CASE REPORT SECTION

Coronary Embolism and Acute Myocardial Infarction Secondary to Rheumatic Heart Disease

MURRAY S. HOFFMAN, M.D., JOHN FORREST, M.D. and E. LEE GARLETT, M.D.
Denver, Colorado

Rheumatic heart disease is not frequently considered a predisposing factor in the genesis of acute myocardial infarction. Coronary artery embolism is a diagnosis that is frequently not considered during life and is easily missed at the post mortem examination. The purpose of this article is to report and discuss a proved case of coronary artery embolism producing an acute fatal myocardial infarction in a patient with underlying rheumatic heart disease.

Case Report: A 64 year old white man entered the hospital for the first time on November 21, 1955. On the morning of admission, while in bed, he noted the sudden onset of crushing substernal pain which was extremely severe and radiated down the ulnar aspects of both arms. He felt quite weak and began to sweat profusely. At the same time, he noticed severe shortness of breath and appeared pale.

Past history was unremarkable. There were no known previous serious illnesses or operations. He had no known manifestation of rheumatic fever. System review and family history were non-contributory.

Physical examination at the time of admission to the hospital revealed a moderately obese man who was quite dyspneic and complained of severe chest pain. Temperature was 99° F., pulse 80, respiration 26 and blood pressure 170/106 mm. of mercury. There was no cyanosis, clubbing or edema. Neck vessels were not unusually prominent. The lungs were clear. The point of maximum intensity of the apex beat was diffuse and extended to the fifth intercostal space in the left mid-clavicular line. A faint right ventricular thrust was present. A-2 was louder than P-2 and the second pulmonic sound was finely reduplicated. The mitral first sound was normal and no murmur was detected. Heart tones were of poor quality.

Laboratory examination revealed the following data: Urinalysis showed a trace of acetone but was otherwise negative. Hemoglobin was 17.7 gms. White blood cell count was 16,700 cells cu./mm. with 57 per cent segmented neutrophils, 23 per cent unsegregated neutrophils, 3 per cent eosinophils, 15 per cent lymphocytes and 2 per cent monocytes.

An electrocardiogram taken on admission (Fig. 1) demonstrated the classical findings of a massive, acute antero-lateral myocardial infarction. An electrocardiogram taken two days later was essentially unchanged except that the rate had increased to 115, and premature atrial contractions were much more frequent.

After admission to the hospital, he was placed on bed rest and given supplementary oxygen. Morphine sulfate grains one quarter was administered frequently, but failed to completely control the pain. Additional therapy included papaverine grains 3, 4 times a day and dicumarol 100 mg. on three successive days. During the first four hospital days, his condition remained critical and he frequently complained of chest pain. At 2:00 A.M. on the morning of the fifth hospital day, he became restless; his blood pressure fell to 80/30 mm. of mercury and his skin became cool and clammy. At 4:00 A.M., he had a slight convulsion and was essentially non-responsive thereafter. At 9:20 A.M. he expired.

At autopsy, the body weighed 98 Kg. and measured 170 cm. The pleural cavities contained 150 cc. of straw colored fluid bilaterally. The pericardial sac was lined by roughened membranes over which were several layers of fibrin. The parietal and visceral pericardium contained multiple petechiae. The heart weighed 530 gms., and the myocardium appeared soft and flabby. The left ventricle was dilated and the left

From the Denver Rheumatic Fever Diagnostic Service, University of Colorado School of Medicine and the Department of Cardiology, National Jewish Hospital at Denver.

546
ventricular wall measured 1.5 cm. The right ventricular wall measured 0.5 cm. in thickness. The leaflets of the tricuspid and pulmonic valves were thin and delicate. The aortic cusps were minimally calcified, thickened and fused with one another. Over the free edges and the aortic surfaces were occasional calcified verrucous masses. Slight aortic stenosis was thought to be present. The mitral leaflets as well as the chordae tendineae were markedly thickened and fused. The mitral valve was definitely and significantly stenotic. There was dilatation of the right and left auricles, and the left auricular appendage was almost completely filled with a thrombus. The left coronary ostium was widely patent and appeared two to three times normal size. Both the circumflex and anterior descending branches of the left coronary artery were partially to completely occluded by embolic masses. The right coronary artery was hypoplastic. On section the myocardium of the anterior wall, septum, apex and adjacent sections of the posterior wall was grayish yellow and hemorrhagic in appearance. This was thought to represent an acute myocardial infarct.

The left lung weighed 330 gms. and the right 360 gms. The lungs were crepitant.

Microscopic examination of the heart revealed typical findings of an acute myocardial infarction approximately five days old. The wall of the left coronary artery was thin with minimal atheromatous changes. In the center of the vessel was completely occluding it was a solid fibrinous mass (Figure 2) consistent with the diagnosis of an embolus from the atrial appendage. The embolus appeared to be the same age on microscopic examination as the left atrial thrombus (Figure 3).

Discussion

The first case of coronary embolism was reported by Virchow\(^1\) in 1856. In 1953 Cheng, Cahill, and Foley\(^2\) collected a series of 54 proved cases. More recently individual cases have been described by Watts\(^3\) and Flake\(^4\). Although there have been other reports, it is apparent that there have been relatively few documented cases of coronary embolization. Hamman\(^5\) estimates the incidence of coronary embolism to be 1 to 2 per cent of all coronary occlusions.

Based on the reported cases, there are several sources of the embolic material. In order of frequency they are: (1) Bacterial vegetations on the mitral and aortic valves due to bacterial endocarditis (2) mural thrombi

FIGURE 1: Electrocardiogram taken on admission. Tracing reveals typical findings of acute antero-lateral myocardial infarction.
FIGURE 2: Thrombus in left atrial appendage. Below this is seen the intra-mural left coronary artery occluded by an embolus.

FIGURE 3: Left coronary artery. An embolus is clearly visualized with little or no attachment to the adjacent arterial wall. There are only minimal atherosclerotic changes in the wall of the artery itself.
on atherosclerotic or luetic lesions at the root of the aorta (3) intracardiac mural thrombi (4) thrombi in peripheral veins (paradoxical embolism) (5) thrombi in the pulmonary veins.

The difficulty in making an accurate ante mortem diagnosis of coronary embolism has been described by Hamman. Certain features, however, stand out in the reported cases. Bacterial endocarditis is a well known source of emboli, and the sudden appearance of a myocardial infarction in a patient with bacterial endocarditis should make one suspicious. Indeed, any cardiac patient or a patient with peripheral thrombophlebitis with sudden severe chest pain should alert the physician to the possibility of coronary embolism. Conditions that predispose to the formation of intracardiac thrombi, such as mitral stenosis, create the possibility of coronary emboli. Acute myocardial infarction in young individuals should also suggest the possibility of coronary embolism.

There are a number of post mortem findings in this case which help to definitely establish the pathological diagnosis of coronary embolism. The source of the embolus was clearly demonstrated in the left auricular appendage and the thrombus can be explained as being secondary to the rheumatic heart disease. In general, the arteriosclerotic changes in walls of the coronary arteries were minimal and were insufficient to explain the massive myocardial infarction on the basis of atherosclerosis.

Sections of the left coronary artery clearly showed this artery to have an intra-mural course. This anatomical variation was first described by Geiringer who concluded that the incidence of atheromatous change in these intra-mural vessels was much less than is normally seen in extra-mural arteries. Although this conclusion has not been substantiated by Edwards, certainly the left coronary artery in the present case showed minimal atheromatous changes. Another factor of importance in the present case is that the left coronary ostium was two to three times the normal size while the right coronary artery was hypoplastic thus resulting in a greater ease of embolization to the left coronary artery than would normally be present.

The diagnosis of rheumatic heart disease and coronary embolization probably could not have been made in this case prior to the patient's death, since there were none of the clinical criteria necessary for the ante-mortem diagnosis. Although the diagnosis of rheumatic heart disease with mitral stenosis was quite evident at autopsy, there was no suggestive history and no murmurs were detected. While the typical auscultatory findings may have been missed, this would seem to be unlikely, because of the repeated and frequent examinations of the patient. Mitral stenosis without murmurs does occur and has been described by Levine. He attributes this phenomenon primarily to decreased blood flow across the stenotic valve which might occur in shock, congestive failure, atrial fibrillation, or marked dilatation of the heart. The typical auscultatory findings are made more difficult to hear by such factors as pulmonary emphysema, obesity, fluid in the chest cavity, and distant heart sounds. Dilatation of the heart and shock may well have been important factors in the present case.
REFERENCES


Effect of Lateral Recumbency on Pulmonary Function

ZOLTAN MANN, M.D., F.C.C.P.
Alexandria, Louisiana

It is the general belief that in the lateral recumbent position, which is a frequent position during bed rest, the lower chest, the one which is in contact with the mattress, expands less and consequently its function is diminished. Several authors1-6 have investigated this function by means of clinical observations, radiographical and spirometric examinations but the result of these investigations and the interpretations of the findings were contradictory.

Adams and Pillsbury1 believed that the excursion of the lower hemidiaphragm had a greater respiratory efficiency. Webb, Foster and Gilbert2 found the tidal movement of the lower hemidiaphragm greater than that of the upper; however, after an interval of about an hour it equalized. Contrary to this finding, Pierson and Newell,3 who made their investigations primarily in dogs, did not find the tidal ventilation to be equalized after a longer time.

Vaccarezza and his collaborators4 found increased oxygen consumption through spirometric determination, greater complemental air, and increased vital capacity of the recumbent lung on a person who maintained the same position for two weeks; however, they supposed that the recumbent lung is in elastic repose.

Rothstein, Landis and Narodic5 found spirometrically that oxygen consumption of the recumbent lung is increased. This finding was perplexing to them and they explained this increase as due to the larger capillary bed from relative congestion.

From the Veterans Administration Hospital.