Brucella abortus Endocarditis

A Case

By Raymond A. Hudson, M.D.

The diagnosis and successful treatment of a case of bacterial endocarditis due to Brucella abortus is described. Therapy consisted of the combination of aureomycin and streptomycin.

The purpose of this report is to describe the successful treatment of a case of bacterial endocarditis due to Brucella abortus. As far as I have been able to discover at the date of writing, no successfully treated case of this disease has been reported previously, with the exception of a case of mixed bacterial endocarditis presented in 1954 by Quinn and Brown.1 A second case of treated Br. abortus endocarditis, referred to by Keefer and Hewitt,2 is sketchily described and the follow-up seems brief, being only 1 month.

Case History

J. T., a 33-year-old white man, a worker in a local radiator plant, was admitted to the E. J. Meyer Memorial Hospital on November 2, 1954, complaining of chills and fever for 3 days, malaise and lack of energy for 1 week, and some anorexia. He denied weight loss and claimed good general health although he knew he had rheumatic heart disease dating from an episode of chorea at age 7. In 1946, while living in Illinois, he had a transient right hemiplegia. There was no other history of serious illness. Recent sore throat or other respiratory illness was denied as was any history of joint pain. One tooth had been extracted 6 months before admission and he had had his teeth cleaned 2 months before admission. There was no history of ingestion of raw milk.

Physical examination on entry into the hospital revealed a young man who appeared both acutely and chronically ill. Rectal temperature was 104°F. The heart was slightly enlarged, with a regular rhythm and a rate of 116. A high-pitched systolic and early diastolic murmurs were noted at the base, down the left sternum to the apex. The mitral first sound was accentuated with an opening snap and mid and late diastolic rumbling murmurs at the apex. The blood pressure was 150/40, with a Corrigan pulse. The spleen was enlarged 2.5 cm. below the left costal margin, and was firm and nontender. There was no evidence of cardiac failure. The neurologic examination showed minimal evidence of an old left hemiplegia. The only embolic phenomena were 2 questionable splinter hemorrhages on the fingers of the left hand.

Laboratory findings on admission showed the following: urinalysis negative; hemoglobin 15.4 Gm. per cent; RBC 4.6 million; WBC 6200 with 55 filaments, 16 band forms, 29 lymphocytes, and 1 eosinophil. The erythrocyte sedimentation rate was 10 mm. with a hematocrit level of 47. The electrocardiograph was within normal limits. The lungs were clear on x-ray, with slight cardiac enlargement. The antistreptolysin O titer was 100 units. Nose and throat cultures were negative for hemolytic streptococci.

The impression on admission was that the patient had rheumatic heart disease with aortic insufficiency and mitral stenosis, probably complicated by subacute bacterial endocarditis following dental surgery. While blood cultures were being obtained, salicylate therapy was administered, but it exerted no effect on the febrile state and was discontinued after 48 hours. The patient remained febrile but otherwise unchanged during the first week. On November 9, partial deafness was noted. On November 13, blood cultures taken November 4 and 10 were reported positive for Staphylococcus albus hemolyticus, coagulate negative, and more splinter hemorrhages were noted in the nailbeds and buccal mucosa. Treatment was therefore begun with aqueous penicillin 300,000 units every 3 hours intramuscularly. On November 17, the dosage was increased to 600,000 units every 3 hours, since there had been no temperature response. On November 20, more splinter hemorrhages and petechiae were noted and the fever persisted; penicillin was increased to 1,000,000 units every 3 hours. On November 23, since the isolated Staph. albus was insensitive to penicillin but sensitive to aureomycin, 500 mg. of the latter drug was given every 6 hours by mouth and the penicillin was omitted. That evening the patient developed a splenic infarction, and more buccal petechiae were seen. By November 27, the patient became afebrile and remained so. On this date, aureomycin had been increased to 3 Gm. a day. On December 2 Br. abortus was recovered from...
2 blood cultures taken on November 13. This organism was sensitive to aureomycin in a test tube dilution of 1.6 units per ml. It was sent to the New York State Health Department Laboratory in Albany, where identification was confirmed. 

**Br. abortus** agglutination on the patient's serum was 1:5120 on December 3. On December 10, combination streptomycin and dihydrostreptomycin, 0.5 Gm. b.i.d., was begun and continued during hospitalization and later at home.

All blood cultures, both aerobic and anaerobic, subsequent to November 13 were negative save for 1 on November 19, which showed diphtheroids. Such cultures were taken at least weekly. The agglutination against **Br. abortus** fell steadily to 1:80 at the time of discharge in February 1955. When last checked on August 28, 1956, no agglutinins were demonstrable. Repeated white cell counts ranged from 9,100 to 9,200, with a lymphocytosis of 49 to 53 per cent. The highest sedimentation rate recorded was 19 mm. per hour on January 21, 1955; it was 5 at the time of discharge. Repeated urinalyses showed little save a 1 + albumin on 2 occasions early in the course. The patient's serum of December 4, 1954, agglutinated the patient's brucella organism up to a dilution of 1:2560. (Patient's serum of December 3, 1954, agglutinated a stock antigen up to 5120.)

On February 7, 1955, the patient was discharged at his own demand. On that date he was afebrile and asymptomatic. The spleen was no longer felt and he had gained about 40 pounds in weight. The last splinter hemorrhage was noted on January 27, 1955, and for this reason as well as the type of basic disease, therapy was continued at home. No petechiae were noted after December 13, 1954. The early diastolic blowing murmur of aortic insufficiency changed in pitch during treatment, acquiring a shriller quality. Repeated electrocardiograms showed no change and the heart did not enlarge. No cardiac symptoms were found at discharge or later. After leaving the hospital, the patient intermittently showed a few splinter hemorrhages in the nailbeds of the hands as recently as January 10, 1956. When last seen, on August 28, 1956, he continued to remain asymptomatic, save for some slight residual deafness. There has been no evidence of recurrence, and the patient enjoys full activity. Total duration of treatment was 112 days of aureomycin 3 Gm. daily and 105 days of streptomycin 1 Gm. daily.

**DISCUSSION**

Several features of this case are worthy of comment. First, the diagnosis of endocarditis due to *Brucella abortus* is difficult to make, as other authors have noted, because brucellosis uncomplicated by endocarditis may present with similar features. It was considered that the diagnosis of bacterial endocarditis was well substantiated in view of the repeated showers of petechiae and splinter hemorrhages, the episode of splenic infarction, and the definite change in the quality of the aortic diastolic murmur. Direct bacterial valvular involvement seemed implicit in the changing murmur in the absence of some other factor such as failure, anemia, or rheumatic fever.

Second, it is interesting that the patient continued to show subungual splinter hemorrhages as recently as January 10, 1956. These seemed to occur after discharge from the hospital in relation to some manual task. No evidence of either bacterial recurrence or of blood dyscrasia was ever found to account for this. I have not been able to find a reference in the literature to the persistence of this phenomenon.

Third, the 2 cultures of hemolytic *Staphylococcus albus*, coagulase negative, on November 4 and November 10 have to be interpreted. The possibility of double infection as in the case of Quinn and Brown cannot be ruled out entirely. However, the staphylococcus obtained was not found in cultures of the nose and throat or urine before or after antibiotic treatment and this organism had been a fairly common contaminant current in the bacteriology laboratory. Such an organism is not a pathogen under most circumstances.

As has been found in most reported cases of brucella endocarditis, the aortic valve is the seat of involvement here. In common also with most of the previous cases, the underlying heart disease was rheumatic.

No source of brucella infection could be uncovered in spite of investigation by the Erie County Health Department. The patient denied ingestion of raw milk, but he had spent some time teaching in a rural area in New York State in the year prior to his illness and might possibly have encountered some infected dairy product without realizing it. The patient's deafness persisted in part, although it improved as he was treated. Deafness has been reported as a part of brucella infections and presumably is the result of direct damage to the acoustic nerve. Since it was present prior to treatment and improved as treatment continued, it was thought not to be related to the streptomycin.
A case of bacterial endocarditis due to Brucella abortus engrafted on a previous rheumatic valvular lesion has been described. It is of interest because treatment of the disease with aureomycin and streptomycin was successful.

SUMMARY

References


Most dogs survived ligation of all the major arterial vessels that supply the brain if these were tied in the neck. Ligation of 3 arteries had little clinical effect. After ligation, anastomotic branches were demonstrated by injections of liquid vinyl acetate. The main vessels providing the anastomoses after ligation were observed to be branches of the costocervical and omocervical arteries that join muscular branches of the vertebral arteries. This technic did not demonstrate anastomoses in the carotid system.

Oppenheimer
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RAYMOND A. HUDSON

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