erysipelas and streptococcal toxic shock syndrome are apparent on the basis of clinical features.

Erysipelas. Erysipelas is the distinct form of rapidly-advancing cellulitis that involves the dermis and uppermost portions of the subcutaneous tissue layer. The rapid spread is the reason for its descriptive name, St. Anthony's fire, given in the Middle Ages. In the U.S. literature, the largest description involving 800 children and adults was published in 1913.[35] The earlier depictions of the disease emphasized overrepresentation of neonates and elderly. More recent reviews suggest children 2-12 years of age are predisposed to

Figure 7. Glistening, moist surface, and kissing skin fold infection of intertrigo

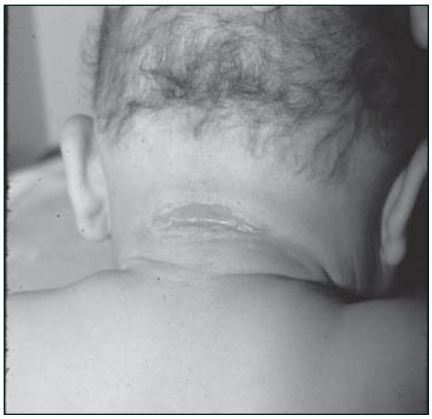
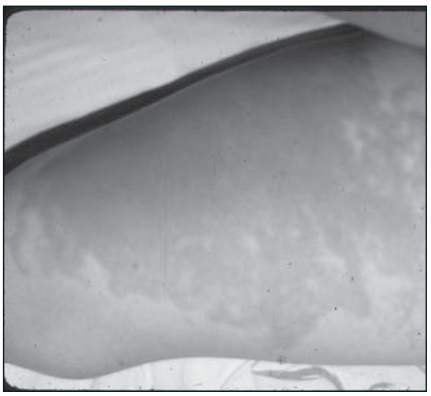


Figure 8. Painful, rapidly advancing infection of erysipelas on a thigh



erysipelas.[36] Of late, there has been a slight increase in the frequency of erysipelas.[37] The portal of GABHS entry is generally through direct inoculation of a break in the skin, such as an abrasion or an underlying inflammatory process such as varicella. GABHS may also enter through a surgical incision, such as an umbilical stump or circumcision site. Occasionally, the mode of skin inoculation is hematogenous.

The initial symptoms include fever, chills, headache, and generalized myalgias. These constitutional manifestations may predate the skin changes by 1-2 days. The first skin sign is a small plaque of redness. The plaque rapidly enlarges during a 12-24 hour period (*see Figure 8*).[38] The skin appears tense and edematous at the center of the inflammatory response. The intense edema may lead to the formation of unilocular or multilocular bullae on the surface of the lesion, which later rupture and crust.[39] As distinguishing factors from other forms of cellulitis, there is a slightly-raised, advancing border of erysipelas that may be sharply demarcated, and the entire area of involved skin is extremely painful.

The face was singled out as the most common site of infection in historical reports. Lesions on the face create a butterfly appearance with involvement of the cheeks and bridge of the nose. This form of erysipelas may resemble a contact dermatitis, angioneurotic edema or the malar eruption of lupus erythematosus. More recent reviews have reported 86% of the lesions occur on the lower extremities and only 6% involve the face.[40]

Patients tend to look toxic consistent with a rapidly evolving infection, occasionally associated with bacteremia.[41] The physician should submit a blood culture, a skin aspirate for culturing, and begin parenteral penicillin or parenteral cephalosporin therapy, such as cefuroxime or ceftriaxone. The febrile response resolves within 24 hours of the initiation of appropriate therapy. The progressive nature of the spreading cellulitis rapidly halts. The tissues may remain edematous for 2-5 days as lymphatic channels are often damaged; this predisposes to recurrence of erysipelas in the same location. In one case series, a recurrent infection appeared in 12% of patients during the first 6 months and in 29% during a 3-year follow-up among 233 patients with this diagnosis.[42]

Streptococcal Toxic Shock Syndrome. Streptococcal toxic shock syndrome (STSS) is an acute febrile illness associated with hypotension where there is recovery of GABHS from either a normally sterile site or from a nonsterile body surface where GABHS may be harbored. The disease is associated with hyperacute features that are reminiscent of the malignant scarlet fever detailed in the pre-antibiotic era. The fulfilling criteria for streptococcal TSS were established by a working group in 1993 (*See Table 2*).[43] Streptococcal TSS may occur in people of any age. There is a greater incidence in the elderly and in children less than 10 years of age. In the adult population, those affected may have no predisposing diseases or chronic debilitating illness. In the pediatric population, immunosuppression and chronic debilitating illness are predisposing factors. In children, varicella also predisposes to streptococcal TSS.[44] The incidence of STSS increased from the late 1980s into the new millennium. In the last decade STSS has occurred sporadically. There have

Table 2. Case Definition of Streptococcal Shock Syndrome[37]

I. Isolation of GABHS
   - A. From a normally sterile site
   - B. From a nonsterile body site

II. Clinical Signs of Severity
   - A. Hypotension
     AND
   - B. Two or more of clinical and laboratory abnormalities
     - Erythematous rash that may desquamate
     - Adult respiratory distress syndrome
     - Superficial or deep-seated infection
     - Coagulopathy (platelets $\leq$ 100,000/mm^3 or DIC)
     - Hepatic derangement (LFT's 2 times or more)
     - Renal impairment (creatinine 2 times or more)

Case Classification
   Definite: Fulfilling criteria I , IIA and IIB
   Probable: Fulfilling criteria IB, IIA

Adapted from *JAMA* 1993; 269:390-391

been reports of intrafamilial clusters of cases.[45] Patients with STSS have exposure followed by colonization with a virulent strain of *Streptococcus pyogenes.* The typical isolates that cause the disease are M protein types 1, 3, 12, and 28 that produce streptococcal pyrogenic exotoxins.[46]

Pain is the most common initial symptom of streptococcal TSS. The pain tends to emanate from a singular region of the body, such as a hemipelvis, hip, thigh or shoulder girdle. The pain is typically perceived to be muscular in origin by the patient. The patient will attribute the myalgia to a minor nonpenetrating trauma sustained to that body region in the antecedent 1-3 days. At the patient's encounter, vital signs are generally altered. The majority are at least minimally to moderately febrile. Fever may be absent in a small percentage of patients and up to 10% of patients can present with hypothermia.[47] Hypotension, as defined by a blood pressure less than two standard deviations of the mean for age, is present in half the patients. Tachycardia is prominent. Tachypnea occurs in those with an evolving acute adult respiratory distress syndrome. The patient may exhibit a sandpapery eruption or a generalized erythematous macular rash that ultimately desquamates. Some patients look nontoxic and simply appear uncomfortable. Others will exhibit an altered mental status and overt toxicity. The remainder of the physical examination is highly variable and depends in part on the portal of GABHS entry. Eighty percent of patients have a skin (superinfected varicella pox, cellulitis) or soft tissue (myositis, necrotizing fasciitis) focus of infection. Other foci include pharynx,[48] peritoneum, or wound.[49] Occasionally no portal of entry can be identified.

The emergency management of a patient with presumed streptococcal TSS reflects the steps necessary for management of septic shock. This includes hemodynamic stabilization. Aggressive fluid resuscitation with large volumes of crystalloid and colloid may be required. Ionotropic agents, such as dobutamine, dopamine, and norepinephrine may be considered when fluid resuscitation alone is insufficient to maintain adequate tissue perfusion.[46,50] Practice variation exists among emergency physicians for empiric management of toxic-appearing patients since the emergence of methicillin-resistant *Staphylococcus aureus.* Until recently, empiric antibiotic therapy that provides coverage for GABHS, methicillin-sensitive *Staphylococcus aureus* and anaerobes was acceptable. The combination of ceftriaxone or penicillin and clindamycin was provided. GABHS remains uniformly susceptible to penicillin. However, at high concentrations of bacteria, active replication is prevented by bacterial overcrowding. Killing of bacteria in the stationary phase is achieved by adding clindamycin. Clindamycin acts at the ribosomal level to inhibit protein synthesis. Clindamycin reduces the synthesis of emm proteins and exotoxins providing a benefit in controlling the inflammatory response. The dose of intravenous clindamycin is 25-40 mg/kg/day in 3-4 divided doses.[51] Methicillin-resistant *Staphylococcus aureus* is an extremely rare cause of toxic shock syndrome (less than 1% of cases). However, if epidemiologic clues lend a higher credence to MRSA as a potential cause of TSS, empiric therapy choices are clindamycin plus a beta-lactamase resistant anti-staphylococcal antimicrobial agent, or clindamycin plus vancomycin or linezolid.[52] Intravenous immunoglobulin at a single dose of 2 gm/kg or doses of 400 mg/kg/day for up to 5 days has been used. However, the most effective dosing has not been determined, nor has there been proven benefit in prospective, placebo-controlled trials.[53] Surgical intervention is a requisite with deep, necrotizing infections.

Suspected GABHS Infection

There are a number of circumstances where the practitioner believes a distinct clinical disease is caused by a specific infectious pathogen. The bedside, presumptive microbiologic diagnosis is a result of anecdotal experiences of the practitioner, national and regional epidemiologic data, and issues of clinical presentation that makes an organism's expression distinguishable from other potential organisms that can infect the same focus. GABHS is a pyogenic bacterium that lends itself to recognition by experienced practitioners. There are regional population-based data and national microbiologic data banks that support the constancy of GABHS as a cause of multiple infectious foci. Further, there are clues that suggest GABHS as a causative agent in specific sites of infection.

From a clinical point of view, there are two broad circumstances where GABHS should be high on the list of potential causative pathogens. They include localized infections in the head and neck and several deep-seated infections.

Head and Neck Infections. In the post-Hib, post-Prevnar era, the most commonly recognized aerobic pathogens in pediatric head and neck infections are GABHS, *Streptococcus pneumoniae*, nontypable *Haemophilus influenzae*, MSSA, MRSA, *Neisseria catarrhalis*, *Corynebacterium* species and *Mycobacterium* species.[54] Anaerobic bacteria outnumber aerobes in the normal oropharyngeal flora. Anaerobes, such as *Fusobacterium* species, *Prevotella* species, *Bacteroides* species, and *Peptococcus* species may be recovered from infected surfaces or blood in many head and neck infections.[55] These aerobic and anaerobic organisms have been confirmed pathogens with otitis media, mastoiditis, adenoiditis, sinusitis, uvulitis, preseptal cellulitis, orbital cellulitis, and dacryocystitis.[56-58] There are no clinical or biologic differences distinguishing GABHS as the causative pathogen from other pathogens in these sites of infection. GABHS can be placed at the height of a presumptive pathogen list based on history and physical examination with certain cases of pharyngitis and supraglottitis.

Pharyngitis. There is some imprecision in the clinical diagnosis of GABHS pharyngitis. However, certain epidemiologic, historical, and physical examination findings can be used to predict who is likely and who is unlikely to have GABHS pharyngitis.[59] A high prevalence of GABHS infection in the community and the history of a close contact with a well-documented case of GABHS infection is helpful. There is a high probability of GABHS as the pathogen in the febrile pediatric patient who presents with a chief complaint of sore throat if there has been an absence of coryza or cough.[60] A history of headache, abdominal pain, or nonbilious vomiting adds credence to the diagnosis.[61] On examination a scarlitiniform rash or tender, enlarged cervical lymph nodes are suggestive.[62] Oropharyngeal examination may be characteristic for GABHS infection. GABHS pharyngeal infection may involve one or several sites concurrently. The anatomical sites include lingual tonsils, palatine tonsils, adenoids, soft palate and posterior pharyngeal wall. Physical findings suggestive of GABHS infection include pharyngeal erythema, tonsillar exudates or petechial lesions on the palate.[63-65] A number of oral antibiotic agents may be prescribed to ameliorate the symptoms of GABHS pharyngitis (*see Table 1*).

Supraglottitis. Supraglottitis has replaced the now-antiquated term, epiglottitis. Supraglottitis refers to the inflammatory reaction to multiple insults of the glottic and extra epiglottic structures. In supraglottitis, the tissues around the glottis are potentially affected in all directions. Cephalad inflammation may involve the base of the tongue or uvula.[66] Outward inflammatory response may extend to the soft tissues and skin about the hyoid bone or into the submental lymph glands. Caudal extension typically inflames the mucosa over the arytenoids and aryepiglottic folds. The appellation supraglottitis is technically a misnomer when the inflammatory response extends deeply into the infraglottic structures. Invasion may extend into the trachea and larger bronchial trees.

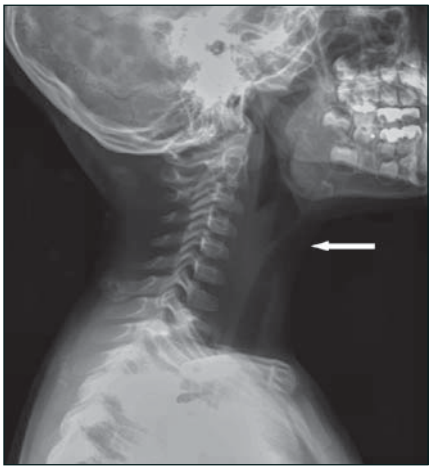
Supraglottitis can result from many insults. Examples of noninfectious origins include angioneurotic edema; inhalation of noxious fumes such as smoke, volatile hydrocarbons or illicit drugs; oropharyngeal thermal injury; and oropharyngeal blind finger sweep.[67,68] Infections remain the most common cause of supraglottitis. Nonbacterial causes are infrequent but include varicella zoster, herpes simplex and *Candida* species.[69] Bacteria are the primary cause of supraglottitis. Prior to the 1980s, *Haemophilus influenzae* accounted for all but 1% of the cases. As *Haemophilus influenzae* disease disappeared following the Hib conjugate vaccine, other organisms have emerged as pathogens. They include *Streptococcus pneumoniae*, *Pseudomonas* species, *Haemophilus parainfluenzae*, MSSA, group B, group C, and group G streptococci and GABHS.[70] The largest and earliest case series of GABHS supraglottitis was reported in 1986.[71]

The clinical features of supraglottitis are age dependent.[72] In the classic, peak 2-6 year range group, there is a rapid onset of high fever (≥ 39.5°C), sore throat, dysphagia, odynophagia without recognizable respiratory prodromal events. Patients are ill appearing, exhibit airway preserving posturing, or may evidence respiratory distress. Air hunger and inspiratory stridor may be present even if symptoms have been noted for less than 24 hours.[73] There are several pivotal findings in patients with GABHS supraglottitis. Patients tend to be older. They are more likely to have a slower progression of symptoms. They may have rhinorrhea or a barky cough during a prodromal 1-2 day period. Their sore throat may be present for several days prior to presentation to a physician. Fever may be low grade or lacking. Dysphagia, odynophagia, and respiratory embarrassment are more variable than when the disease is caused by other pathogens.[71,73,74] Patients with GABHS supraglottitis tend to have involvement of the epiglottis. However, they may have a propensity to develop edema of the arytenoid cartilage and the aryepiglottic folds. At the time of ED evaluation, a lateral radiograph may demonstrate a normal epiglottis, isolated aryepiglottic fold swelling, or supraglottic narrowing (*see Figure 9*). The radiograph may be erroneously interpreted as viral laryngotracheobronchitis.[75]

The mainstay of management for GABHS supraglottitis is airway stabilization. Endotracheal intubation should be carried out in a controlled fashion, preferably in an operating room setting. Intravenous ceftriaxone and vancomycin should be administered after epiglottic and blood cultures are obtained. The patient should be monitored in an intensive care setting. There is a tendency for GABHS supraglottitis to have a longer protracted course, including a more prolonged intubation.

Deep-Seated Infections. The definition of a deep-space infection may vary by author. However, the broad concept implies a de novo infection within a typically sterile

Figure 9. Lateral soft tissue neck radiograph of patient with GABHS supraglottitis demonstrating swelling of the aryepiglottic folds and subglottic narrowing (arrow)



space that is deeply seated from the body surface. An array of aerobes and anaerobes has been recognized with deep-seated infections. Over the decades, there has been modification of the incidences of specific pathogens, but many reports over time from different geographic areas suggest a constancy of GABHS infection with pneumonia, empyema, meningitis, myocarditis, pericarditis, primary peritonitis, osteomyelitis, and pyarthrosis.[76-79] Infections from GABHS in these deep locations are largely indistinguishable from other pyogenic bacteria. In contrast, there are population-based data supporting an increased incidence of GABHS infection in fascial planes and muscle. In the past few decades, GABHS has played a major role in causing necrotizing fasciitis and primary, nontropical (temperate) pyomyositis. Further, there are clinical differences, albeit minor, that provide clues of GABHS as the offending pathogen in fasciitis and primary temperate pyomyositis patients.

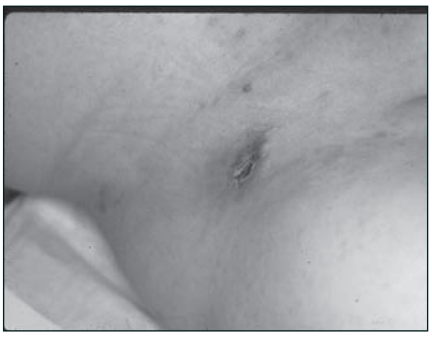
Necrotizing fasciitis. Necrotizing fasciitis (NF) is a rapidly-progressive soft tissue infection with severe involvement of the skin and subcutaneous tissues, including the superficial and deep fascia with sparing of the underlying muscle. For almost two centuries after the first depiction of this entity during the American Civil War, our acute care literature did not suggest a need for cognizance of this rare clinical syndrome. In the past two decades, multiple reports in the emergency medicine literature have alerted practitioners to this potentially devastating infection. The focused attention followed a resurgence of the disease in adults and children. NF attacks all age groups;[80] however, NF is not commonly seen in the pediatric population. In a large population-based study, NF has been reported to occur in 0.8 cases/million children/year.[2] Males from 1-5 years of age have the highest disease burden.[81]

Pediatric NF typically begins with the inoculation of bacteria into the subcutaneous tissue. In a newborn, scalp electrodes, umbilical, or foreskin incisions may promote bacterial invasion. In childhood, trivial blunt or penetrating trauma (such as injection, puncture wound), surgical incision or skin condition (such as atopic dermatitis, insect bite or varicella) that permits disruption of the epithelial barrier may lead to disease.[82] Although the inciting cause is not always apparent, after entry into the subcutaneous tissue, the offending bacteria multiply. The microbial and leukocytic infiltrate results in regional vasculitis, thrombosis of blood vessels with necrosis of skin, subcutaneous fat, subcutaneous nerves, and fascia.

NF is generally classified into two types. NF type I is a mixed floral infection with aerobe and anaerobic organisms. NF type II is secondary to GABHS and this type predominates in children. The initial clinical features of GABHS NF are nonspecific and include fever, chills, malaise, rhinorrhea, sore throat and cough with or without vomiting for a day prior to or concurrent with myalgia. In contrast to influenza, which NF simulates in the early presentation, NF myalgia is localized,[83] and typically occurs in the abdominal wall, groin or extremities.[33] If children are evaluated within a day of the onset of the symptoms, vital signs are minimally altered and children are not toxic appearing. The only clue in an early presentation to the diagnosis is localized muscle pain that is disproportionately severe. Over the next 24 hours muscle pain intensifies. Parents may witness antalgic gait or refusal to ambulate. This functional deficit may precipitate the emergency department encounter.[84,85]

With progression of the illness, temperatures range from 38.1°-41°C. Tachycardia is disproportionately seen in relation to the height of the fever. If the NF is associated with a streptococcal toxic shock toxin, hypotension may occur rapidly or insidiously.[86] Children may have a blunted affect and look systemically toxic. Positive findings are limited to the involved body part. Although skin findings early in the disease process may be absent,[87] they progress

Figure 10. Violaceous halo about secondarily infected varicella lesion overlying the clavicle heralding necrotizing fasciitis



depending upon the duration of disease. Initially the skin looks shiny and erythematous. Soft tissue swelling without distinct borders is apparent. The skin is warm and excruciatingly painful to light touch.[88] Within hours to a day the skin and subcutaneous tissues look and feel wooden to the examiner. Skin hypoesthesia or anesthesia develops as a prelude to frank skin necrosis.[89] The latter is typically heralded by blister formation. Coalescent, large bullae first fill with clear liquid. The fluid turns serosanguineous or frankly hemorrhagic. If lysed, they emit a foul smell. Deep to the skin, necrosis of fat and superficial fascia produces a thin, watery "dishwater pus."[82] Skin sloughing follows. If not properly addressed, deep veins may thrombose leading to distal gross edema, arterial and venous compromise with gangrene and loss of tissue.[90]

When GABHS NF results from superinfected varicella, the systemic and regional myalgia events are similar. The skin changes evolve differently. An individually infected pox that seeds deeper tissue first develops a faint, violaceous halo (*see Figure 10*). The area about the pox turns darker as the skin necroses. With varicella-induced GABHS NF, adjacent pox lesions may lead to localized, deeper invasion, or there may be concurrent multifocal areas of fasciitis.[91]

Emergency management of

patients suspected GABHS NF consists of initial stabilization with frank sepsis or, in the worst case scenario, septic shock. This is done by ensuring the patient's airway is maintained and signs of hypoperfusion are managed aggressively with intravenous fluid therapy and vasopressors. Submit blood, skin, aspirate and any wound tissues for culture. Since MRSA has been more frequently described as a cause of NF,[92] current empiric treatment suggestions include the combination of clindamycin and vancomycin.[93,94] Intravenous immunoglobulin has been suggested as adjunctive therapeutic modality. Blood component therapy may need to be provided prior to surgical debridement, which is the mainstay of NF treatment. Prompt, extensive surgical debridement may improve outcome. The goals of surgical intervention are to halt the progression of NF by removing all necrotic tissues. Hyperbaric oxygen treatment may be of benefit.[84]

Pyomyositis. Pyomyositis is an acute, invasive bacterial infection of muscle. Secondary pyomyositis is defined as infected muscle associated with a contiguous focus of infected skin, subcutaneous fat, fascia, or bone.[95] Most cases of pyomyositis are primary, where a solitary muscle group, or rarely, multiple distant muscle groups ultimately abscess. Worldwide, primary pyomyositis is predominately a disease within Africa and other tropical regions.[96] The disease was documented in a Japanese patient in 1885.[97] The first reported case in the United States was in 1991.[96] Primary temperate pyomyositis (TePM), particularly cases reported in the United States share similar epidemiology, microbiologic profiles and clinical features to those in warmer climates.

Primary TePM has a predilection for adolescents and young adults, but it may occur at any age. Approximately one-third of the cases occur in children.[98,99] The average age of pediatric cases is 8.4 years. Boys are affected two to three times more often than girls.[50,99] In a regional pediatric study, primary TePM was responsible for 1 in roughly 4,000 hospital admissions.[98] Future collective reviews will likely report an upward trend.

The pathogenesis of pediatric primary TePM is unclear. The final common pathway is muscle abscess formation with or without antecedent or concomitant bacteremia.[100] Most commonly the disease affects previously healthy individuals. Several populations have been described as at risk, including those with various hematologic disorders, particularly HIV and diseases with neutrophilic disorders. Other disease risks include an intramuscular venous malformation, Crohn's disease, nutritional deficiencies, and preceding infections, including varicella and pharyngitis.[101,102] In 1987, nonsteroidal anti-inflammatory use had been postulated as a precipitant due to blunting of the immune response.[97] Most people refute this earlier assertion. Antecedent blunt trauma to the area of ultimately inflamed muscle has been seen in 40% of cases caused by *Staphylococcus aureus* and *Streptococcus pyogenes.* These are the first and third respective most frequent organisms involved with primary TePM. The other agent that causes primary TePM, *Streptococcus pneumonia* is second in frequency. Cases of GABHS primary TePM tend to be unassociated with blunt trauma.[98]

Classically, primary TePM has three stages. In the earliest, "invasive stage," the muscle is seeded. Pain in a seeded body location is the uniform chief complaint. Typically, the pain is poorly localized in the area of inflamed muscle. Unilateral muscle groups in the thigh are the site most frequently involved. The patient, therefore, complains of an antalgic gait. Calf, buttock, arm, scapular region, chest wall, and anterior abdominal wall are sites in descending frequencies of pain from muscle involvement.[50,103,104] Infection in "hidden" muscles, such as the iliopsoas, obturator externus, may cause paraspinal pain, flank pain, groin pain, or hip pain.[95,98,105] Fever is present in 85-94% of children at the time of presentation and generally is low grade.[99,106] Patients examined during the invasive stage are generally nontoxic, and there are few clues to the ultimate diagnosis. Those with localized pain over a rather superficial muscle group may exhibit a rubbery or hard, woody feel to the muscle. There is no overlying erythema or blister formation. Those with deeper muscle groups may have an examination that is most consistent with other diagnoses, such as sarcoma, appendicitis, primary peritonitis, inguinal hernia, inguinal adenitis, pyelonephritis, vertebral osteomyelitis, intervertebral disk infection, or pyarthrosis of the hip.[107] In non-GABHS primary TePM, the onset of symptoms until presentation approaches 9 days.[99] In contrast, patients who seek attention with GABHS often will present early. The time course of their invasive stage is foreshortened. Occasionally, patients with GABHS have a nonexistent invasive phase and present in an advanced stage.

In the second "purulent phase," edematous muscle advances to abscess formation. In non-GABHS infection, this may occur over an additional 1-3 week timeframe. GABHS advances more rapidly. In the purulent phase, patients experience higher fever, chills, malaise, anorexia, nausea, vomiting and worsening muscle pain. All causes of primary TePM are associated with toxicity in the purulent phase. Skin overlying the affected muscle may now be edematous. The muscle belly is exquisitely tender and will feel full as the muscle belly has been replaced by pus.

In the third, "septicemic phase," patients demonstrate high fever, toxicity, and occasionally septic shock.[50,99,106] There may be multiple abnormalities on examination beyond vital sign changes. The patient may demonstrate multiple localized abscesses or distant abscess formation in other body parts, such as cardiac and pulmonary. In a superficial abscess, the muscle bed is tender and fluctuant. There may be

evidence of compartment syndrome with neurologic compromise secondary to elevated intracompartmental pressure.[108] Deeper space muscle abscesses may perforate leading to peritoneal soiling or extension to the thecal sac resulting in meningitis.[110]

Emergency management of patients suspected of primary TePM will vary depending upon the apparent stage at the time the diagnosis is being contemplated. In the invasive stage, broad-spectrum antibiotic therapy alone is usually sufficient. Those diagnosed in the purulent phase should have ultrasound-guided aspiration or surgical incision and drainage as well as broad-spectrum intravenous antibiotics.[113] Those in the septicemic phase have an attendant mortality between 40-80%. They require significant supportive therapy as well as surgical drainage and intravenous antibiotics. There is variation in clinical practice with respect to empiric therapy with suspected primary TePM. Suitable empiric antibiotic therapy includes a third generation cephalosporin combined with vancomycin or clindamycin.[111-114]

Conclusion

GABHS infections are common in pediatrics. The spectrum of disease varies from simple noninvasive skin infections to massive septicemia. Important to the clinician is the recognition of simple disease processes such as scarlet fever and perianal dermatitis that may be easily managed. A high degree of suspicion, early recognition, and aggressive management may improve the outcome for patients with significant infections. Overall, an awareness of the syndromes associated with GABHS is important to the ED physician.

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CME Questions

41. GABHS invasiveness is determined by which virulence factors?
    A. Exotoxins
    B. Surface M proteins
    C. Hyaluronic acid
    D. Immune complexes

42. Streptococcal pyrogenic exotoxins are responsible for which of the following?
    A. Nephritis and rheumatic fever
    B. Erysipelas
    C. Intertrigo
    D. Scarlet fever

43. The erythroderma of scarlet fever:
    A. spares the perioral region.
    B. first appears on palms and soles.
    C. spares the trunk.
    D. desquamates in the second to third week.

44. Fever and constitutional manifestations are absent in which of the following GABHS-induced diseases?
    A. Toxic shock syndrome
    B. Impetigo contagiosa
    C. Scarlet fever
    D. Erysipelas

45. Patients tend to look toxic in which of the following GABHS-induced diseases?
    A. Impetigo contagiosa
    B. Blistering dactylitis
    C. Erysipelas
    D. Perianal dermatitis

46. Myalgia is a prominent manifestation in all but one of the following GABHS-induced diseases?
    A. Toxic shock syndrome
    B. Pyomyositis
    C. Necrotizing fasciitis
    D. Scarlet fever

47. The patient suffering from GABHS-induced supraglottitis will likely:
    A. be younger than 2 years of age.
    B. seek attention within hours of onset.
    C. have aryepiglottic fold edema.
    D. require a shorter intubation.

48. Aggressive surgical debridement is a requisite adjunct in the management of GABHS-induced:
    A. supraglottitis.
    B. pharyngitis associated with toxic shock syndrome.
    C. necrotizing fasciitis.
    D. blistering dactylitis.

Answers: 41. B. 42. D, 43. A, 44. B, 45. C, 46. D, 47. C, 48. C.

Pediatric Emergency Medicine Reports

CME Objectives

Upon completion of this educational activity, participants should be able to:

1. recognize specific conditions in pediatric patients presenting to the emergency department;
2. describe the epidemiology, etiology, pathophysiology, historical and examination findings associated with conditions in pediatric patients presenting to the emergency department;
3. formulate a differential diagnosis and perform necessary diagnostic tests;
4. apply up-to-date therapeutic techniques to address conditions discussed in the publication;
5. discuss any discharge or follow-up instructions with patients.

CME Instructions

Physicians participate in this continuing medical education program by reading the article, using the provided references for further research, and studying the questions at the end of the article. Participants should select what they believe to be the correct answers, then refer to the list of correct answers to test their knowledge.

To clarify confusion surrounding any questions answered incorrectly, please consult the source material. After completing this activity, you must complete the evaluation form that will be provided at the end of the semester and return it in the reply envelope provided to receive a credit letter. When your evaluation is received, a credit letter will be mailed to you.

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Pediatric Emergency Medicine Reports

The Practical Journal of Pediatric Emergency Medicine

Recognizable and Suspected Group A Beta-Hemolytic Streptococcal Syndromes

Suitable Ambulatory Therapeutics for Superficial GABHS Infection

Penicillins & Macrolides

Agent	mg/kg/day	Divided
Penicillin V	25-50	2-4
Amoxicillin	40	2-3
Amox/clavulanate	43.8/6.2	2
Azithromycin	12	1
Clarithromycin	15	2
E. estolate	20-40	2-4
E. ethylsuccinate	40	2-4

Key: E = erythromycin

Cephalosporins

Agent	mg/kg/day	Divided
Cephalexin	25-50	2-4
Cefadroxil	30	1
Cefuroxime	20-30	2
Cefpodoxime	10	2
Cefprozil	20	2
Cefixime	8	1
Ceftibuten	9	1
Cefdinir	14	2

Case Definition of Streptococcal Shock Syndrome

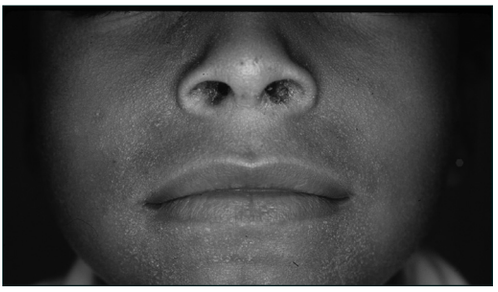
I. Isolation of GABHS
   A. From a normally sterile site
   B. From a nonsterile body site
II. Clinical Signs of Severity
   A. Hypotension
      AND
   B. Two or more of clinical and laboratory abnormalities
      - Erythematous rash that may desquamate
      - Adult respiratory distress syndrome
      - Superficial or deep-seated infection
      - Coagulopathy (platelets $\leq$ 100,000/mm^3 or DIC)
      - Hepatic derangement (LFT's 2 times or more)
      - Renal impairment (creatinine 2 times or more)

Case Classification
Definite: Fulfilling criteria I , IIA and IIB
Probable: Fulfilling criteria IB, IIA

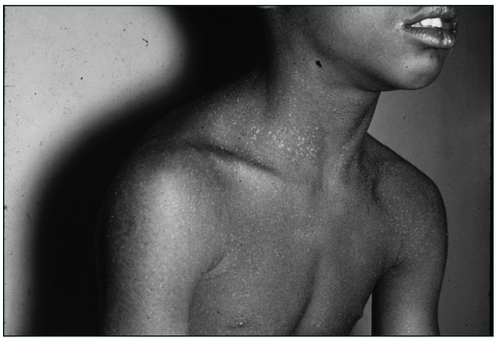
Adapted from *JAMA* 1993; 269:390-391

Partially denuded 'white strawberry tongue' of scarlet fever

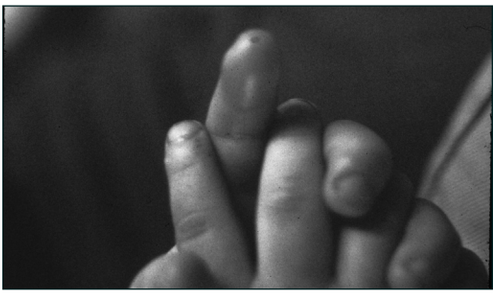
'Milk sign' of angular chelitis during scarlet fever



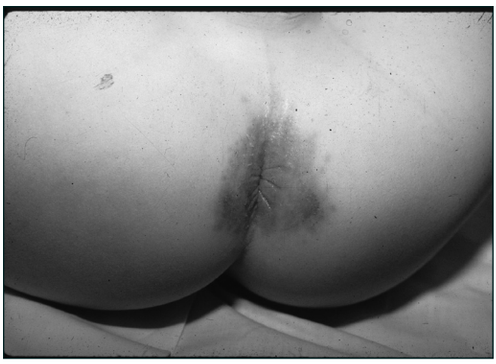
Fine, thin, flaking skin desquamation on the trunk in scarlet fever



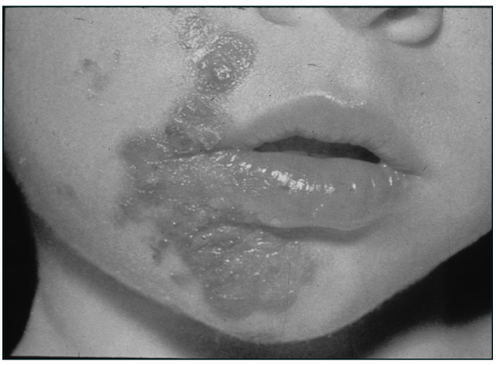
Distal volar pad bulla of blistering dactylitis



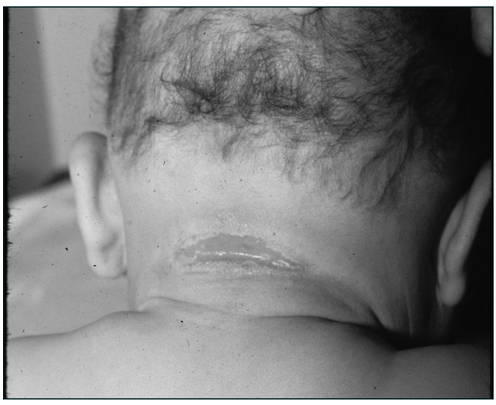
Superficial, sharply marginated perianal erythema of perianal cellulitis



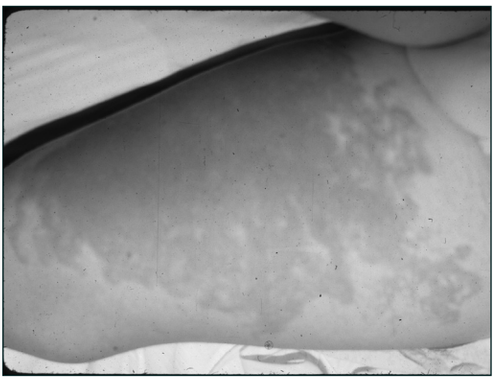
Honey-colored crusted pyoderma of impetigo contagiosa in perioral region



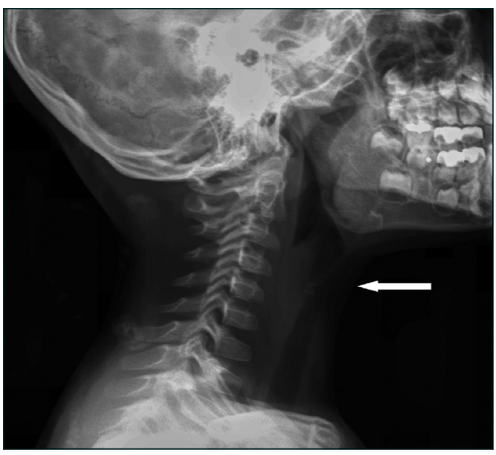
Glistening, moist surface, and kissing skin fold infection of intertrigo



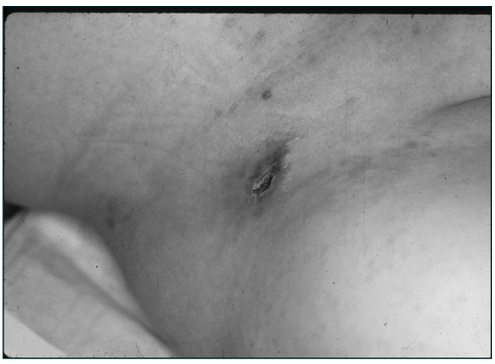
Painful, rapidly advancing infection of erysipelas on a thigh



Lateral soft tissue neck radiograph of patient with GABHS supraglottitis demonstrating swelling of the aryepiglottic folds and subglottic narrowing (arrow)



Violaceous halo about secondarily infected varicella lesion overlying the clavicle heralding necrotizing fasciitis



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