ACUTE RHEUMATIC MYOCARDITIS AT AGE 84

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This patient is of interest for three reasons: (1) acute rheumatic myocarditis occurred at the age of 84 years—the oldest age in which such an event has been recorded, (2) the interval between the final and the previous acute rheumatic episode was 70 years—the longest such interval reported, and (3) Aschoff nodules were present in skeletal muscles also, a finding not adequately documented and studied so far.

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Acute Rheumatic Myocarditis
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The isolation of coagulate-negative Staphylococci from the heart, blood and vegetations is another interesting feature of this case. This microorganism rarely causes endocarditis. In a review of the Mayo Clinic records, it was encountered in 1.5-13 percent of patients with bacterial endocarditis. All cases hitherto reported described valvular lesions; in our case, this etiologic agent was responsible for a true mural (non-valvular) endocarditis.

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This case report concerns the oldest patient with documented acute rheumatic myocarditis. Patients with acute rheumatic fever and carditis in childhood or adolescence and minor degrees of residual valvular involvement have been known to live to 80 years and beyond. However, recurrence of acute rheumatic fever at the age of 84 which extensively involved the myocardium and produced clinical congestive heart failure has not been reported before.

CASE REPORT

At the age of 11 years, the patient had her first attack of acute rheumatic fever characterized by joint pains and fever. She was confined to bed for several weeks but recovered without any murmurs. At the age of 14 years, there was a recurrence of acute rheumatic fever with the same manifestations of fever and joint pains lasting for several weeks. No murmurs were heard during the acute attack or at any time following recovery. For the next 70 years, she led a very active life without history of any cardiac symptoms.

At the age of 77 years, she was admitted to another hospital for resection of a polyp in the transverse colon. Detailed preoperative evaluation revealed a normal heart without murmurs. Heart size was normal and electrocardiogram was unremarkable. At the age of 81 years, she was admitted to this hospital for evaluation of intermittent claudication in her calves. Plain film of the abdomen revealed an aortic aneurysm extending from the second to the fifth lumbar vertebrae. Blood pressure was normal. There were no murmurs. Heart size and contour were radiologically normal. Electrocardiogram showed no abnormality.

At the age of 84 years, one month before death, she entered the hospital because of diminishing appetite, excessive fatigue and low grade fever (99.6° to 101.5°F) of two weeks’ duration. Soon after admission, she developed congestive heart failure with considerable dyspnea, distended neck veins, enlarged liver and +++ edema in feet and legs. Examination of the heart revealed sinus rhythm with a rate of 86 per minute. An S3 gallop was present. There were no murmurs. The electrocardiogram revealed a sinus rate of 86 beats per minute. The mean QRS duration, the PR-interval and the QT interval were normal. ST and T components showed no significant abnormality. Chest x-ray film showed a “full-sized” heart with a cardiothoracic ratio of 51 percent.

Despite intensive therapy with digitalis and diuretics, congestive heart failure persisted unabated and the patient died one month after admission as a result of rupture of her abdominal aortic aneurysm.

At autopsy, the heart weighed 510 gm. The histologic sections showed extensive involvement of the myocardium by typical Aschoff bodies in their fully developed granulomatous stage with their distinctive histologic and diagnostic appearances as seen in acute rheumatic myocarditis of about four to six weeks’ duration. Aschoff bodies were seen in great profusion in all sections of the myocardium in all the four cardiac chambers. These bodies were in their fully developed form and were made up of myocardial histiocytes with a few giant cells. The Aschoff nodules were scattered throughout the fibrous tissue of the myocardium in close relationship with the adventitial coat of the small and medium-sized arteries. In the central portion of some of the nodules was swollen and, sometimes, fragmented collagen. The interstitial tissue was edematous and contained scattered monocytes (Fig 1, 2). Worthy of note was the fact that all the valves were normal. The coronary arteries showed minimal atherosclerosis and there was no evidence of recent or old occlusion.

Also of interest was myositis with small perivascular granulomatous lesions in the routinely examined skeletal muscle sections. The perivascular lesions consisted of large mononuclear cells, histiocytes, an occasional giant cell and a few
scattered lymphocytes in the periphery. The lesions very much resembled older Aschoff nodules in rheumatic myocarditis (Fig 3).

**DISCUSSION**

This case has some remarkable features. There was extensive myocardial involvement with Aschoff nodules at age 84. The congestive heart failure during the last four weeks of this patient's life had clinically been attributed to coronary artery disease but the presence of fever during the last six weeks of her life and the refractoriness of congestive heart failure to digitalis and diuretic therapy had remained unexplained. The paucity of coronary artery disease and extensive myocardial involvement with Aschoff lesions at postmortem examination leave little doubt that the clinical manifestations were due to active rheumatic myocarditis. Although Aschoff nodules have frequently been an incidental finding in adults and older patients with the background of acute rheumatic fever, the involvement in this case was far too extensive to be an incidental finding.

This patient had her initial attack of acute rheumatic fever at the age of 11 years and a recurrence at age 14 years. After an interval of 70 years, she had another recurrence of acute rheumatic fever. This is the longest interval between two acute episodes so far reported in the literature; also, acute rheumatic fever at the advanced age of 84 years has not been reported before. In a clinical-pathologic study of the pattern of rheumatic heart disease in old age, Kaufman and Poliafko found evidence of rheumatic activity in the myocardium in a 78-year-old man who was the oldest patient in whom such activity was found. However, this patient also had considerable mitral valve disease and the role of the myocardial lesions in the genesis of his congestive heart failure was uncertain. The longest interval between the onset of acute rheumatic fever and the recurrence of activity in old age in this study was 58 years.

Little is known regarding the state of skeletal muscles in rheumatic fever and the presence of Aschoff bodies in the skeletal muscle of the patient is of considerable interest. Except in the report of Shaw, there have been no references in the literature mentioning Aschoff bodies in the skeletal muscles in rheumatic fever. This aspect of rheumatic fever has not been studied and Adam and co-workers have rightly stressed the need for a detailed study of skeletal muscles in rheumatic fever to determine how frequently Aschoff bodies occur in muscles.

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**Chylous Effusions, Extravasation of Lymphographic Contrast Material, Hypoplasia of Lymph Nodes and Lymphocytopenia**

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A patient presented with chylous effusions in the chest and abdominal cavities. Lymphography demonstrated extravasation of contrast material into both cavities which leaked from the lymphatics, as had the chylous lymph which caused the effusions. The lymph loss was accompanied by lymphocytopenia. The presence of unexplained lymphocytopenia in a patient with pleural or abdominal effusion should suggest the possibility that the effusion is chylous. There was notable iliopelvic and juxta-aortic lymph node hypoplasia, but we could not be certain whether this was an initial component of the lymphatic abnormality or whether this represented lymph node depletion of lymphocytes. Before death, immature lymphocytes appeared in the chylothorax fluid which led to a suspicion of malignant lymphoma, but this was not confirmed. Attention is directed to the possibility of confusing grossly immature lymphocytes which appear after lymphocyte depletion, as with recurrent chylous effusion, with cells from a malignant lymphoma.

Chylous ascites or chylothorax, the presence of free chyle in the abdominal or thoracic cavity, is a sign of lymphatic abnormality associated with lymph extravasation. This extravasation, whether due to seepage through a distended lymphatic wall, or to frank outpouring through a diseased or severed vessel is always a sign of interference to the flow of lymph through those lymphatic vessels and their nodes which normally or collaterally transport chyle from the intestines into the left subclavian vein via the mesenteric lymphatics, cysterna chyli or thoracic duct.

We have previously described a patient with chylous ascites in whom, following lymphography, there was noted contrast material lying free in the peritoneal cavity, substantiating leakage from the lymphatics. This phenomenon is not invariably demonstrated, but has since been observed by others. Our patient, with chylothorax and chylous ascites, is reported because

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