Splenic Abscess Caused by Propionibacterium Acnes

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A 59-year-old male diabetic was admitted with an acute myocardial infarction and had recurrent Propionibacterium acnes bacteremia. Fifteen months after the initial admission a splenectomy was required for removal of a large splenic abscess caused by P. acnes. Although this organism represents part of the normal skin flora, its presence in blood cultures requires serious evaluation since it may signify clinical disease, not merely contamination of blood cultures by skin flora.

INTRODUCTION

Both since its earliest descriptions by Hippocrates, and until recently, the diagnosis of splenic abscess has generally been made postmortem. Reid and Lang reported an autopsy incidence for splenic abscess of 0.4 percent in the largest combined series published during the pre-antibiotic era [1]. Such abscesses were generally attributed to splenic trauma and/or bacterial seeding from a contiguous or distant focus of infection. Accurate diagnosis of splenic abscess can be even more difficult when such abscesses are associated with an unusual pathogen.

We report a case of bacteremia and splenic abscess caused by the anaerobic diphtheroid, Propionibacterium acnes. The normal habitats for P. acnes are skin, mucous membranes, and the gastrointestinal tract. Although P. acnes may contaminate cultures taken from these sites, failure to consider its potential pathogenicity could lead to erroneous decisions in patient management. This case illustrates the need to evaluate clinical isolates of P. acnes with this potential in mind, particularly isolates which are obtained from blood cultures.

CASE REPORT

A 59-year-old male with adult onset insulin-requiring diabetes mellitus was admitted to the West Haven VA Medical Center, West Haven, CT, in April 1978 for substernal chest pain, nausea, vomiting, and pulmonary edema. On admission he was afebrile and physical examination was remarkable for jugular venous distention, bilateral rales, and hepatosplenomegaly. Electrocardiogram documented an acute anterior wall myocardial infarction. His course was complicated by ventricular ectopy, post-infarction angina, and he required pulmonary intubation for intractable pulmonary edema.
On the eighth hospital day the patient developed fever (100.7°F) and leukocytosis (WBC 16.9 cells/μl; differential showing 89 PMNs, 5 lymphocytes, and 6 monocytes). Urine cultures grew more than 10⁸ enterococci per ml and he was treated for five days with parenteral ampicillin (4 gm/day) for a presumed urinary tract infection. Blood cultures were negative. On the eleventh hospital day the patient was again febrile (101°F) with episodes of left upper quadrant, neck, and shoulder pain. Urine cultures and four venipunctures were performed for blood cultures which did not grow organisms. Chest X-ray showed a left pleural effusion; uncomplicated thoracentesis demonstrated a sterile transudate. Technetium 99mTc sulfur colloid liver-spleen scan and ultrasound examination showed the spleen to be moderately enlarged (approximately 16 cm in length) with a lobulated 11 cm cystic deformity involving its superior region and compressing adjacent tissue. Gallium⁶⁷ citrate scan showed no abnormalities. These findings were considered to be consistent with a splenic cyst. Because of recurrent fevers, rigors, nausea, and vomiting, eight venipunctures were performed for blood cultures over a twelve-hour period. Seven to ten days later, four were positive for P. acnes and thought to represent skin contamination. Echo-cardiogram did not demonstrate valvular vegetations. Fever resolved following a parenteral course of ampicillin (6 gm/day for eleven days) and gentamicin (180 mg/day for eight days). He was discharged on the thirty-sixth hospital day.

Over the next 15 months, he was routinely seen as an outpatient with complaints unrelated to his previous hospitalization. In June 1979 he again noted chills, nausea, and vomiting with recurrent left upper quadrant pain. He was readmitted and chest X-ray again showed a left pleural effusion. Liver-spleen scan showed a small decrease in the size of the 16 cm spleen and its upper pole defect, compared to fifteen months previously. He was intermittently febrile to 101–102°F rectally. Several blood cultures (13/16) and pleural fluid were positive for P. acnes. A gallium⁶⁷ citrate scan now showed increased uptake in a peripheral pattern around the splenic defect which was felt to represent a splenic abscess. He was treated with parenteral ampicillin. Laparotomy revealed an abscess-containing 405-gram spleen adherent to the anterior abdominal wall and the diaphragm. Gram stain of splenic tissue, splenic bed, and purulent abscess drainage showed many polymorphonuclear leukocytes and gram-positive bacilli. Cultures grew P. acnes. No other organisms were obtained. The spleen was removed and histological examination showed multiple foci of splenic necrosis with both leukocytes and rod-shaped bacteria evident (Fig. 1) on Brown and Brenn stain. Gomori silver stain showed no fungi. The patient was treated with a fourteen-day course of parenteral ampicillin (6 gm/day) followed by a fourteen-day course of oral ampicillin (4 gm/day).

**BACTERIOLOGY**

Blood cultures were performed in 45 ml supplemented peptone broth Becton-Dickenson Vacutainer culture tubes (Becton-Dickson Co., Rutherford, NJ). During the two admissions, 14 of 18 anaerobic tubes grew a thin gram-positive bacillus. Cultures were identified as positive five to ten days after subculture on blood agar plates incubated anaerobically. Purulent drainage and tissue from the splenic abscess and the splenic bed grew the same organism anaerobically. All isolates showed positive reactions for indol, glucose, gelatin, glycerol, mannose, and catalase. Negative reactions were shown for urea, mannitol, lactose, sucrose, xylose, arabinose, esculin, cellobiose, melezitose, raaffinose, sorbitol, rhamnose, and
SPLENIC ABSCESS CAUSED BY PROPIONIBACTERIUM ACNES

FIG. 1. Tissue gram stain of infected spleen showing both tissue necrosis and a rod-shaped bacterium (arrow) identified as Propionibacterium acnes.

trehalose. This characterization was consistent with an identification pattern for P. acnes. Using a broth-disk method all isolates of the organism were sensitive to chloramphenicol, clindamycin, penicillin, carbenicillin, and cefoxitin.

DISCUSSION

Our patient had recurrent Propionibacterium acnes bacteremia and a splenic abscess. At the time of his second admission, 15 months following an initial episode of P. acnes bacteremia, both splenomegaly and a left-sided pleural effusion were also evident. P. acnes was again isolated from several blood cultures, from pleural fluid, and from a splenic abscess following surgical removal of his spleen.

The etiology of his initial bacteremia remains unclear. The patient may have inoculated himself with P. acnes during insulin administration, resulting in an inapparent subcutaneous focus of infection. An infected intravenous catheter site during his first admission could have served as an initial focus of infection; it is unlikely that bacteremia with a low-grade pathogen would result in abscess formation within a normal spleen. However, the presence of a splenic cyst or hematoma, which was suggested on ultrasonic examination, might have predisposed to abscess formation in the event of bacteremia from such a source.

Apart from C. diphtheriae, diphtheroids were rarely considered pathogenic for man until two decades ago. Several reports have demonstrated that other diphtheroids might be associated with serious infections including endocarditis, meningitis, osteomyelitis, suppurative adenitis, pneumonia, and lung abscess [2,3]. In general, serious clinical diphtheroid infections can be associated with some identifiable predisposing factor such as congenital or acquired heart disease, valvular prosthesis, anatomic abnormalities of the central nervous system, recent surgery, vasculitis, or diabetes mellitus [2]. Although 31 of 34 diphtheroid isolates causing endocarditis were found to be aerobic by Johnson and Kaye, they also noted that microaerophilic and anaerobic diphtheroids such as the Propionibacteria were associated with infections other than endocarditis [2].

The anaerobic Propionibacteria are pleomorphic, gram-positive bacilli which, like Corynebacteria, may be arranged in palisades or “Chinese characters.” Although anaerobic, these propionic acid-producing bacteria were traditionally grouped with aerobic Corynebacteria until 1972 when Johnson and Cummins separated three species of anaerobic coryneforms on the basis of cell wall antigens and DNA homology studies [4]. These species are designated Propionibacterium acnes, P.
granulosum, and P. avidum. P. acnes has been associated with post-surgical infections of implanted prostheses, especially prosthetic valves, ventricular shunts, and orthopedic devices [5,6]. Although a relatively indolent course has been reported in chronic meningitis associated with P. acnes, at least one patient with P. acnes shunt infection also developed immune-complex glomerulonephritis [7]. A recent report of hepatic botryomycosis caused by P. acnes in a patient receiving corticosteroid therapy emphasizes its role as a potentially serious pathogen [8]. Our report emphasizes the broader clinical spectrum of disease associated with P. acnes bacteremia, which may include a soft tissue focus not associated with a prosthetic nidus of infection.

During our patient’s first admission, four of eight sets of blood cultures were positive for P. acnes. These positive cultures represented separate samples drawn at different times during the day, yet these isolates were subsequently dismissed as contaminants. This misinterpretation resulted from the assumption that P. acnes was merely a part of the normal skin flora which had contaminated the blood cultures. An additional problem in evaluating such infections has been noted by Johnson and Kaye and was also seen in this case. In their review of diphtheroid infections they note a median of five days of incubation, and as long as two weeks, was required for growth of some isolates [2]; this may also contribute to the isolates inappropriately being considered contaminants. At the time of this patient’s second hospitalization, 15 months later the picture was more complete. Nine of 16 blood culture bottles taken over a four-day period grew P. acnes, as did pleural fluid, splenic tissue, and splenic abscess drainage, all with identical antimicrobial sensitivity patterns.

The classification of what were formerly termed diphtheroids into the aerobic Corynebacteria and the anaerobic Propionibacteria also has therapeutic significance. Murray et al. recently reported the antimicrobial sensitivity patterns for eighteen aerobic diphtheroids causing prosthetic valve endocarditis. Most showed intermediate or high-level resistance to penicillin, ampicillin, and oxacillin [9]. In contrast, Wang et al. examined sensitivity patterns of 96 strains of P. acnes and found the penicillins among the most active antibiotics against these organisms [10].

The anaerobic diphtheroids or Propionibacteria require the same careful evaluation as any other organism isolated from multiple blood cultures. The occurrence of bacteremia and a soft tissue abscess caused by Propionibacterium acnes in this patient emphasizes the potential pathogenicity of this organism even in clinical situations unassociated with an endovascular focus of disease such as a prosthesis.

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REFERENCES

1. Reid SE, Lang SJ: Abscess of the spleen. Am J Surg 88:912-917, 1954
2. Johnson WD, Kay D: Serious infections caused by diphtheroids. Ann NY Acad Sci 174:568-576, 1970
3. Kaplan K, Weinstein L: Diphtheroid infections of man. Ann Intern Med 70:919-929, 1969
4. Johnson JL, Cummin CS: Cell wall composition and deoxyribonucleic acid similarities among the anaerobic coryneforms, classical propionibacteria, and strains of Arachnia Propionica. J Bacteriol 109:1047-1066, 1972
5. Skinner PH, Taylor AJ, Coakham H: Propionibacteria as a cause of shunt and postneurosurgical infections. J Clin Pathol 31:1085–1090, 1978
6. Petrini B, Welin-Berger T, Nord CE: Anaerobic bacteria in late infections following orthopedic surgery. Med Microbiol Immunol 167:155–159, 1979
7. Beeler BA, Crowder JG, Smith JW, et al: Propionibacterium acnes: Pathogen in central nervous system shunt infection. Am J Med 61:935–938, 1976
8. Schlossberg D, Keeney GE, Lifton LJ, et al: Anaerobic botryomycosis. J Clin Microbiol 11:184–185, 1980
9. Murray BE, Karchmer AW, Moellering RC: Diphtheroid prosthetic valve endocarditis: A study of clinical features and infecting organisms. Am J Med 67:838–848, 1980
10. Wang WLL, Everett ED, Johnson M, et al: Susceptibility of Propionibacterium acnes to seventeen antibiotics. Antimicrob Agents Chemother 11:171–173, 1977