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SUBACUTE ENTEROCOCCAL ENDOCARDITIS FOLLOWING EPIDEMIC HEMORRHAGIC FEVER

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Since the Korean war American physicians have become acquainted with *epidemic hemorrhage fever*.[1] Despite intensive study many of its basic features continue to defy definition. The etiological agent remains unknown. Sequelae and recrudescenses have not been reported.

Recently, we treated a young man with subacute bacterial endocarditis who previously had epidemic hemorrhagic fever. The case is reported to present a possible sequel of epidemic hemorrhagic fever not heretofore described, and to suggest that this sequel be kept in mind when treating returning Korean war veterans.

REPORT OF A CASE

A 22 year old white male tool crib attendant presented himself at the Henry Ford Hospital Emergency Room, May 22, 1953, complaining of chills and fever of 12 hours duration. For three days he had noted asthenia and mild malaise. The day of admission he developed a severe frontal headache, sore throat, and dry hacking cough, followed by backache, urinary frequency, urgency, and dysuria.

In September 1951 while with the army in Korea, 75 miles north of Seoul, he contracted epidemic hemorrhagic fever. His symptons were identical to those of the present illness, except that in addition he had had gross hematuria. From September to December of 1951, he was treated in an army hospital in Japan. Following this hospitalization, he remained "weak" until the summer of 1952. By August he felt well enough to work regularly until the present illness.

Except for the hemorrhagic fever, he had enjoyed good health while in the army. He took chloroquine regularly as prescribed in Korea and primaquine on the ship enroute back to the United States.

The patient had never had rheumatic fever and denied ever having had joint pains, epistaxis, chorea, or "growing pains." Neither at the time of his army physical examinations, nor at any other time had he ever been noted to have any evidence of heart disease.

On admission May 22, 1953, the physical examination revealed the following: He appeared acutely ill, with an oral temperature of 103° F., and was shaking with a chill. The conjunctivae were markedly injected, and there was photophobia. Funduscopic was normal. The pharynx was intensely inflamed and exhibited shallow ulcerations. The gums were spongy, swollen, and bleeding. The abdomen was diffusely tender. The patient appeared nauseated.

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The blood pressure was 125/80 mm. of Hg. Pulse was 124 per minute. In the aortic area there was a soft to-and-fro pericardial friction rub. There were no murmurs. The lungs were resonant and no rales were heard; breath sounds were slightly harsh but well transmitted. There were no petechiae and all joints were freely mobile and non-tender. There was no significant lymphadenopathy. The remainder of the physical examination was not remarkable.

Hospital Course—A venous blood culture drawn the day of admission yielded an enterococcus.

The patient remained febrile for two days. With the preliminary report of the positive blood culture on the third hospital day, intramuscular injections of streptomycin (½ gram) and penicillin (400,000 units) twice daily were begun. The temperature promptly fell to normal by lysis the next three days.

Substernal pain, worse on coughing, relieved by sitting up and leaning forward, persisted for over one week, but the pericardial friction rub was no longer audible. Electrocardiograms taken the second hospital day and periodically thereafter were characteristic of pericarditis.

On the fourth hospital day two splinter hemorrhages were detected beneath the fingernails. That day, a soft systolic aortic murmur was audible for the first time. This murmur increased in intensity to a maximum the third hospital week; it subsided somewhat then, but remained distinctly present.

When complete studies of the organism causing the bacteremia proved that it was an enterococcus, antibiotic therapy was intensified. On the twelfth hospital day an indwelling intravenous polyethylene catheter was inserted, and a continuous infusion of 5% glucose in water was begun, via which 20 million units of penicillin were administered daily. The intravenous solution also contained 50 mg. heparin sodium per 1000 cc., a dosage which in our experience will obviate intra-catheter clotting without producing detectable systemic anticoagulation.[2] One-half gram of streptomycin plus one-half gram of dihydro-streptomycin were injected intramuscularly twice daily.

Although the patient had remained afebrile, on the eighteenth hospital day he suddenly developed acute generalized abdominal pain and vomited some material containing a small amount of fresh blood. A scout film of the abdomen revealed a paralytic ileus. Several large petechiae appeared on the soft palate and buccal mucosa, as well as some new ones in the nailbeds. One of the petechiae of the nailbed was removed and examined microscopically; numerous pleomorphic short-chained cocci were observed. Blood cultures obtained at this time remained sterile. Conservative treatment with Miller-Abbott tube drainage, hot stupes, and antispasmodics was successful.

On the twenty-fifth hospital day, dental x-rays revealed five devitalized and infected teeth, as well as two retained root fragments. These teeth and root fragments were removed at two sittings. Blood cultures at the time of extraction proved sterile. The teeth were cultured and yielded Candida albicans, Staphylococcus albus (coagulase negative), Aerobacter aerogenes, and Escherichia coli.

Except for two days of temperature up to 100° F at the time of the abdominal crisis, the patient had remained afebrile since the first few days after admission. On June 25 the antibiotics were discontinued, and on June 27, the 35th day of hospitalization, he was discharged well.

Laboratory Data—The day after admission the red cell count was 5.19 million per cu.mm., hemoglobin 14.5 grams per cent, white cell count 10,900 per cu.mm. with 75% neutrophiles and 25% lymphocytes. Counts were obtained daily thereafter for one week and then three times a week until discharge. Except for some neutrophilia at the time of the abdominal crisis (12,650 white cells per cu.mm. with 81% neutrophiles), the counts were remarkable only in that there was usually a slight monocytosis (average 10%) and eosinophilia (average 3%). There were no leucocytoid lymphocytes and no malaria parasites.

Erythrocytic sedimentation rates were obtained semi-weekly and ranged from 5 to 15 mm. per hr. (Wintrobe, corrected.) Hematocrit ranged from 48 to 54 mm. Three platelet counts were made the first week after admission and were 246,000, 228,000 and 368,000 per cu.mm. Bleeding and clotting times and tourniquet tests were normal.

Urinalysis was done daily. Traces of albumin were present the ninth and seventeenth hospital days; granular casts were present those days and also on the twenty-eighth hospital day. There was never any hematuria or pyuria. From the 13th to the 16th hospital days, all voided urine specimens were checked for albumin; one of these contained three plus albumin, but all others were negative.

Fasting blood sugar, blood cholesterol, and frequent blood non-protein nitrogen determinations, were normal. The Kline exclusion test was negative.

Agglutinations for typhoid, paratyphoid A and B, Brucella abortus, Proteus OXK, OX2 and OX19 were done on the 2nd, 10th, and 31st hospital days. All were negative except the proteus OX19, which exhibited titers of 1:80 and 1:40 the first two tests, but was negative the third time. Weil's agglutination tests done on the 10th and 31st hospital days were negative. Heterophile agglutinations on the 2nd and 31st hospital days were negative. Antistreptolysin 0 titers on the 2nd, 10th, 18th and 31st hospital days showed no significant rise.

In addition to the first blood culture, which was positive, nine others taken at four to six day intervals were negative. Throat culture yielded, beta, hemolytic streptococcus, Hemophilus influenza, and Neiseria catarrhalis. Two urine cultures, two stool cultures, and two warm stage stool examinations were negative.

Urea clearance test demonstrated normal clearance. Phenolsulphonphthalein excretion was 65% the first hour. Intravenous pyelograms were negative. Chest x-ray was normal.

Prothrombin time was 13 seconds, 100% of normal. Cephalin cholesterol, thymol turbidity, and thymol flocculation were done the 3rd and 13th hospital days and were normal. Van den Bergh determination revealed a total of 0.77 and direct of 0.19 mg. per cent. Icterus index was 4 units. Serum albumin was 4.1 grams per cent and serum globulin was 2.1 grams per cent.

Case Comment—The diagnosis of subacute bacterial endocarditis was established by the demonstration of positive blood culture (enterococcus), bacterial emboli, development of a murmur, and presence of fever and chills. The diagnosis of pericarditis was established by the presence of a pericardial friction rub, substernal pain aggravated by coughing and deep breathing and relieved by sitting forward, and by the presence of typical electrocardiographic changes.

DISCUSSION

Two features of this case are of particular interest. The first is the nidus upon which bacterial endocarditis developed; the second is the etiology of the pericarditis.

Bacterial endocarditis rarely involves a normal valve and when it does, the clinical course and bacteriology are characteristic. The simultaneous onset of subacute bacterial endocarditis and acute rheumatic endocarditis has been reported[3] but is exceedingly uncommon. If the endocardium, valvular or otherwise, was damaged in this patient prior to his present illness, three possibilities may be considered: Congenitally deformed valves or septa are sometimes involved by bacterial endocarditis, but these are almost always detectable by auscultation. Previous valvulitis, for example from rheumatic fever, also should be detectable clinically, but in addition, there was no history of any form of rheumatic fever. The sedimentation rate was not elevated on admission, the antistreptolysin titer did not rise significantly, and the electrocardiogram did not reveal any delay in the auriculoventricular conduction time.

Another possible explanation for the valvular or other endocardial damage is the previous epidemic hemorrhagic fever. Although no valvulitis per se was described in the autopsies recently reviewed,[1d] all these were in patients in the acute phase of the disease. Subendocardial hemorrhages are a hallmark of the disease and all four chambers as well as the aorta itself have been involved.[1e] It then seems logical to deduce that slight residual roughening of the endocardium not detectable by auscultation, may follow epidemic hemorrhagic fever. We consider this the most tenable explanation in this case.

Pericarditis may be associated with subacute bacterial endocarditis. It is not uncommon in acute rheumatic fever. It also occurs following upper respiratory infections. All three of these possibilities can be considered in this case. A fourth consideration, however, is introduced by the fact that subepicardial hemorrhages have been found in a number of cases of epidemic hemorrhagic fever.[1e] In this case it is difficult to assign the etiology of the pericarditis. The similarity of the symptoms, at the onset, to those which ushered in the hemorrhagic fever, i.e., the presence of tracheobronchitis, gastritis, pharyngitis, rhinitis, conjunctivitis, and possible peritonitis, as well as dysuria and frequency, suggest the possibility that the patient's epidemic hemorrhagic fever residuals, if not the disease itself, underwent a transient recrudescence.

Intermittent albuminuria and the cylindruria have been described as occurring in epidemic hemorrhagic fever.[1] These also occur as a consequence of renal emboli

during the course of subacute bacterial endocarditis. In view of the history of gross hematuria with the Korean illness, and because of the marked urinary tract symptoms despite minimal laboratory and physical findings, it is possible two pathological processes existed. Even though there were probably some renal emboli, the early urinary symptoms may be the result of acute toxicity at the onset of the illness aggravating damage in the kidneys residual from the epidemic hemorrhagic fever.

Teleologically we have reconstructed the case as follows. The diagnosis of epidemic hemorrhagic fever in Korea is accepted. Poor oral hygiene produced a focus from which enterococcemia arose.[2] Subacute bacterial endocarditis developed in the region of the aortic valve roughened by the epidemic hemorrhagic fever. When the subacute bacterial endocarditis was adequately treated, the entire illness subsided.

SUMMARY AND CONCLUSIONS

1. A case of subacute enterococcal endocarditis occurring in a patient who had had epidemic hemorrhagic fever is described.

2. Reasons for concluding that the endocarditis involved endocardium previously roughened by epidemic hemorrhagic fever are presented.

3. It is suggested that this possibility be kept in mind when treating patients who have had epidemic hemorrhagic fever.

4. The validity of these conclusions will depend on long-range follow up of other patients who have had epidemic hemorrhagic fever.

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