included fever, weakness, headache, retrobulbar pain, gustatory alterations, lumbar backache and myalgias. A saddleback fever and leukopenia were documented during the course of the illness, and results of serological evaluation confirmed the causative agent to be a dengue virus. Dengue fever is rarely seen in the United States, despite its worldwide endemicity and the continued infestation of the southern states with its vector, Aedes aegypti mosquitoes. However, the rapidity of modern travel readily allows for the introduction of sporadic cases. This report emphasizes the continued need both for the physician to be familiar with diseases endemic to distant locales and for his acquisition of a travel history from a febrile patient.

REFERENCES

1. Deller JJ Jr, Russell PK: An analysis of fevers of unknown origin in American soldiers in Vietnam. Ann Intern Med 66: 1129-1143, 1967
2. Ehrenkranz NJ, Ventura AK, Cuadrado RR, et al: Pandemic dengue in Caribbean countries and the southern United States—Past, present and potential problems. N Engl J Med 285:1460-1469, 1971
3. Morlan HB, Tinker ME: Distribution of *Aedes aegypti* infestations in the United States. Am J Trop Med Hyg 14:892-899, 1965
4. Dwork KG: Dengue in New York City. New York State J Med 64:543-545, 1964
5. Dengue fever—U.S.A. Morbidity Mortality Weekly Rep 13: 235, 1964
6. Dengue fever—New Jersey. Morbidity Mortality Weekly Rep 13:404, 1964
7. Clark DH, Casals J: Arboviruses; Group B, *In* Horsfall FL, Tamm I (Eds): Viral and Rickettsial Infections of Man, Philadelphia, JB Lippincott Co, 1965, pp 615-622
8. Hammon W McD: Diseases transmitted by an arthropod vector—Viral Infections, *In* Sartwell PE (Ed): Preventive Medicine and Public Health, New York, Appleton-Century-Crofts, Inc, 1965, pp 306-308
9. Obeyesekere I, Hermon Y: Myocarditis and cardiomyopathy after arbovirus infections (dengue and chikungunya fever). Br Heart J 34:821-827, 1972
10. Halstead SB, Nimmannitya S, Margiotta MR: Dengue and chikungunya virus infection in man in Thailand, 1962-1964—II. Observations on disease in outpatients. Am J Trop Med Hyg 18: 972-983, 1969
11. Hammon W McD: Dengue hemorrhagic fever—Do we know its cause? Am J Trop Med Hyg 22:82-91, 1973
12. Nimmannitya S, Halstead SB, Cohen SN, et al: Dengue and chikungunya virus infection in man in Thailand, 1962-1964—I. Observations on hospitalized patients with hemorrhagic fever. Am J Trop Med Hyg 18:954-971, 1969
13. Bokisch VA, Top FH, Russell PK, et al: The potential pathogenic role of complement in dengue hemorrhagic shock syndrome. N Engl J Med 289:996-1000, 1973
14. Davis BD, Dulbecco R, Eisen HN, et al: Togaviruses and other arthropod-borne viruses; arenaviruses, *In* Microbiology, 2nd Ed. Hagerstown, Maryland, Harper and Row Publishers Inc, 1973, pp 1377-1397
15. Pond WL, Ehrenkranz NJ, Danauskas JX, et al: Arboviruses and human disease in South Florida. Am J Trop Med Hyg 15:205-210, 1966
16. Hammon W McD: Observations on dengue fever, benign protector and killer: A Dr. Jekyll and Mr. Hyde. Am J Top Med Hyg 18:159-165, 1969
17. Bond JO, Hammon W McD: Epidemiologic studies of possible cross protection between dengue and St. Louis encephalitis arboviruses in Florida. Am J Epid 92:321-329, 1970
18. Sather GE, Hammon W McD: Protection against St. Louis encephalitis and West Nile arboviruses by previous dengue virus (types 1-4) infection. Proc Soc Exp Biol Med 135:573-578, 1970
19. Churdboonchart V, Harisdangkul V, Bhamarapravati N: Countercurrent immunoelectrophoresis for rapid diagnosis of dengue hemorrhagic fever. Lancet 2:841, 1974
20. Pond WL, Ehrenkranz NJ, Danauskas JX, et al: Heterotypic serologic responses after yellow fever vaccination: Detection of persons with past St. Louis encephalitis or dengue. J Immunol 98:673-682, 1967
21. Halstead SB, Udomsakdi S, Singharaj P, et al: Dengue and chikungunya virus infection in man in Thailand, 1962-1964—III. Clinical, epidemiologic and virologic observations on disease in non-indigenous white persons. Am J Trop Med Hyg 18:984-996, 1969

Refer to: Borchardt KA, Sullivan RW, Blumberg RS, et al: Erysipelothrix rhusiopathiae endocarditis. West J Med 127:149-151, Aug 1977

Erysipelothrix Rhusiopathiae Endocarditis

KENNETH A. BORCHARDT, PhD
ROBERT W. SULLIVAN, MD
ROBERT S. BLUMBERG, MD
ROBERT H. GELBER, MD
VERONICA BOTCH, BS, MT
SANDRA CRULL, BS, MT
DANIEL J. ULLYOT, MD
San Francisco

ERYSIPELOTHRIX RHUSIOPATHIAE has been recorded in both humans and animals, dating to 1882 when Loeffler first identified the infection in pigs, calling it swine erysipelas. In 1884 Rosenbach described the cutaneous disease in humans as erysipeloid of Rosenbach.[1] Erysipelothrix infection is quite common in animals, and man's vulnerability appears to result from occupational contacts. In humans, infection is usually a minor cutaneous one caused by contamination of a small cut; only rarely does it produce the complication of bacterial endocarditis. This paper will discuss a case of E. rhusiopathiae endocarditis. Additional comments will be directed towards the nature of the infection in humans.

Report of a Case

A slightly obese 41-year-old commercial fisherman was referred from his local physician for evaluation of progressive fatigue, fever, malaise, dyspnea with minimal exertion, orthopnea, night sweats, pedal edema and lower extremity rash. The patient said that there was no history of previous murmurs, or known rheumatic or congenital heart disease.

On physical examination the patient was tachypneic, tachycardic at 140, with a temperature of

From the Department of Clinical Microbiology (Dr. Borchardt, Ms. Botch and Ms. Crull), Cardiology Service (Drs. Sullivan and Blumberg) and Infectious Disease Service (Dr. Gelber), USPHS Hospital, San Francisco, and the Department of Thoracic and Cardiovascular Surgery, VA Hospital, San Francisco (Dr. Ullyot).

Presented in part at the XIII International Conference on Internal Medicine, Helsinki, Finland, August 1976.

Submitted, revised, September 27, 1976.

Reprint requests to: Kenneth A. Borchardt, PhD, Chief, Clinical Microbiology, U.S. Public Health Service Hospital, 15th Avenue & Lake St., San Francisco, CA 94118.

100.4°F (38°C) orally, and a blood pressure of 160/50 mm of mercury. A seborrheic rash was present on his chest and petechiae were noted over his extremities. One splinter hemorrhage was present. There were no Roth spots, Janeway lesions, Osler nodes or conjunctival petechiae. There was no lymphadenopathy or hepatosplenomegaly. On cardiovascular examination, a diffuse left ventricular lift displaced several centimeters lateral to the midclavicular line was noted. S-1 was soft and S-2 was single. A loud summation gallop was present. A II/VI systolic ejection murmur was evident at the left sternal border and base; a III/VI decrescendo diastolic murmur was present in the aortic area with a radiation along the left sternal border and to the apex.

Laboratory data included an electrocardiogram showing sinus tachycardia and nonspecific ST-T abnormalities; a hematocrit reading of 31 percent; leukocyte count of 9,700 with 91 percent polymorphs, 3 bands and normal red blood cell indices. Reticulocyte count was 2.6 percent and erythrocyte sedimentation rate 64 mm per hour (Wintrobe method). An x-ray study of the chest showed borderline cardiomegaly with increased pulmonary vascularity. Values for creatine phosphokinase, lactic dehydrogenase, serum glutamic oxaloacetic transaminase and electrolytes all were within normal limits. Analysis of urine showed one to five white cells per high powered field with neither red cells nor casts seen. A rheumatoid factor test was positive with a titer of 1:80. Serum electrophoresis showed an elevated gamma globulin of 1.8 percent and depressed albumin of 2.7 grams per 100 ml.

Seven blood cultures, taken upon admission, were positive with Gram-positive to Gram-variable pleomorphic rods morphologically resembling E. rhusiopathiae, which was subsequently confirmed by biochemical tests. Identification of the organism was substantiated by the Center for Disease Control in Atlanta.

Treatment with 18 million units per day of penicillin, digoxin, and intravenously and orally given furosamide was continued for six weeks. At this time blood cultures were sterile, and right and left heart catheterization and selective coronary arteriography were carried out.

On December 30, 1975, aortic valve replacement was done with a 9A - #2320 composite seat Starr-Edwards prosthetic valve. Surgical findings included normal sized ascending aorta and pronounced left ventricular dilatation. The aortic valve had three leaflets and was not calcified. It appeared anatomically normal execpt for findings of gelatinous vegetations on the ventricular aspects of all three cusps which grossly distorted the valve causing central aortic regurgitation. The patient's immediate and late postoperative course has been uncomplicated and at six month follow-up (June 30, 1976) he was asymptomatic and had returned to full time work as a commercial fisherman.

Discussion

Economically, swine erysipelas is a serious problem in Europe, Asia and North America, costing American farmers approximately twenty-five million dollars annually.[2] One of the major reasons for this economic impact is the ubiquitous nature of E. rhusiopathiae. The disease has been reported in an assortment of wild mammals, various fowl, and many domestic animals including pigs, sheep, cattle and horses. Shellfish, fish and some insects are capable of harboring Erysipelothrix as a commensal, and swine can have a carrier state contaminating barnyards, water supplies and food through fecal spread.[2] The organism is extremely hardy being resistant to pickling, salting and smoking. In nature it has remained viable in a buried carcass for nine months.[2] Schiffman and Black reported the case of a construction engineer infected at the site of a barnyard that had been vacant for a year.[3]

Considering its prevalence in nature and the opportunity for occupational contact, man is fortunately relatively immune to Erysipelothrix. As in swine erysipelas, human infection occurs with greater frequency during the summer months.[9,10] Erysipelothrix infection in man manifests itself in three forms. The first, erysipeloid, sometimes called fish-handler's disease, is a cutaneous infection which occurs at the site of a scratch, injury, or puncture wound usually on the hand or arm.[4-5] Infection from an insect bite has been reported.[6] Symptoms appear in one to five days with intense pain, swelling, itching, and purple-red erythema which extends peripherally as the central area fades. Erysipeloid is characterized by remissions and relapses[4,5] and an absence of suppuration.[7-9] The disease is usually self-limiting with spontaneous recovery common in one to four weeks.

The infection may progress to a more generalized constitutional form characterized by polyarthritis, extensive erythema, swelling and intense

itching, even though the initial cutaneous lesion appears to have healed.[9] With these symptoms, misidentification as rhus contact dermatitis can occur.[10]

The last and most serious form of the disease is endocarditis. The symptoms are those usually seen with subacute bacterial endocarditis including dyspnea, arthralgia, weight loss, fatigue and fever.[9] Murmurs, splenomegaly and petechiae covering the trunk are sometimes noted. Since 1912 a total of 30 cases of human endocarditis or septicemia have been reported.[1-3,7,11-20] In reviewing these cases, several clinical characteristics are significant. The age of the patients ranged from 10 to 69 years, although most were middle aged. A predominance of males is indicative of the occupational hazard presented by this organism. All patients had opportunity for contact with E. rhusiopathiae through their occupations, although in many cases a primary erysipeloid lesion was not observed. Overall, 15 of the 30 died. Seven of these deaths occurred before the advent of penicillin therapy in 1944. The remaining eight deaths are attributed to various physical conditions of the patients. In one woman there were symptoms of subacute bacterial endocarditis for one year before diagnosis, with death being attributed to aortic insufficiency.[2] Presumably normal heart valves, as was the case with our patient, as well as those involved with congenital and rheumatic heart disease seemed vulnerable to endocarditis. Nine of the patients did have an antecedent history of rheumatic heart disease. Frequently underlying these deaths were physical disabilities, as well as alcoholism and poor nutrition, which may have affected the patient's immune response.

In conclusion, E. rhusiopathiae, while a common organism, is not a frequent pathogen. Occasionally a severe form of infection in the form of endocarditis occurs, but this event is rare as shown by the infrequent accounts in the literature.

Summary

A slightly obese forty-one-year old commercial fisherman was referred for evaluation of progressive fatigue, fever, malaise, dyspnea with minimal exertion, orthopnea, night sweats, and pedal edema. Purpuric spots were present on the ankles and hips. Subsequently, seven blood cultures were positive for Erysipelothrix rhusiopathiae. Treatment was carried out for seven weeks with intravenously given penicillin G, 18 million units per day. At this time blood cultures were sterile, and cardiac catheterization was done because of evidence of pronounced aortic insufficiency and left ventricular failure. Aortic valve replacement was recommended based on the catheterization findings. An aortic valve replacement was done and the immediate and late postoperative course was unremarkable.

REFERENCES

1. Morris CA, Schwabacher H, Lynch PG, et al: Two fatal cases of septicaemia due to *Erysipelothrix insidiosa*. J Clin Path 18:614-617, Sep 1965

2. Grieco MH, Sheldon C: *Erysipelothrix rhusiopathiae*. Ann NY Acad Sci 174:523-532, Oct 1970

3. Schiffman WR, Black A: Acute bacterial endocarditis caused by *Ersipelothrix rhusiopathiae*. N Engl J Med 255:1148-1150, Dec 1956

4. King P: Erysipeloid, survey of 115 cases. Lancet 2:196-198, Aug 1946

5. Nelson E: Five hundred cases of erysipeloid. Rocky Mount Med J 52:40-42, 1955

6. Jackson R: The shapes of infectious disease lesions in the integument of plants, animals, and humans: A dermatologist's point of view. Canad Med Assoc J 97:573-579, Sep 1967

7. Russell WO, Lamb ME: Erysipelothrix endocarditis: A complication of erysipeloid. JAMA 114:1045-1050, Mar 1940

8. Klauder JV: *Erysipelothrix rhusiopathiae* infection in swine and in human beings. Arch Derm Syphil 50:151-159, Sep 1944

9. Klauder JV, Kramer DW, Nicholas L: *Erysipelothrix rhusiopathiae* septicemia: Diagnosis and treatment. JAMA 122: 938-943, Jul 1943

10. Ehrlich JC: *Erysipelothrix rhusiopathiae* infection in man. Arch Intern Med 78:565-577, Nov 1946

11. Mandal BN, Malloch JA: Endocarditis caused by *Erysipelothrix rhusiopathiae*. N Z Med J 73:355-357, Jun 1971

12. McCracken AW, Mauney CU, Huber TW, et al: Endocarditis caused by *Erysipelothrix insidiosa*. Am J Clin Pathol 59:219-222, Feb 1973

13. Simerkoff MS, Rahal JJ: Acute and subacute endocarditis due to *Erysipelothrix rhusiopathiae*. Am J Med Sci 266:53-57, Jul 1973

14. Townshend RH, Jephcott AE, Yekta MH: Erysipelothrix septicemia without endocarditis. Br Med J 1:454, Feb 1973

15. Alexander WD, Goodwin CS: Erysipelothrix septicemia. Br Med J 1:804, Mar 1973

16. Heggers JP, Buddington RS, McAllister HA: Erysipelothrix endocarditis, diagnosis by fluorescence microscopy. Am J Clin Path 62:803-806, Dec 1974

17. Blount JG: Bacterial endocarditis. Am J Med 38:909-922, Jun 1965

18. Lawes FA, Durie EB, Goldsworthy NE: Subacute bacterial endocarditis caused by *Erysipelothrix rhusiopathiae*. Med J Aust 1:330-331, Mar 1952

19. Procter WI: Subacute bacterial endocarditis due to *Erysipelothrix rhusiopathiae*. Am J Med 38:820-824, May 1965

20. Silberstein EB: Erysipelothrix endocarditis—Report of a case with cerebral manifestations. JAMA 191:158-160, Mar 1965