Fusobacterium nucleatum Pericarditis

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A pericardial effusion was diagnosed by echocardiography in a 49-year-old man who suffered acute cough, orthopnea, and chest pain. Because of a positive tuberculin skin test, mycobacteria were initially suspected as the cause of the pericarditis. The patient was therefore treated with antituberculosis drugs. The pericardial effusion failed to resolve, however, and pericardiectomy was performed. Cultures of the pericardial fluid yielded pure Fusobacterium nucleatum growth. The patient responded to antibiotic therapy and was in good health 3 weeks after being discharged from the hospital. This represents the first report of F. nucleatum pericarditis.

Anaerobic bacteria, as well as facultative bacteria, have been described as etiological agents in infectious endocarditis (3). Among the most common organisms incriminated in anaerobic endocarditis are Propionibacterium acnes and Bacteroides fragilis. Other anaerobic etiologies include Clostridium spp., Bacteroides spp., Fusobacterium spp. and Peptostreptococcus sp. The most common portal of entry in patients with endocarditis regardless of etiology, is the oropharynx. The endocarditides precipitated by anaerobes also appear in a significant number of patients with gastrointestinal disease.

Infectious pericarditis, too, has been described as being caused by both facultative bacteria (2, 4, 6, 8, 9, 12, 13) and anaerobic bacteria (10, 11). Microaerophilic species have also been found in cases of infectious pericarditis (1, 5, 7). This report describes the first documented case of infectious pericarditis caused by Fusobacterium nucleatum.

CASE REPORT

A 49-year-old man was admitted to the emergency room at the University of Texas Medical Branch Hospitals, Galveston, with a 4-day history of cough, orthopnea, diarrhea, and progressively worsening epigastric and substernal pain. He had been working steadily in a warehouse at the time of admission and had been well until the onset of his present illness. His medical history was unremarkable except for alcohol abuse.

Physical examination at the time of admission revealed an oral temperature of 37.8°C; the blood pressure was 120/80 mmHg (without paradox), the pulse rate was 80/min, and the respiratory rate was 36/min. Oral examination revealed severe dental caries and pyorrhea. Neck examination showed jugular venous distention at 30°. Heart sounds were distant, and there was a three-component friction rub. Although rhythm was regular, both an S3 and an S4 were present. A hepatogallop reflex was present. The liver was tender and extended 5 cm below the right costal margin. All other physical characteristics at the time of admission were normal.

Laboratory results for the patient at admission revealed a hemoglobin of 9.8 g/100 ml, a hematocrit of 28.6%, and a leukocyte count of 12,800/mm3, with 75% segmented neutrophils, 3% bands, 14% lymphocytes, 7% monocytes, and 1% basophils. Electrolytes were within normal limits, except for sodium at 134 meq/dl.

A purified protein derivative test with 5 tuberculin units (5-TU) was positive at 24 mm. Hepatitis B surface antigen was not present. Chest X-ray revealed pulmonary edema and cardiomegaly. An electrocardiogram showed S-T elevation in leads I through III and V6 through V4. An echocardiogram demonstrated a moderately large posterior pericardial effusion.

The patient was admitted to the University of Texas Medical Branch Hospitals and was started on indomethacin (50 mg every 8 h). On hospital day 2 he had a temperature of 39.4°C and shaking chill; over the next 48 h he became afebrile. Serial echocardiograms showed persistence of the pericardial effusion. On hospital day 14 he had a paradoxical blood pressure of 12 mmHg which subsequently resolved. Within 1 week he was started on rifampin and ethambutol; isoniazid was added on hospital day 15 (Fig. 1).

The persistent effusion indicated that constrictive pericarditis was developing. On hospital day 29 the patient underwent pericardiectomy. His postoperative course was complicated by hypotension, requiring pressors for 2 days and by poor pulmonary function, including edema, bronchiolitis and bilateral effusions.

Pulmonary status improved with the administration of antibiotics, intensive pulmonary therapy, and closed chest drainage. Postoperative antibiotics included tobramycin, cephalothin, and penicillin. Postoperative echocardiogram showed a persistent small pericardial effusion. Pathology of the pericardium showed acute and chronic inflammation and necrosis.
Drug Dose Summary:
1. Indomethacin
   50 mg po q 8h
2. Rifampin 600 mg po daily
   Ethambutol 1200mg po daily
3. Isoniazid 300 mg po daily
4. Tobramycin 80 mg IV q6h
5. Cephalothin 1 gm IV q6h
6. Penicillin G 5 million units IV q6h
7. Penicillin V-K 500 mg po q6h

A gram stain of the pericardial fluid showed numerous leukocytes and no microorganisms. A culture of the pericardial fluid was positive for fusiform anaerobic gram negative bacilli. Cultures of sputum samples and pericardial fluids were negative for mycobacteria. Postoperative blood cultures were negative, and cultures of sputum samples revealed numerous organisms, suggesting oropharyngeal flora. The remaining hospital course was one of continued improvement and the patient was discharged on hospital day 50 while still taking rifampin, ethambutol, isoniazid, penicillin, digoxin, pyridoxine, iron and Coumadin. At follow-up, the patient was doing well 3 weeks after having been discharged from the hospital.

RESULTS
Pericardial fluid was initially cultured on a prereduced blood agar plate and a phenethyl alcohol plate and in prereduced anaerothically sterilized chopped meat glucose broth. The solid media were incubated at 37°C in an anaerobic chamber (Forma Scientific). Both plates demonstrated no visible growth and were discarded after 5 days of incubation. After 72 h of incubation the chopped meat glucose broth yielded long filamentous gram-negative bacilli. The bacilli produced acetic and propionic acids and large amounts of butyric acid when analyzed for volatile metabolic acids by gas-liquid chromatography. They were identified with standard protocols established by the Virginia Polytechnic Institute and State University, Blacksburg. The isolate was indole positive and converted threonine to propionate. All carbohydrate fermentations were negative. Escluin and starch were not hydrolyzed, and no gas was produced. These results indicated that the isolate was F. nucleatum. Antimicrobial susceptibility tests were not performed. Routine quality control procedures were performed by the suppliers and by in-house staff for all media, which was found to be within normal limits.

DISCUSSION
There are relatively few case reports describing anaerobic endocarditis or pericarditis. F. nucleatum has been shown to be an etiological agent in infectious endocarditis (3). This report,
however, describes the first documented case of infectious pericarditis caused by *F. nucleatum*.

Previous experience with endocarditis caused by anaerobic bacteria has demonstrated that the oropharyngeal portal of entry is a significant factor in the establishment of this disease. *Fusobacterium* species are commonly found as normal flora in the oral cavity, a likely portal of entry in this case in view of the oral examination.

Although infectious pericarditis is a rather rare disease, antibiotic therapy and surgical drainage of the pericardium may favorably affect the condition of the patient. Therefore, we point out the need for more information about infectious pericarditis and for a special awareness of the unusual pathogens that cause it.

**LITERATURE CITED**