Infectious Disease Epidemiology Section
Office of Public Health, Louisiana Dept of Health & Hospitals
800-256-2748 (24 hr. number)
www.infectiousdisease.dhh.louisiana.gov

Revised 7/17/2014

NECROTIZING FASCIITIS

Necrotizing fasciitis (NF), commonly known as flesh-eating disease, flesh-eating bacteria or flesh-eating bacteria syndrome, is a rare infection of the deeper layers of skin and subcutaneous tissues, easily spreading across the fascial plane within the subcutaneous tissue. The most consistent feature of NF was first described in 1952 as necrosis of the subcutaneous tissue and fascia with relative sparing of the underlying muscle.

Necrotizing fasciitis progresses rapidly, having greater risk of developing in immunocompromised conditions such as advanced diabetes, chemotherapy and transplantation. It is a severe disease of sudden onset and is usually treated immediately with surgical debridement and high doses of intravenous antibiotics, with delay in surgical treatment being associated with higher mortality.

Many types of bacteria can cause necrotizing fasciitis:
Group A streptococcus (*Streptococcus pyogenes*),
Staphylococcus aureus,
Clostridium perfringens,
Bacteroides fragilis,
Aeromonas hydrophila,
Vibrio vulnificus …..and more.

The disease is classified as:
Type I: polymicrobial, due to a number of different organisms, with 55% to 75% of cases in different series,
Type II: monomicrobial, due to a single infecting organism, with 25% to 45% of cases in different series.

Historically, most cases of Type II infections have been due to group A streptococcus and staphylococcal species. Since as early as 2001, a particularly difficult to treat form of monomicrobial NF has been observed with increasing frequency caused by methicillin-resistant *Staphylococcus aureus*.

STREPTOCOCCUS GROUP A NECROTIZING FASCIITIS

Necrotizing fasciitis is a deep-seated soft tissue infection resulting in progressive destruction of subcutaneous tissue, fat and fascia. If not diagnosed and treated in a timely fashion, this infection results in gangrene, with substantial morbidity and mortality. Streptococcal NF has been dubbed the "flesh-eating disease" by the public and lay press, and has struck panic into those communities in which cases have appeared in clusters.

NF caused by group A streptococci is the most rapidly progressive and devastating form of the disease. Approximately 50% of adult cases are associated with toxic shock and multiorgan failure and the mortality rate ranges from 30% to 70%. Although NF may be caused by a variety of other aerobic and anaerobic microorganisms, those that are caused by group A streptococci are the most likely to present difficulty in early diagnosis and to result in devastating consequences, should the diagnosis be missed.

Epidemiology

The CDC estimates the overall incidence of invasive group A streptococcal infections in the general population to be approximately three per 100,000; only a minority of such infections are NF. The incidence may vary widely by time and place, dependent on the virulence of circulating group A streptococcal strains and the immunity of the local populace to them.

Clinical Presentation

The infection often begins at a site of significant local trauma: surgical incisions or penetrating injuries or minor injuries, such as cuts and burns, or non-penetrating injuries, such as blunt trauma and muscle strain. Secondary infection of varicella lesions is a well-recognized precursor of NF, and streptococcal bacteremia in children and occasionally, in adults.

The time elapsed between onset of symptoms and initial visit to a health care provider ranged from hours to seven days (median, two days).

The major presenting complaints are:

- Localized pain with or without associated swelling, tenderness, or erythema (87%)
- GI complaints (nausea, vomiting and diarrhea) (53%)
- Influenza-like symptoms of aches, chills and fever (47%)
- Afebrile patients may have received antipyretics, particularly NSAIDs. These findings led to diagnoses of musculoskeletal strain, viral gastroenteritis and influenza.

Suspicion should be increased by fever, tachycardia, GI symptoms (nausea, vomiting, diarrhea), and generalized myalgias and signs suggestive of impending STSS, such as hypotension (systolic blood pressure less than 100 mm Hg), generalized erythematous macular eruption and altered mentation.

Once the diagnosis is seriously considered, antimicrobial therapy should be instituted promptly. Crepitus, or obvious gas in tissues is not a feature of streptococcal NF and indicates that other organisms, most likely anaerobes, are involved. Although there are no controlled studies of the response to therapy, experimental evidence suggests that clindamycin is the agent of choice in streptococcal NF and penicillin as well. Although clindamycin-resistant group A streptococci are exceedingly rare in the United States at the present time, the theoretical possibility of such resistance exists and penicillin and clindamycin are not antagonistic. If the microbiologic diagnosis has not been established, then an expanded-spectrum penicillin or cephalosporin should be added to clindamycin to ensure coverage against Gram-negative bacteria. It is imperative to realize, however, that antimicrobials are adjunctive therapy for NF and complete surgical debridement is the sine qua non for cure.

CLINICAL SIGNS OF STREPTOCOCCAL TOXIC SHOCK SYNDROME (STSS)

(Based on the STSS case definition published in JAMA 1993; 269:390-391)

Please enter clinical finding and/or laboratory information on the following components of the STSS definition. Record the HIGHEST or LOWEST value within 48 hours of admission.

(Note: Actual laboratory results are preferred to clinical findings. Where the baseline laboratory values are abnormal, please include baseline value or answer .yes. to criteria if value is twice the baseline value or, for children, twice the 95th percentile for age)

A. Hypotension [] Y [] N [] DK Lowest systolic BP _____ mm Hg

(Systolic BP # 90mm Hg)

B. Multisystem involvement

1. Renal impairment [] Y [] N [] DK Highest creatinine _____mg/dL

(Creatinine > 2 mg/dL)

2. Coagulopathy [] Y [] N [] DK Lowest platelets _____ (000)/mm2

(Platelets < 100,000/mm2)

DIC [] Y [] N [] DK

3. Liver involvement [] Y [] N [] DK Highest SGOT (AST) _____IU/ml

(SGOT or SGPT > 70 IU/ml) Highest SGPT (ALT) _____ IU/ml

(Total bilirubin > 2 mg/dL) Highest Bilirubin _____ mg/dL

4. Adult respiratory distress syndrome [] Y [] N [] DK

Generalized edema [] Y [] N [] DK

Pleural/peritoneal effusion with hypoalbuminemia[] Y [] N [] DK

(hypoalbuminemia = serum albumin < 3 mg/dL)

5. Rash [] Y [] N [] DK

If yes, was it: [] Generalized [] Focal (location_____) [] DK

Rash type: ____ (1 = macular, 2 = papular, 3 = macularpapular, 4 = petechial,

5 = bullous, 6 = vasicular, 7 = other, specify: _____)

6. Soft-tissue necrosis [] Y [] N [] DK If yes, location _____

Surgery [] Y [] N [] DK

If yes, amputation [] Y [] N [] DK or debridement [] Y [] N [] DK

These data were collected by: [] physician interview [] chart abstraction [] other

Your initials: _____