TRUE BACTERIAL MURAL ENDOCARDITIS

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A rare example of acute bacterial mural endocarditis and myocardial abscess is presented. The patient is notable clinically by the absence of any specific manifestation of endocarditis or myocarditis. The unusual features disclosed by autopsy were: focal interstitial myocarditis, large myocardial abscesses, localized mural endocarditis with septic vegetations (caused by coagulase-negative Staphylococcus), and the lack of involvement of the valvular endocardium. Pathogenesis of the disease is discussed.

Mural endocarditis, wherein the inflammatory process is limited to the nonvalvular endocardium, is extremely rare. Ribbert" attributed the initial observation of this entity to Leitz and Naumerick and noted that additional descriptions were given by Luschka, Schultz, Nagayo and Kackel. Since this review by Ribbert, there have been sporadic reports relating mural endocarditis to specific (usually congenital) cardiac defects or to endocardial changes of unknown origin.\textsuperscript{5} The rarity of the condition, which has raised doubts concerning its identity and questions concerning its pathogenesis, and the demonstration of several unusual anatomic-pathologic aspects, warrant this brief communication.

\textbf{Case Report}

A 65-year-old white woman was transferred to Goldwater Memorial Hospital on October 11, 1968 for chronic care of idiopathic paralysis agitans of some ten years' duration. The patient had been treated during that time with belladonna and trihexyphenidyl HCL (Artane) and had been given physiotherapy, but her disease had advanced steadily.

At the time of admission, the patient had severe flexion adduction contracture of both lower limbs. Sensation was diminished, notably in the legs, and there was marked impairment of urinary bladder function. Multiple decubiti were present over the trunk, buttocks, and extremities. The lungs were clear and the size of the heart was normal upon clinical examination. The heart sounds, rate and rhythm were normal; murmurs, gallop rhythm, pericardial friction, and abnormal ejection sounds were absent. The arterial blood pressure was 110/80 mm Hg. The peripheral arterial pulses were appropriately present and there was no evidence of disease of the veins. The 12 lead electrocardiogram was normal. The telerontgenogram disclosed a normal cardiovascular silhouette with slight calcification of the aortic arch.

A program of rehabilitation was initiated. The prior use of Artane was continued, and the patient's course was uneventful for several weeks. During her fifth week in the hospital, she developed elevated temperature which persisted until her death. The rectal temperature fluctuated in irregular fashion between 100.4° F and 103.1° F. Daily examinations disclosed no significant change in the physical findings in the heart, except for increase in rate. During the 25th hospital week, rales were heard at both lung bases (and the chest radiogram exhibited changes consistent with bronchopneumonia); the lungs became clear shortly thereafter.

Repeated urinalsises were done and the findings were the same: 1 to 2+ proteinuria and innumerable white blood cells, the range of specific gravity was from 1.010 to 1.026. The leukocyte count ranged from 7,000/mm\textsuperscript{3} to 17,000/mm\textsuperscript{3} with no significant deviation in the differential formula. Anemia developed as the illness progressed, with a

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\textbf{References}


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fall in hemoglobin from 13.0 gm/100 ml to 7.4 gm/100 ml. The blood sugar, urea nitrogen, serum electrolytes, and \( \text{CO}_2 \) remained normal.

Many bacteriologic studies were done. Venous blood cultures were made six times, the last one two weeks before the patient's death. On each occasion, three specimens, obtained at hourly intervals, were subjected to aerobic and anaerobic conditions on appropriate media. All blood cultures were sterile. Culture of urine and decubitus exudate grew out \( E. \text{Coli, Ps. aeruginosa, B. proteus, alpha Streptococcus and coagulase-negative Staphylococcus.} \)

Antibiotic therapy was instituted according to the bacteriologic findings. Sodium colistimethate (Coly-Mycin M) was administered for \( \text{Pseudomonas aeruginosa}; \) kanamycin sulfate and nitrofurantoin (Furadantin) were given for \( B. \text{proteus (Providence strain) infection. Nalidixic acid (NegGram), ampicillin and sodium cephalothin (Keflin) also were employed. General supportive measures were continued. Nevertheless, there was gradual deterioration of the patient's condition without demonstrable change in the findings upon physical examination of the heart, or in the electrocardiogram. The patient died 83 days after admission to the hospital.}

Pathologic Findings: The necropsy was done six hours after death. The body was that of a well-developed but emaciated elderly woman who had multiple infected decubiti. There were thromboemboli of a secondary branch of the left pulmonary artery with infarction of the lower lobe of the left lung; the spleen was mushy and the liver was fatty.

The heart weighed 250 grams. The pericardium was normal. The myocardium was flabby and the left ventricle was dilated. In the lower half of the interventricular septum there were multiple irregular shaped, but well-defined pinkish gray masses of friable soft tissue attached to the left ventricular endocardium (Fig 1). They measured between 0.3 to 0.6 cm in their greatest diameter. At the slightest squeeze, impressive amounts of purulent material could be expressed from each of these formations. The endocardium at the site of these lesions was opaque and grayish. There were no valvular lesions or vegetations and there were no similar formations in any of the other chambers of the heart. Upon sectioning the septum the cut surface disclosed beneath some of the vegetations a large abscess measuring 2.0 x 0.5 x 0.3 cm in its greatest dimensions (Fig 2). The coronary arteries were patent with mild atheromatosis.

Microscopic examination of most areas of the heart disclosed normal myocardial fibers with occasional foci of cloudy swelling. However, sections taken from the myocardium close to the endocardium of the left ventricle showed loose edematous myocardial interstitium which contained numerous chronic and rare acute inflammatory cells (Fig 3). In one large area there was an accumulation of acute inflammatory cells enmeshed in fibrinous material. An extension of this process toward the endocardium and continuous with a mass of organizing vegetation could be seen. The endocardium itself, when not disrupted, showed focal thickening and granulation tissue to which vegetations were closely attached. These vegetations consisted of a central area with numerous polymorphonuclear leukocytes and multiple coccal organisms and a peripheral zone of fibrinous material, red blood cells, platelets and cellular debris (Fig 4). A postmortem culture of heart blood and vegetations yielded pure colonies of coagulase-negative Staphylococcus.

**DISCUSSION**

The clinical diagnosis of acute bacterial endocarditis is often missed, particularly in the elderly patient. The error has been attributed to either the regrettable tendency to identify the complaints of elderly patients with arteriosclerosis, or the concept that endocarditis in this age group is a terminal and clinically insignificant phenomenon. The clinical diagnosis is made more difficult when the cardiac lesion is limited to the mural (non-valvular) endocardium. Although the possibility of bacterial endocarditis was considered, and corroborative evidence for the condition was sought, the persistently negative blood cultures, the unchanging cardiac physical

![Figure 1. Left ventricle with septic vegetations on the mural endocardium.](http://journal.publications.chestnet.org/pdfsaccess.ashx?url=/data/journals/chest/21507/ on 04/19/2017)

![Figure 2. Large abscess in the interventricular septum of heart.](http://journal.publications.chestnet.org/pdfsaccess.ashx?url=/data/journals/chest/21507/ on 04/19/2017)

![Figure 3. Interstitial myocarditis and abscess.](http://journal.publications.chestnet.org/pdfsaccess.ashx?url=/data/journals/chest/21507/ on 04/19/2017)
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
at Age 84*

The isolation of coagulase-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.

References


Findings, and the invariant electrocardiograms made it impossible to establish clinically this diagnosis in the case at hand.

Exudative mural endocarditis secondary to bacterial infection, without a similar reaction in the valvular endocardium, is uncommon though Trasoff and Meranze mentioned this possibility in association with a Pneumococcal infection. They described a heart with a large thrombus which arose from the left atrial wall slightly beyond the line of attachment of the posterior mitral valve leaflet. The leaflets themselves were moderately thickened.

The sequence of events and the significance of the endocardial and myocardial lesions, in the evolution of the fully developed abnormality reported herein merit comment and speculation. Bacterial invasion of the bloodstream, from a septic decubitus ulcer or another site, undoubtedly occurred and produced focal interstitial myocarditis and a large abscess. A solitary abscess of this size, in itself, is an uncommon finding. Shoenfeld et al found myocardial abscesses in 0.2-0.5 percent of autopsies while Ryon et al reported a frequency of 0.18-1.5 percent. For the most part, these were small abscesses.

We believe that this large myocardial abscess initiated the endocardial lesions. The uniform integrity of the endocardium everywhere except over the myocardial lesion, the absence of a demonstrable congenital defect at the infected site, and the rather benign appearance (apart from the inflammatory reaction) of the involved endocardium support this belief. The idea that the vegetations represent a local thrombotic process with subsequent organization and septic degeneration was considered, but the gross and the microscopic features of the involved tissues suggested the mural origin of the vegetations: a) gentle squeezing of the myocardium, at the diseased area, expressed a thick purulent material which adhered to the surface of the endocardium; b) microscopic examination of these sites disclosed foci of an interstitial exudative inflammatory process which extended from myocardium to endocardium, disrupted the endothelium, and resulted in the deposition of a necrotic mass of fibrin, bacteria and blood elements on the surface of the myocardium. Focal organization of the process was also present.

Figure 4. Endocarditis and the attached septic vegetation.