

Oropharyngeal *Bacteroides Melaninogenicus* Infection with Septicemia: Lemierre's Syndrome

George W. Shannon, MD, Clarence V. Ellis, MD, and William P. Stepp, MD
Jackson, Tennessee

In 1936 Lemierre¹ reviewed the then-current knowledge about anaerobic septicemia. The clinical syndrome described in his paper was caused by oropharyngeal anaerobic infection with local and systemic spread. Common findings due to these infections included peritonsillar, tonsillar, and internal jugular venous thrombophlebitis with neck pain, septic pulmonary emboli, rigors, icterus, elevated urinary urobilinogen, albuminuria, and joint involvement ranging from simple arthralgias to suppurative arthritis. *Bacteroides* septicemia in the preantibiotic era most commonly arose from infections in the oropharynx, middle ear, and adjacent structures.² Reviews of bacteroides septicemia following the development of antibiotics have indicated a much lower incidence of this infection derived from oropharyngeal sources.³⁻⁸

The precipitous drop in the incidence of bacteroides septicemia derived from oropharyngeal flora is presumed to have resulted from the early use of penicillin in the treatment of upper respiratory tract infections.² The following case report illustrates a clinical situation in which oral penicillin was given to an adolescent with a sore throat, but was soon discontinued when the throat culture was negative for β -hemolytic streptococcus. This patient then developed a syndrome secondary to

Bacteroides melaninogenicus septicemia similar to that described by Lemierre.

Case Report

A 14-year-old black girl was first seen at the Jackson-Madison County General Hospital emergency room on February 11, 1979, with the chief complaint of a sore throat. Physical examination in the emergency room revealed an exudative pharyngitis. A three-day prescription for oral penicillin was given after a throat culture was obtained, and the patient was instructed to call back for the results of the culture. The culture was reported negative for β -hemolytic streptococcus, and the oral penicillin was not continued. Five days later the patient experienced the onset of epigastric pain without nausea or vomiting and returned to the emergency room. Physical examination at that time showed an oral temperature of 39.4°C, a normal ear, nose and throat examination except for tender swelling in the area of the right sternocleidomastoid muscle, and diffuse abdominal tenderness without localizing signs and with normal bowel sounds. At that time, a chest x-ray film showed a questionable bibasilar pneumonitis, but the lung fields were otherwise normal. Laboratory evaluation included a white cell count of 21,000/mm³ with a marked shift to the left, and an elevated serum amylase, bilirubin, and lactic dehydrogenase (Figure 1). Arterial blood gases on room air showed hypoxemia (PO₂ of 59 torr).

The patient was admitted to the hospital, and blood, throat, and urine cultures were obtained prior to the initiation of antibiotic therapy with ampicillin and gentamycin. Acute and convalescent

From the Departments of Family Medicine, Internal Medicine and Pediatrics, University of Tennessee College of Medicine, Jackson, Tennessee. Requests for reprints should be addressed to Dr. George W. Shannon, The Medical Center, 710 Center Street, PO Box 951, Columbus, GA 31902.

sera during the hospitalization were negative for parainfluenza type I and II, *Mycoplasma pneumoniae*, adenovirus, respiratory syncytial virus, *Legionella pneumophila*, and A-Texas, B-Hong Kong, and A-USSR influenza viruses. Monospot tests, done on hospital days 2 and 11, were both negative.

On the third hospital day, the three admission blood cultures were reported positive for *Bacteroides* organisms. Species identification of these cultures was requested. The antibiotics were switched to intravenous penicillin and chloramphenicol (Figure 1). Pelvic and abdominal ultrasound evaluations done on the third hospital day were within normal limits. Repeat chest x-ray examination on the seventh hospital day showed the presence of septic pulmonary emboli. Gallium scanning was performed on the same day because of persistence of fever and the then-unknown source of the bacteroides septicemia. The scan was positive for an area coinciding with the right jugular vein (Figure 2). Surgical exploration of this area was performed on the eighth hospital day in an attempt to drain any abscesses. Examination at surgery was remarkable only for the presence of an inflamed jugular vein, which was not opened or ligated.

Species identification of the blood cultures was reported on the ninth hospital day as *Bacteroides melaninogenicus*. The patient's tachypnea, arterial hypoxemia, and abdominal discomfort gradually improved over the next three days.

The remainder of the clinical course and laboratory studies are presented in Figure 1. Other unusual manifestations included the development of a Coombs-positive hemolytic anemia, the development of the syndrome of inappropriate secretion of antidiuretic hormone (SIADH), persistent hyperamylasemia without an elevation of serum lipase, and a transient period of areflexia thought by the neurological consultant to be consistent with transverse myelitis.

The patient received a full ten-day course of intravenous penicillin and chloramphenicol. The penicillin was continued orally for eight additional days. After two weeks, follow-up examination in the office showed complete resolution of the clinical syndrome including clearing of the chest x-ray findings, the abnormalities in blood chemistry, the areflexia, and the tenderness in the right neck. Subsequent follow-up examinations have shown the patient to be in good health.

Comment

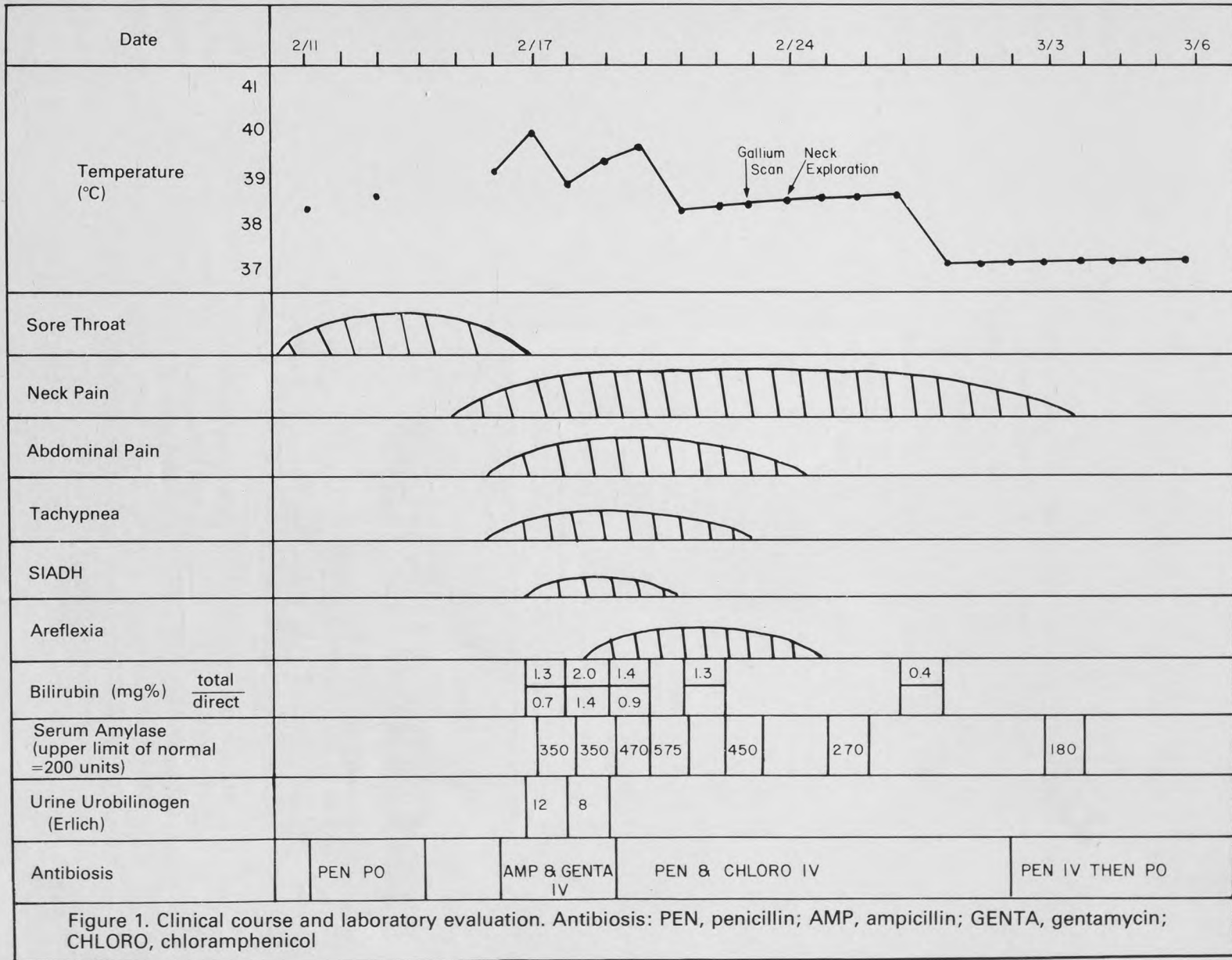
Bacteroides melaninogenicus is almost never found colonizing the oropharynx of preschool children, but its presence becomes increasingly common with age until it is almost universally found in adolescents.^{9,10} *B melaninogenicus* has been reported as a pathogen in abdominal surgical infections¹¹ as well as having been a common pathogen in infections derived from the oropharynx in the preantibiotic era.

On initial presentation to the emergency room, the patient was felt to have a β -hemolytic streptococcus infection; however, the physician elected to begin oral therapy with only a three-day prescription while awaiting the results of the culture. When the culture was reported negative, the physician elected not to continue the penicillin therapy. The discontinuation of penicillin was followed by the development of a malignant clinical picture consistent with the syndrome described by Lemierre.¹ The patient manifested all the classical phenomena seen in bacteroides septicemia originating from an oropharyngeal source. The clinical course included jugular vein thrombophlebitis (demonstrated both by gallium scan and at surgery), septic pulmonary emboli, albuminuria, elevated urinary urobilinogen levels, icterus, rigors, and the demonstration of *B melaninogenicus* in all blood cultures drawn prior to initiation of in-hospital antibiotic therapy.

Several phenomena not previously described were observed, including Coombs-positive hemolytic anemia, hyperamylasemia of unknown cause (thought in retrospect to be due to salivary gland involvement or irritation), and electrolyte abnormalities consistent with SIADH. The "transverse myelitis" may have been secondary to either the infection or to the antibiotic therapy.¹² The bacteroides-induced hemolytic anemia has been previously reported.^{13,14} This is the first report, to the authors' knowledge, that has demonstrated Coombs positivity.

The syndrome described by Lemierre apparently has become a very rare phenomenon since the development of antibiotics and their use in the treatment of upper respiratory tract infections.² A number of clinical points are of help to the physician in the diagnosis of this syndrome. Internal

Continued on page 163



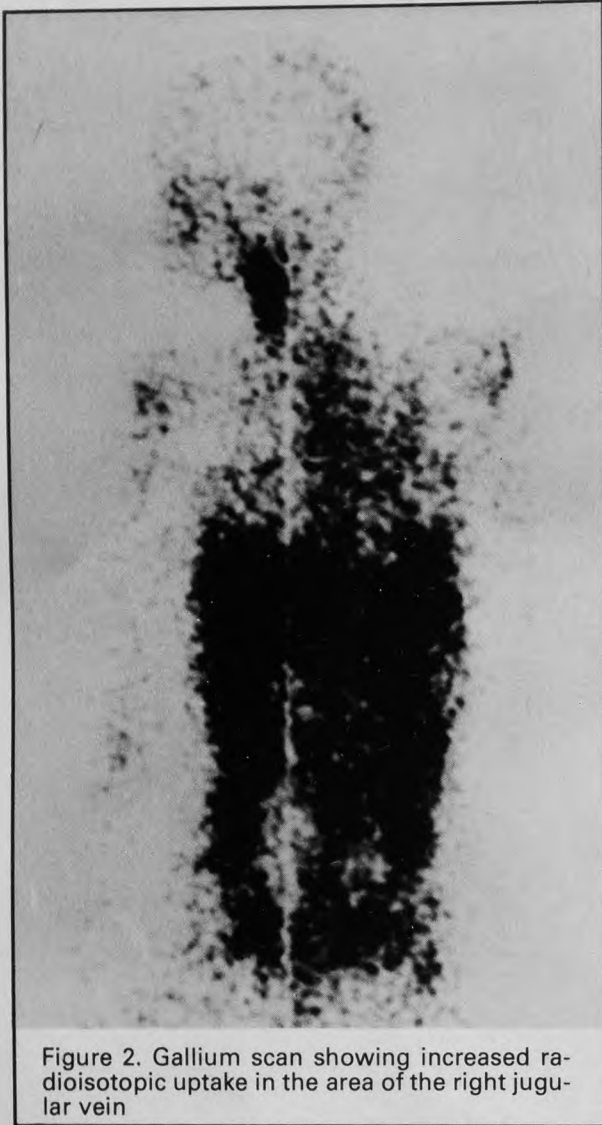


Figure 2. Gallium scan showing increased radioisotopic uptake in the area of the right jugular vein

jugular vein thrombophlebitis can be easily confused with painful, swollen cervical adenopathy commonly seen in upper respiratory tract infections such as mononucleosis and streptococcal pharyngitis. The absence of palpable cervical adenopathy, however, and the presence of tenderness occurring in a linear fashion along the course of the vein are of help in the differentiation by physical assessment of patients presenting with these problems. The presence of rigors, which characteristically occur on the fourth or fifth day after the onset of upper respiratory tract symptoms, are indicative of anaerobic oropharyngeal infections. The presence of tachypnea due to sep-

tic pulmonary emboli indicate a systemic infection derived from septic thrombophlebitis. Anticoagulation is often difficult in these cases, presumably because of the production of a "heparinase" by the *Bacteroides* organisms.

It is important for physicians to be aware that bacteroides oropharyngeal infections may present with a clinical picture that can be initially confused with streptococcal pharyngitis or infectious mononucleosis. Patients should be considered candidates for Lemierre's syndrome when fever is persistent or when their clinical picture deteriorates with negative cultures for β -hemolytic streptococcus and negative serologies for mononucleosis. Penicillin therapy is quite effective in the treatment of *B melaninogenicus* infections starting in the oropharynx. The development of unilateral or bilateral neck swelling and tenderness consistent with jugular venous thrombophlebitis, the presence of rigors on the fourth or fifth day after the onset of upper respiratory tract symptoms, elevated urinary urobilinogen, icterus, and albuminuria are clues that should lead the physician to consider the diagnosis of Lemierre's syndrome.

References

1. Lemierre A: On certain septicemias due to anaerobic organisms. *Lancet* 1:701, 1936
2. Gunn AA: *Bacteroides* septicemia. *J R Coll Surg Edinb* 2:41, 1956
3. Gelb AF, Seligman SJ: *Bacteroidaceae* bacteremia: Effect of age and locus of infection upon clinical course. *JAMA* 212:1038, 1970
4. Bodner SJ, Koenig MG, Goodman JS: Bacteremic bacteroides infections. *Ann Intern Med* 73:538, 1970
5. Felner JM, Dowell BR: *Bacteroides* bacteremia. *Am J Med* 50:787, 1971
6. Wilson WR, Martin WJ, Wilson JA: Anaerobic bacteremia. *Mayo Clin Proc* 46:639, 1972
7. Chow AW, Guze LB: *Bacteroidaceae* bacteremia: Clinical experience with 112 patients. *Medicine* 53:93, 1974
8. Olsen H: *Bacteroides* bacteremia: A clinical and bacteriological analysis of 51 patients. *Scand J Infect Dis* 8:107, 1976
9. Bailit HL, Baldwin DC, Hunt EE: The increasing prevalence of gingival *Bacteroides melaninogenicus* with age in children. *Arch Oral Biol* 9:435, 1964
10. Kelstrup J: The incidence of *Bacteroides melaninogenicus* in human gingival sulci and its prevalence in the oral cavity at different ages. *Periodontics* 4:14, 1966
11. Weiss C: The pathogenesis of *Bacteroides melaninogenicus* and its importance in surgical infections. *Surgery* 13:683, 1943
12. Grouse C, Henle W, Henle G, Feorino PM: Primary Epstein-Barr virus infections in acute neurologic diseases. *N Engl J Med* 292:392, 1975
13. Tynes BS, Frommeyer WB: *Bacteroides* septicemia: Cultural, clinical and therapeutic features in a series of 25 patients. *Ann Intern Med* 56:12, 1962
14. McVay LD, Sprunt DA: *Bacteroides* infections. *Ann Intern Med* 35:56, 1962

Continued on page 169