Lemierre Syndrome: Postanginal Sepsis

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Background: **Lemierre syndrome, or postanginal sepsis, was first described in the early part of this century and is characterized by pharyngitis, followed by high fever and rigors, cervical adenopathy, thrombophlebitis of the internal jugular vein, distant abscess formation, and icterus, associated with isolation of *Fusobacterium necrophorum* from blood.**

Methods: **This report describes a case of postanginal sepsis and reviews the medical literature on postanginal sepsis obtained through the MEDLINE data base using *Fusobacterium* as the key search word.**

Results: **The features of Lemierre syndrome have changed little since the original description, though the prognosis has improved dramatically since the development of antibiotics. Appropriate management includes prompt administration of an antibiotic with good anaerobic coverage, drainage of persistent abscesses, and continued antibiotic therapy until radiographic resolution of abscess is achieved.**

Conclusions: **Although Lemierre syndrome is a relatively uncommon disease, the primary care physician needs to be aware of the clinical features and management to treat appropriately. (J Am Board Fam Pract 1995; 8:384-91.)**

Lemierre syndrome is characterized by nasopharyngitis or peritonsillar abscess, followed 4 to 12 days later by high fever and rigors, swelling of the lymph glands below the maxillary angle, tenderness along the lateral aspect of the sternocleidomastoid muscle (representing thrombophlebitis of the internal jugular vein), distant metastatic abscess formation, and icterus or subicterus, associated with isolation of *Fusobacterium necrophorum* (formerly *Bacteroides funduliformis*) from blood. This syndrome was first described by Schottmuller in 1918.[1] Reuben[2] described the features of pediatric cases of postanginal sepsis in 1931, though not associated with *F. necrophorum*. Lemierre[1] is credited with the earliest case review of 20 patients with what is now termed Lemierre syndrome, also referred to as postanginal sepsis or necrobacillosis.[1] Typically, the pharyngitis had resolved before the onset of fever and rigors. The illness was rapidly fatal in 18 of the 20 cases he reported. Finally, Lemierre noted that the striking similarity of the cases enabled diagnosis on purely clinical grounds. He also described septicemia and thrombophlebitis with similar features arising from infection with *F. necrophorum* in other tissues, among them middle ear, gums and dental pulp, pelvic organs, and appendix.

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Fusobacterium necrophorum (previously named *Bacillus funduliformis*, *Bacillus necroformis*, *Bacteroides funduliformis*, *Necrobacterium fundiliforme*, and *Sphaerophorus necrophorus*) is the organism most commonly isolated in cases of postanginal sepsis. *Bacteroides* species and other *Fusobacterium* species, including *F. naviforme* and *F. nucleatum*, have been less commonly associated.

We recently cared for a patient with Lemierre syndrome caused by *F. necrophorum*. The following case report and literature review are presented to alert clinicians about this uncommon infection. We stress that clinical diagnosis is possible, and appropriate medical and surgical management is crucial.

Case Report

A previously healthy 20-year-old male college student complained of an 8-day history of sore throat, productive cough, joint pain, myalgia, malaise, and fever. On examination in the Family Medicine Center, his temperature was 100.8°F (38.2°C), pulse 135 beats per minute, blood pressure 116/64 mmHg, and he appeared moderately ill. Findings on oropharyngeal and lung examinations were normal; the right shoulder was painful with movement but was neither erythematous nor swollen. A chest radiograph, complete blood count, and culture and Gram stain of sputum were ordered. The patient was discharged home with a working diagnosis of viral respiratory infection. A chest radiograph report received later that day described a cavitary lesion in the right

upper lobe and indistinct lesions in both the lower lobes, interpreted by the radiologist as possible cavitations. These findings, in a patient with weight loss, fevers, and cough, suggested possible tuberculosis.

The following day the patient said he might have been exposed to tuberculosis by a classmate who had active disease diagnosed a year before, but the patient had no history of a positive tuberculin skin test. He had experienced an 8-kg weight loss (11 percent of body weight) during his illness. Sputum was obtained for Gram stain, an acid-fast bacilli smear, and culture, and tuberculin and Candida skin tests were performed.

The next day the patient returned to the clinic with erythema, pain, and swelling in the right knee. His temperature was 102.7°F (39.2°C) and he appeared moderately ill. His tonsils and posterior pharynx were beefy red; the patient denied sore throat but had tender anterior cervical lymph nodes. The sputum culture obtained 2 days earlier grew *Staphylococcus aureus* (3+) and normal flora (3+); acid-fast bacilli smears were negative. The patient was admitted to the hospital with a diagnosis of staphylococcal pneumonia and because of clinical signs of tachycardia, spiking fevers, anemia, and multiple abscess sites, possible staphylococcal endocarditis. He was prescribed intravenous nafcillin 1 g every 4 hours. Admission laboratory values included total bilirubin 6.6 mg/dL, lactate dehydrogenase 864 U/L, aspartate aminotransferase 76 U/L, and alanine aminotransferase 55 U/L. His white cell count was elevated at 21,400/mm^3, with 66 percent segmented neutrophils.

The following day his PPD skin test was read as negative, with a positive control. Findings on two-dimensional echocardiography and abdominal sonograms were normal. On hospital day 3 he underwent arthroscopic irrigation and drainage of the right knee, and purulent synovial fluid was sent for culture, Gram stain, aerobic and anaerobic, and acid-fast bacilli smear. Numerous polymorphonuclear cells were seen, but no organisms were identified. Despite appropriate antibiotic therapy (nafcillin and vancomycin) as determined by sensitivity testing of the *S. aureus* isolated from sputum, the patient continued to have fevers to a maximum of 104.2°F (40.1°C), although he was improving and feeling better. The elevations in liver enzymes and bilirubin gradually resolved. The patient continued to lose weight during his hospital stay, totaling 13 kg during his illness (18 percent of body weight). On hospital day 6, a gram-negative bacillus was reported to be growing in anaerobic blood cultures obtained on admission, identified on day 7 as *Fusobacterium*. Antibiotic therapy was changed to ampicillin-sulbactam, 3.2 g every 6 hours. On hospital day 8 *Fusobacterium* species were also isolated from the knee joint aspirate obtained at incision and drainage. The patient's temperature finally returned to normal on hospital day 11. The blood and synovial fluid isolates were identified as *F. necrophorum* on hospital day 14, and the patient was discharged to complete an additional 28 days of intravenous antibiotic therapy at home. At discharge there was no radiographic evidence of the pulmonary abscesses seen on admission. Six weeks later the patient had resumed all his normal activities, had gained 5.4 kg since hospital discharge, and his right knee was normal when examined.

Methods

Review of the Literature

The medical literature from the 1950s to the present was reviewed for case reports of postanginal sepsis caused by *Fusobacterium necrophorum*, using the MEDLINE data base with *Fusobacterium* as the key search word. References were included in this report based on clinical features of Lemierre syndrome (antecedent pharyngitis, evidence of disseminated infection, and isolation of *F. necrophorum* from blood). If a particular clinical feature was not mentioned in the case report, we assumed it was absent. We assumed local thrombophlebitis occurred in patients with evidence of disseminated infection, consistent with earlier case reports; however, we acknowledge that disseminated infection can occur in other infections with bacteremia.

The search was complicated because many names have been given to the syndrome and because the name and taxonomy of *F. necrophorum* have changed several times in this century. For this reason it is possible that historical cases of Lemierre syndrome have been omitted from the review. In addition, several case reports lacked sufficient clinical data for inclusion in the review. Thirty-nine cases were found that met the inclusion criteria; 17 of the cases have been discussed in other literature reviews.

Institutional Review

All positive blood cultures in our institution from January 1986 to November 1993 were reviewed for cases of *F. necrophorum*. Only one other case was found, in a 54-year-old female patient with myelosuppression following chemotherapy for acute myelogenous leukemia. Her clinical presentation did not suggest a diagnosis of postanginal sepsis, and the source of *F. necrophorum* was not described.

Results

A number of cases of *F. necrophorum* bacteremia were described in the medical literature, though they were not associated with Lemierre syndrome. The majority of these isolates were associated with either an oropharyngeal or pelvic source of infection.

Our patient's clinical presentation was representative of most cases of Lemierre syndrome described in the literature and of Lemierre's original description. Clinical data are summarized in Table 1.[3-22] Patients were young, with an average age of 18.9 years; no patient was older than 38 years of age. Male patients made up 75 percent of all cases. All patients were previously healthy with the exception of 4 patients who had had infectious mononucleosis.[6] Two patients had peritonsillar abscesses.[12,15]

By definition, metastatic foci of infection were present in all patients. The more common site of metastatic infection was the lung, and involvement of this organ occurred in 33 of the 39 cases reviewed (85 percent). Pulmonary abscess or necrotizing pneumonia (characterized by multiple abscesses) were reported in 16 patients. These individuals had cough, exertional dyspnea, pleuritic chest pain, and blood-tinged, purulent sputum. Patients rarely had gross hemoptysis. Pulmonary symptoms usually developed suddenly, but in several cases a more indolent course was observed. Four patients developed empyema, which required surgical drainage, and one developed pneumothorax. Sterile pleural effusion was a common manifestation of pulmonary involvement and was found in 20 patients (51 percent).

Ten patients (26 percent) had joint involvement; findings ranged from sterile effusions and simple joint pain to suppurative arthritis. The hip and knee were the joints most frequently involved.[7] Only one case of osteomyelitis was reported.[15] Other sites of metastatic infection were pericardium (two cases)[3,6] and brain, bone, epidural space, myocardium, and skin (one case each). Thrombophlebitis was assumed in all patients, given that all had evidence of metastatic infection, though only 10 cases were radiographically proved.

Jaundice was a relatively common feature and was reported in 19 cases (49 percent). Hepatomegaly was reported in 6 patients (15 percent) and occurred independent of jaundice. Weight loss was reported in 7 patients, 1 of whom lost more than 25 percent of body weight. A number of electrolyte disturbances have been reported, particularly hyponatremia and hypokalemia. An average of 8.2 days was required for defervescence (range 2 to 16 days). The length of antimicrobial treatment ranged from 9 to 128 days.

Seven patients (18 percent) developed shock. Patients with shock had a higher mortality rate and these individuals tended to be slightly older. One of the 7 patients with shock died (14 percent) compared with 2 deaths in the 32 patients without shock (6 percent). One death in the nonshock group was due to cardiac arrhythmia, and microabscesses were found in the myocardium at autopsy. The other death occurred as a result of aspiration following bronchoscopic drainage of a pulmonary abscess.[15]

Two of the fatal cases had cardiac involvement; in both cases, antibiotic therapy was delayed because the illness was initially thought to be of viral origin.[3,9] Antibiotics were instituted only following isolation of an anaerobic organism from blood culture. Whether the delay contributed to the fatal outcomes is not known.

Discussion

Fusobacterium species are gram-negative, anaerobic bacilli that are normal flora in the oropharynx, gastrointestinal tract, and female genital tract. *F. necrophorum* has been long recognized as an animal pathogen, causing disease in a wide range of domesticated animals.[23] The earliest reported cases of *F. necrophorum* septicemia were zoonoses.[24] *F. necrophorum* can cause nasopharyngitis, sinusitis, dental abscesses, peritonsillar abscess, endocarditis, appendicitis, tropical foot ulcers, meningitis, and puerperal sepsis, among other infections. In abdominal and pelvic infections, as well as in pharyngeal infections, *F. necrophorum*

Table 1. Clinical Characteristics of Reported Lemierre Syndrome Patients.

Patient No. (ref)	Age (y), Sex	Underlying Medical Problems	*F. Necrophorum* Isolated from Synovial Fluid	Jaundice	Duration of Fever after Initiation of Treatment (days)	Duration of Antibiotic Treatment (days)	Pulmonary Involvement	Complications	Outcome
1 (3)	20,M	None	No	Yes	Not reported	9	Pulmonary consolidation, pleural effusion	None	Cured
2 (3)	17,M	None	No	Yes	Not reported	Not reported	Pneumonia, empyema	Pericarditis, myocardial microabscesses; died hospital day 6	Died
3 (4)	38,M	None	No	No	5	22	Pneumonia, empyema	Left internal jugular vein thrombophlebitis, posterior thigh abscess	Left eye blindness
4 (5)	19,M	None	No	No	Not reported	Not reported	Pneumonia, adult respiratory distress syndrome	Thrombocytopenia, septic arthritis	Cured
5 (6)	24,F	Infectious mononucleosis	No	No	7	18	Pulmonary abscesses	Shock	Cured
6 (6)	23,M	Infectious mononucleosis	No	No	7	22	Pneumonia, pleural effusion	Hepatosplenomegaly, pericardial effusion, weight loss	Cured
7 (6)	18,M	Infectious mononucleosis	No	No	5	22	Pulmonary edema	Shock, pericardial effusion	Cured
8 (7)	24,M	None	Yes	No	Not reported	42	Pneumonia, adult respiratory distress syndrome	Shock, septic arthritis, acute renal failure	Cured
9 (8)	11,M	None	Yes	No	>7	50	Pulmonary abscesses	Septic arthritis, weight loss, anemia	Cured
10 (8)	15,F	None	No	No	9	14	Pulmonary infiltrates, pleural effusion	Shock, left internal jugular thrombophlebitis	Cured
11 (8)	16,F	None	No	No	2	21	Pulmonary nodules, pleural effusion	Right internal jugular thrombophlebitis, anemia	Cured
12 (9)	20,M	None	No	Yes	7	Not reported	Pneumonia, pleural effusions	Hyponatremia	Cured
13 (9)	17,M	None	No	Yes	Not reported	Not reported	Pulmonary abscess, pleural effusion	Anemia, shock, cardiac arrhythmias, interventricular conduction delay, jugular vein thrombophlebitis, acute renal failure, coagulopathy	Died hospital day 6
14 (10)	20,M	None	No	Yes	Not reported	14	Pulmonary abscesses, empyema	Septic arthritis	Cured
15 (11)	13,F	None	No	No	10	21	Pulmonary abscesses, pleural effusion	Arthralgia, hepatomegaly, heme-positive stools	Cured
16 (12)	18,M	None	No	Yes	Not reported	60	Pulmonary abscesses, pleural effusion	None	Cured
17 (12)	20,F	None	No	Yes	Not reported	Not reported	Pulmonary abscess, pleural effusion	Joint effusions, small bowel obstruction	Cured
18 (12)	21,M	None	No	Yes	5	28	Pneumonia, pleural effusion	Hematuria	Cured
19 (12)	24,F	None	No	Yes	Not reported	21	Pneumonia	Hematuria	Cured
20 (12)	25,M	None	No	Yes	Not reported	Not reported	Pneumonia	None	Cured
21 (13)	18,M	None	No	Yes	13	42	Pulmonary abscesses, pleural effusion	Arthralgias, hepatosplenomegaly, anemia	Cured
22 (14)	12,F	None	No	No	Not reported	Not reported	Pneumonia, pleural effusion	None	Cured
23 (14)	14,M	None	Yes	No	Not reported	128	None	Septic arthritis, weight loss >25% body weight	Cured
24 (14)	9,M	None	Yes	No	Not reported	70	None	Septic arthritis, weight loss	Cured

Continued

Table 1. *Continued*

Patient No. (ref)	Age (y), Sex	Underlying Medical Problems	*F. Necrophorum* Isolated from Synovial Fluid	Jaundice	Duration of *Fever after* Initiation of Treatment (days)	Duration of Antibiotic Treatment (days)	Pulmonary Involvement	Complications	Outcome
25 (14)	17,M	None	No	No	Not reported	49	None	Brain abscesses	Cured
26 (15)	23,M	None	No	No	13	42	Pulmonary abscesses, pleural effusion	Right external jugular thrombophlebitis, pneumothorax	Cured
27 (15)	13,F	None	Yes	Yes	10	42	Pulmonary abscesses	Septic arthritis, osteomyelitis	Cured
28 (15)	18,F	None	No	No	Not reported	Not reported	Necrotizing pneumonia, pleural effusion, empyema	Left internal jugular vein thrombophlebitis, hepatomegaly	Died hospital day 9, complications of bronchoscopy
29 (15)	14,M	None	No	No	Not reported	Not reported	Pneumonia	None	Cured
30 (16)	15,M	None	No	Yes	14	14	None	Weight loss, left internal jugular vein thrombophlebitis	Sensorineural hearing loss
31 (17)	15,F	None	No	Yes	>5	36	Pneumonia, pleural effusion	Weight loss	Cured
32 (18)	20,M	None	No	Yes	8	42	Pulmonary abscesses, pleural effusion	Weight loss	Cured
33 (18)	20,M	None	No	Yes	16	42	Pneumonia, pleural effusion	None	Cured
34 (19)	24,M	None	No	No	Not reported	10	Pulmonary abscesses, pleural effusion	Shock, thrombophlebitis	Cured
35 (20)	20,M	None	No	Yes	4	Not reported	None	Arthritis, jaundice, anorexia	Cured
36 (20)	23,M	None	No	Yes	7	Not reported	Pulmonary abscesses, pleural effusion	Shock, thrombophlebitis right femoral vein	Cured
37 (21)	16,F	None	No	No	18	110	Pneumonia, pleural *effusions*	None	Cured
38 (21)	12,M	None	No	No	10	77	None	Diffuse encephalopathy, *arthritis, hematuria,* hepatosplenomegaly	Cured
39 (22)	15,M	Lumbar spondylolysis	No	No	Not reported	42	None	Epidural abscess	Cured
(Current case)	20,M	None	Yes	Yes	11	42	Necrotizing pneumonia	Septic arthritis, hepatomegaly, anemia, *weight loss*	Cured

can cause suppurative thrombophlebitis of the local vasculature.

Unlike other gram-negative septicemias, which tend to occur in the chronically ill or elderly, Lemierre syndrome typically affects young, healthy persons. The relatively low mortality rate in this syndrome could be the result of the youth and relative health of those affected. Similar age-dependent differences in mortality were described by Bodner, et al.[25] and Gelb and Seligman[26] in their reviews of Bacteroidacae septicemias.

Pathophysiology

The rarity of this syndrome suggests that a number of factors must be present to permit the development of invasive disease. The role of antecedent pharyngeal infection in the pathogenesis of this syndrome deserves some discussion. *F. necrophorum* is a commensal in the oropharynx and is known not to penetrate mucosal surfaces well; it could become pathogenic only in the setting of an altered host-defense mechanism. Alterations in the pharyngeal mucosa caused by viral or bacterial pharyngitis might play such a role in development of the syndrome.[27] That pharyngitis precedes the development of septicemia by 4 to 8 days and has often resolved by the time septicemia develops supports this hypothesis. In addition, synergy has been demonstrated between *Fusobacterium* species and aerobic bacteria in abscess formation in ani-

mal models.[28,29] Whether a similar synergistic relation plays a role in the pathogenesis of Lemierre syndrome is unclear.

Another possible factor in development of postanginal sepsis is acquisition of a virulent strain of organism, and a number of potential virulence factors have been described. The lipopolysaccharide in the cell wall of pathogenic strains has strong endotoxic properties.[30] *Fusobacterium* isolates recovered from patients with invasive disease are more likely to be encapsulated strains.[28] Bovine strains of *F. necrophorum* differ considerably in their ability to cause invasive disease.

Virulent strains can activate human platelets in vitro, a property lacking in nonvirulent strains and in cell-free culture broth.[31] Microcirculatory thrombus formation following inoculation with a virulent strain of *F. necrophorum* has been shown in an animal model.[32] Thrombus formation is central in the pathophysiology of Lemierre syndrome. Pharyngeal infection causes thrombosis of the tonsillar veins and sometimes of the larger neck veins, particularly the internal jugular, which results in embolization of the infected material and development of metastatic foci of infection that characterize the syndrome.

The production of a heat-stable exotoxin (leucocidin), which is believed to be responsible for the inflammatory response, occurs in virulent strains but not nonvirulent strains. Additionally, virulent strains possess a heat-labile exotoxin that is cytopathic for porcine kidney cells.[33] The high spiking fevers and the considerable weight loss seen in several patients suggest possible activation of tumor necrosis factor or a similar mediator by *F. necrophorum*.

Diagnosis

Our patient had fever, multiple lung nodules, abundant growth of *S. aureus* from sputum, weight loss, and septic arthritis, all of which led to the presumptive diagnosis of right-sided endocarditis. The literature review found 4 other patients treated initially with antistaphylococcal antibiotics for presumed staphylococcal endocarditis. All of these individuals gave a history of pharyngitis, but this feature was thought not to be important to the presenting illness. A history of recent sore throat is crucial in diagnosis, but as mentioned previously, the pharynx often appears normal by the time sepsis develops. This latent period can range from a few days to 3 weeks.[34] The oropharynx can appear erythematous or exudative, and peritonsillar abscess has also been infrequently reported.

Diagnosis of suppurative thrombophlebitis is lacking in the majority of cases. Of the patients described in the literature review, only 25 percent had radiographically or surgically proven thrombophlebitis. Although palpation of a fusiform mass along the sternocleidomastoid is the classic presentation, tenderness along the lateral side of the sternocleidomastoid has also been described. The swelling and tenderness can be quite subtle and might be ascribed to cervical lymphadenitis. Definitive diagnosis of thrombophlebitis requires both a high index of suspicion and specific diagnostic testing; computerized tomographic scanning and sonography are the diagnostic tests most frequently employed. Sonography is generally preferred, though no systematic comparison of the sensitivity and specificity of imaging techniques has been undertaken.[16,35] The role of magnetic resonance imaging in this setting has not been addressed.

Early in the illness the chest radiograph might show only diffuse interstitial infiltrates with or without pleural effusion. Single or multiple nodular infiltrates are also characteristic pulmonary findings. These lesions typically progress to cavitation and usually resolve with prolonged antibiotic therapy alone. Empyema can develop, though usually much later in the course of illness.

The difficulty of isolating and identifying the causative organism plays a role in delayed diagnosis. Fusobacteria are fastidious organisms and can be difficult to culture and characterize. In a number of the cases reviewed, anaerobic organisms were not identified until after 5 to 8 days of incubation, which can delay institution of appropriate antibiotic treatment. If found on sputum Gram stain, *Fusobacterium* species would not be recognized as potential pathogens, as they are part of normal upper airway flora. The only respiratory tract secretions suitable for anaerobic culture are those obtained by transtracheal aspiration, so *F. necrophorum* is rarely identified from sputum.

Treatment

There are several important issues with regard to treatment of this syndrome. A major issue is antibiotic choice. Studies of antibiotic sensitivity

have shown widely variable results, with a recent report of 22 percent of *F. necrophorum* isolates producing β-lactamase.[36] Some of the variability could result from the lack of consistent methods for antibiotic sensitivity testing of anaerobes. Clindamycin, metronidazole, antipseudomonal penicillins, and ampicillin-sulbactam offer good coverage for anaerobes such as fusobacteria.[37]

Delayed defervescence and progression of illness despite appropriate antibiotic therapy are features common to the cases reviewed. In a number of cases, prolonged fever and progression of disease have been described as treatment failure, and clinicians have changed antibiotic therapy on that basis. It is not clear whether these cases represent infection with resistant organisms or simply the natural history of appropriately treated postanginal sepsis. The prolonged fever has been attributed to poor antibiotic penetration of loculated abscesses and is a common feature of other anaerobic abscesses.[38]

It is important to note that delayed antibiotic treatment was associated with poorer outcomes in the cases described in the literature review. Two deaths were reported in the 8 patients in whom antibiotic therapy was delayed more than 4 days (25 percent) compared with 1 death in 29 patients who were treated promptly (3 percent). Both patients with delayed therapy had cardiac involvement, which was thought to be of viral origin, and they were treated supportively until the results of anaerobic culture were known, on day 4 and day 5, respectively.[3,9]

Given the role of suppurative thrombophlebitis in both the pathophysiology of the syndrome and in delayed response to therapy, systemic anticoagulation has been advocated as an adjunct to antibiotic therapy.[8] The authors cite the potential for faster resolution of the thrombophlebitis and bacteremia, thus limiting the development of new metastatic foci. The limited clinical data that support this position are anecdotal.[4,8] Finegold and colleagues[39] have questioned the use of anticoagulants in this setting, citing the potential risks of serious hemorrhage and extension of infection. In the preantibiotic era, surgical ligation of the internal jugular vein was the treatment of choice, but this surgery has been only rarely used after the development of penicillin and is now reserved for those who fail conventional therapy.

Surgical drainage of purulent fluid collections can also be important in managing postanginal sepsis. This recommendation is based on suggested management of other anaerobic abscesses.[37] Necrotizing pneumonia and solitary pulmonary abscess often resolve simply with prolonged antibiotic therapy, but drainage is required for those abscesses that do not respond. Open drainage of anaerobic empyema is associated with decreased morbidity and mortality compared with thoracentesis and is the treatment of choice for persistent fluid collections.[40] Early irrigation and drainage of septic joints are crucial in preserving joint function.

The appropriate length of antibiotic treatment is difficult to determine. As noted in Table 1, the duration of antibiotic therapy ranged from 9 to 128 days. Complete radiographic resolution of pulmonary findings on chest radiographs has been suggested as the end point for therapy for pulmonary abscesses caused by other anaerobes. Bartlett and Finegold[40] cited several cases in which relapse has occurred as a result of premature termination of antibiotic therapy. In cases without either pulmonary abscess or necrotizing pneumonia, the end point for antibiotic therapy is difficult to ascertain.

Summary

The diagnosis of postanginal sepsis should be considered in a young, previously healthy person with high fever, disseminated infection, and cervical adenopathy who had evidence of an earlier episode of pharyngitis. Because of difficulties associated with isolation and speciation of *Fusobacterium*, the diagnosis should be considered even in the absence of isolation of the organism from blood culture. *F. necrophorum* is generally sensitive to either penicillin or clindamycin, and persistent fever despite appropriate antibiotic coverage is common. Surgical drainage of abscess cavities should be considered if there is inadequate response to antibiotic therapy alone, and prompt drainage of septic joints is crucial in preserving joint function. Delays in diagnosis and treatment have been associated with excess mortality in this literature review, though overall mortality has been greatly reduced since the advent of antibiotic therapy.

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