Simulation of Electrocardiogram of Apicalateral Myocardial Infarction by Myocardial Destructive Lesions of Obscure Etiology (Myocardiopathy)

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THREE MEN, all under 40 years of age, had electrocardiograms indicating the existence of transmural apicalateral myocardial destruction. Two of these patients have died. In neither was significant coronary atherosclerosis present. The etiology of myocardial destruction and fibrosis remains obscure. The advanced degree and confluent character of these changes would appear to represent an unusual variant of the myocardial alterations in that nebulous diagnostic category, "myocardiopathy."

Case Reports

Case 1

A white man, aged 37 years, was examined on May 5, 1955. He reported that in 1941 he had been accepted for military duty and had tolerated the rather strenuous activities required of him, but had noticed some dyspnea and exhaustion when engaged in long hikes. In 1944, he sought admission to the officers' corps, but was refused because his heart was found to be enlarged. He reported that soon thereafter he was sent overseas as an infantryman. He was discharged from military duty in 1946. In January 1955, he experienced dyspnea of increased severity, orthopnea, and marked fatigue. At no time did he have pain in his chest. Examination in May 1955, revealed the presence of a faint systolic murmur just to the left of the sternum. Roentgenogram of the chest (fig. 1, right) showed marked enlargement of the cardiac shadow, the form of which suggested the presence of an aneurysm of the left ventricular wall in the apicalateral region. An electrocardiogram also was supportive of a destructive lesion involving the myocardium of the apicalateral region of the left ventricle (fig. 1, left). The patient was advised to continue a program of treatment appropriate for congestive heart failure. He died in December 1955, five days after an exacerbation in his symptoms of severe congestive failure.

Relevant Morphologic Data*

The heart weighed 565 Gm. The valves and coronary arteries were normal. The left ventricle in its anterolateral and posterior region, as well as the adjacent portions of the right ventricle posteriorly, showed considerable thinning. Examination of these regions of the myocardium revealed gray streaking, which appeared to be fibrous replacement of myocardium. These lesions involved that portion of the ventricles lying below the junction of the basal third with the apical two thirds. The muscle of the basal third appeared to be of usual thickness and showed only focal fibrosis.

In the lateral aspect of the left ventricle, at about three fifths of the distance from the apex to the base, the thinned portion of the myocardium was underlain by a thin, organizing mural thrombus. The left ventricular endocardium generally, but particularly over the dilated portion, was gray and appeared to be slightly thickened.

The appearance of the left ventricular wall is shown in figure 2, left. Noteworthy, is the extreme degree of thinning of this wall in the region between the columnae carneae, where it is in places only a millimeter in thickness. On exposure to bright light, these portions of the ventricular wall permitted transillumination.

Microscopic examination disclosed that in the thinnest portions of the left ventricle, there was considerable fibrous replacement of myocardium. This new material took a subepicardial position with intact muscle being present under the endocardium (fig. 2, right). Some of the endocardial

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*This pathologic specimen was submitted for review through the kindness of Dr. Edward H. Fisheer, North Kansas City, Missouri. Certain features of this case were presented in "An Atlas of Acquired Diseases of the Heart and Great Vessels," Volume II, p. 806-807, and are reproduced here with permission of the author, Jesse E. Edwards, M.D., and the publisher, W. B. Saunders Company, Philadelphia and London, 1961.
muscle represented fairly well preserved columnae carneae.

Elsewhere in the myocardium, where the thinning was not apparent, were isolated muscle fibers or small bundles of such fibers surrounded by collagenous tissue. Nowhere was there evidence of active inflammation.

Case 2

This patient was 23 years old when he died on June 2, 1960. He had been in good health until January 1958. Between January and July he experienced four febrile episodes, the last of which was attended by cough and hemoptysis. He improved promptly on antibiotic therapy but hemoptysis continued to recur until September 1958. Breathlessness on exertion first occurred in August 1958, and progressed within a period of 3 weeks to dyspnea at rest and orthopnea. When examined in September 1958, he was moderately breathless but not cyanotic. His blood pressure was 110 mm. Hg systolic and 90 mm. Hg diastolic. Auscultation of the heart revealed a moderately loud systolic murmur, heard best at the lower left
sternal border, and a gallop rhythm. Hepatomegaly was present but the venous pressure was only 10 cm. of water.

Electrocardiograms (fig. 3) showed a sinus tachycardia and QRS changes indicative of a destructive myocardial lesion involving the apicolateral region of the left ventricle. Roentgenograms of the chest disclosed a globular cardiac silhouette and evidence of pulmonary congestion.

Anticoagulation and treatment appropriate to congestive cardiac failure were instituted and the patient's response was satisfactory while he was in the hospital. In subsequent months, however, his failure recurred during lapses in treatment. In April 1960, he was admitted to the hospital suffering from congestive failure and hemoptysis. Again he responded to treatment. He left the hospital contrary to medical advice on May 28, 1960. He returned on June 2, 1960, in extremis. His respiration was gasping and he was cyanotic and in shock. He died within an hour.

At necropsy the lower lobes of both lungs were infarcted. The heart weighed 520 Gm., and the left ventricular wall was thinned and scarred in the apicolateral region (fig. 4, left). Microscopic examination disclosed confluent zones of fibrosis surrounding small islands of myocardial fibers (fig. 4, right).

Case 3

This third patient, a white man aged 25 years, is alive and free of symptoms of cardiac disease. He reported that he could play baseball or run three blocks without unusual shortness of breath. He presented himself for examination because he had been refused life insurance. Existence of...
ELECTROCARDIOGRAM SIMULATING MYOCARDIAL INFARCTION

Figure 3
Electrocardiogram from case 2. Note the presence of deep Q deflections in leads I, aVL, V_1, V_2, and V_6 and increased height of R deflection in V_1.

Figure 4
Left. The opened left ventricle in case 2. Note scarring and thinning of apical half of left ventricular wall. Right. Photomicrograph, case 2, showing confluent scarring surrounding patches of preserved myocardial fibers.
a systolic murmur at the base of the heart was given as grounds for refusal of insurance. Physical examination otherwise was negative. Roentgenogram of the chest disclosed findings supportive of left ventricular hypertrophy (fig. 5). Findings derived from catheterization of both the right and left ventricles were normal, including the level of the end diastolic left ventricular pressure.

The electrocardiographic changes are remarkably similar to those encountered in cases 1 and 2 (fig. 6). Q deflections were present in leads aVL, V5, and V6, and the R waves in V1 were unusually tall, suggesting a destructive lesion involving the apicobasal region of the left ventricle.

This patient reported that one of his brothers died in his twenties of a ruptured heart while on military duty. A second brother, now in his thirties, is under treatment for "a serious heart disease" of undetermined type.

Discussion

The remarkable degree of myocardial destruction in case 1 raises question of parallelism with the mysterious "parchment heart" of the Osler collection,¹ so revealingly described by Segall.² If a common process was at work in the two hearts, however, the stage of destruction reached in Osler's case was far more advanced than in case 1.

The heart of the Osler collection weighed 168 Gm. and, if truly, as Dr. Maude Abbott vaguely recalled, it was "found in a man who had died suddenly while walking up a rather steep hill in Montreal,"³ then obviously such myocardial mass as was added by hypertrophy preceding or attending myocardial destruction (and the two processes coexist in most instances of myocardial fibrosis) ultimately was wiped out by the destructive process. In case 1, the heart weighed 565 Gm. in spite of myocardial destruction involving the apical two thirds of the heart. Quite clearly, the mass of myocardium added by hypertrophy in this instance still exceeded the loss consequent to the destructive process.

A second point of difference is suggested by Osler's statement, quoted by Segall, "in places in the right and left ventricle only the epicardium remains." The illustrations in Segall's article disclose flat, thin papillary muscles and columnae carnea. In contrast,
ELECTROCARDIOGRAM SIMULATING MYOCARDIAL INFARCTION

Figure 6

Electrocardiogram from case 3. Note presence of Q deflections in leads I, aV_L, V_5, and V_6. R waves in V_4 are unusually tall.

The columnae carnea in case 1 were better preserved than were the more epicardially disposed portions of the left ventricular free wall (fig. 2, right). The possibility again exists that this contrast is an expression of the less advanced stage of the destructive process in case 1 as compared with that which existed in the Osler case.

Certainly the degree of myocardial destruction in case 2 was of a lesser order than existed in the Osler case and in case 1. Even so, both cases 1 and 2 are distinguished from the usual cases of idiopathic myocardial infarction by the electrocardiographic and pathologic evidences of an advanced myocardial destructive lesion. The fact that the

Circulation, Volume XXV, March 1962
patient in case 2 died as a consequence of embolic complications of his cardiac disease may afford basis for demise at a stage of myocardial destruction not so advanced as in case 1.

In case 3, the question arises whether this patient is in an asymptomatic stage of disease fundamentally the same as that which affected the patients in cases 1 and 2. In case 1, the patient was comparatively free of symptoms for almost a decade after his cardiac silhouette first was observed to be enlarged on a roentgenogram of the chest.

Of interest in case 3, also, is the fact that the patient’s brother died during young adulthood of cardiac disease said to have been nonvalvular in type. This circumstance introduces the possibility of a familial element in the etiology of the cardiac lesion in this patient of case 3.3. 4

The simulation of transmural myocardial infarctions by lesions affecting predominantly portions of the ventricular wall superficial to the columnae carneae is consistent with experimental data indicating that these epicardially disposed layers are responsible for the R wave in direct or semidirect unipolar leads in which the exploring electrode rests on or overlies the left ventricle.5-9

Summary

A myocardial destructive process unrelated to occlusive coronary arterial disease has been documented at necropsy in two patients aged 37 years and 23 years at the time of death.

In the third patient, 25 years of age, electrocardiographic findings of a transmural lesion of the apicolateral wall of the left ventricle existed in the absence of evidence of impaired myocardial reserve.

The nature of myocardial destruction encountered in cases 1 and 2 is compared with that in the case of parchment heart described initially by Osler.

The theoretic implications of total loss of R waves in semidirect leads overlying a region of epicardial destruction are noted.

Addendum

Through the kindness of the United States Air Force, permission was granted to review the medical records of the brother of the patient described under case 3. He died at the age of 21 years while on active military duty. Symptoms of malaise and weakness appeared only a few days before his death. During his final hours, findings were those of acute cardiac failure attended by pulmonary edema, cyanosis, and shock. At necropsy, the heart weighed 800 Gm. The coronary arteries were normal. Apart from hypertrophy, the ventricular myocardium disclosed no lesions. Final diagnosis were myocarditis of undetermined etiology, cardiac hypertrophy and dilatation, pulmonary edema, and passive congestion of the liver and spleen.

References


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