Studies of Myocardial Rupture with Cardiac Tamponade in Acute Myocardial Infarction*

I. Clinical Features

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The clinical features of cardiac rupture with tamponade in acute myocardial infarction have been studied in an attempt to predict this complication and find clues to prophylactic and therapeutic measures. Eleven cases with rupture of the free left ventricular wall in a consecutive series of 529 coronary care unit (CCU) patients with acute myocardial infarction have been analyzed. These 11 patients accounted for 10 percent of the mortality. The significant features were absence of a history of previous myocardial infarction in association with electrocardiographic (ECG) signs of acute infarction present at time of admission. In no instance was the infarct purely inferior in its location. These characteristics would have given 0.2 percent false negative and 17 percent false positive prediction among these 529 patients with acute myocardial infarction. In seven of the patients the rupture occurred during the first day of the illness. In no case did the rupture occur in massive infarction, which provides a basis for hope of success following surgical intervention in the future.

In 1859 Malmsten, then the professor of medicine at Serafinerlasarettet, saw a patient with severe central chest pain.1 The patient died one week after onset of symptoms and autopsy showed what Malmsten refers to as “a broken heart.” It thus seems that it is more than 100 years ago that a physician from this hospital saw a case of rupture of the left ventricle caused by an acute myocardial infarction. With the introduction of a coronary care unit (CCU) in this hospital interest has again been focused on this complication.

Coronary care units have lowered the hospital mortality in acute myocardial infarction to about 15 to 20 percent, mainly by reducing the number of deaths from the arrhythmias.2 About half of the remaining mortality seems to be largely unpreventable, as the infarcts could be shown at autopsy to involve more of the left ventricular myocardium than is ever seen in the fibrotic stage of healing.3 This leaves about half of the mortality as potentially preventable. All cases of myocardial rupture with tamponade are found in this half and account for about 20 percent of this group.4 Thus rupture with tamponade is not unimportant among the potentially preventable causes of death in the acute phase of myocardial infarction. It is therefore of interest to study the clinical characteristics of these patients with the aim of finding some clues to prophylactic or therapeutic measures in this complication.

MATERIALS AND METHODS

The CCU of Serafinerlasarettet, its admission criteria, therapeutic policy and diagnostic criteria have been described elsewhere.4 During January 1968 to April 1970, the CCU had 1,320 admissions, and a diagnosis of acute myocardial infarction was made in 529 of these cases. The delay of admission from the onset of symptoms was three hours or less in 40 percent of the patients with an acute infarction and six hours or less in 62 percent. The mortality during the mean CCU stay of 53 hours was 10 percent, and 21 percent died during the total hospital stay, which averaged 21 days.

RESULTS

At autopsy 11 of the 111 deaths were found to be due to a rupture of the free wall of the left ventricle. This corresponds to 2 percent of the patients with an acute myocardial infarction and 10 percent of the deaths.

Of the 11 ruptures eight occurred during the CCU stay and all were diagnosed clinically on the
basis of a sudden circulatory arrest in association with sustained QRS configuration for some minutes. In one more patient a diagnosis of rupture and tamponade had been made clinically but was not confirmed at autopsy. This patient had a large aneurysm of the left ventricle. In another patient rupture and tamponade was diagnosed clinically but autopsy was refused.

The 11 patients with verified rupture and tamponade were 57 to 85 years old. Seven were men. In neither respect did these patients differ from the rest of the series. None of the 11 patients gave a history of a previous infarction, which significantly differs from 33 percent with one or more earlier myocardial infarctions on admission to CCU (P < 0.05). Four of the 11 patients had a history of arterial hypertension, but this does not differ significantly from the 29 percent for the remainder. Eight of the patients with rupture were admitted within six hours after onset of infarction symptoms, which is in correspondence with 62 percent for the rest of the series. On admission none was in frank pulmonary edema, but five patients showed signs of left heart failure, which does not differ from the prevalence of the rest of the series, 7 and 42 percent respectively. The same is true for shock and hypotension on admission; none was in shock and none hypotensive as compared to a prevalence of 4 percent and 3 percent respectively. The ECG on admission showed the signs of an acute myocardial infarction in 10 out of the 11 cases; in the 11th patient the ECG was not conclusive because of a persistent ventricular tachycardia. Only 36 percent of the rest of the series showed ECG signs of an acute myocardial infarction on admission and this difference is significant (P < 0.05). In eight of the 11 patients the ECG showed an anterior or anterolateral infarction and in the other two an inferolateral one. None had an isolated inferior infarction, which is significantly different from the 33 percent among the remaining ECG positive patients (P < 0.05).

During the stay in CCU there was no significant difference in the incidence of any arrhythmias between the patients with rupture and the rest of the series, although major arrhythmias were uncommon in the rupture group. During the last hour before the rupture two patients showed a slight increase of the systolic blood pressure, 20 and 30 mm Hg respectively.

The rupture and tamponade occurred in eight patients during the CCU stay and in three during the after-care. The interval between admission and tamponade was six hours to seven days and between onset of infarction symptoms and tamponade ten hours to eight days. In seven cases the rupture occurred during the first day after the infarction. The ECG changes following the rupture will be described elsewhere as will be three trials with thoracopercardiotomy and suturing of the rupture.

The autopsies (Dr. H. Nordeusam) demonstrated that the infarct in these patients never was large, involving 20 to 50 percent of the left ventricular myocardium. No previous myocardial infarct was detected at autopsy with the exception of one case in which an inferior infarct about two weeks old was seen. The rupture was always localized in the anterior or lateral wall of the left ventricle, and it varied in size between 5 and 30 mm. Microscopy did not reveal any abnormal inflammatory response in the infaracts.

Thus, the clinical characteristics of these patients with rupture and tamponade were the absence of a history of previous myocardial infarction associated with ECG signs of an acute myocardial infarction present at time of admission. There was a covariation between these two findings but it was not total; 87 percent of the patients with signs of an acute infarction on their admission ECG were found to have a negative history of previous myocardial infarction. Consequently the patients with rupture and tamponade were compared with the rest of the subjects with an infarct-positive ECG on admission and without a history of a previous myocardial infarction regarding clinical characteristics but no significant differences were found.

This series of patients with rupture and tamponade included no case with ECG signs of an inferior infarction without involvement of the lateral wall. As the series is small, the incidence of ruptures in isolated inferior infarctions was studied by scrutinizing all autopsy records of this hospital during 1956-1967. Out of a total of 43 patients with rupture of the free wall nine had infarcts localized to the inferior wall. The admission ECG of these nine patients showed involvement of the lateral wall in five cases leaving four (9 percent) unpredicted by omitting the purely inferior infarcts among the clinical characteristics.

Discussion

Sievers (1966) presented a large consecutive series of ruptures from another Swedish hospital during the pre-CCU era. The autopsy rate, 95 percent, was similar to that of the present study. Rupture with tamponade constituted 13 percent of the deaths and 4 percent of the patients with an acute myocardial infarction as compared to 12 and 2 percent respectively in the present series. The
age distribution is similar in the two materials, but the predominance of women in Sievers' patients was not found in the present study. In accordance to previous reports, most of the ruptures in this series occurred during the first three days after the infarction. A low incidence of rupture among patients with old infarction has been reported previously. A striking finding at autopsy in the present series was that the ruptures never occurred in large infarcts.

The clinical characteristics on admission of a patient prone to rupture with tamponade seem to be: 1) no history of previous myocardial infarction and 2) ECG signs of an acute myocardial infarction on admission involving the anterior or the lateral wall. In this series of 529 patients with acute myocardial infarction these characteristics would have given only one false negative suspicion. In this patient a persistent ventricular tachycardia was present on the admission ECG. On the other hand, these characteristics would have resulted in a false positive suspicion in as many as 90 out of the 529 cases.

Even if a prediction of rupture would allow for prophylactic measures, this study could not reveal any pathophysiological principle to counteract like hypertension or an abnormal inflammatory response to the infarct. The only possibility therefore seems to be to attempt immediate surgical intervention when rupture and tamponade occurs.

Surgical intervention actualizes three questions of practical importance: 1) does the rupture reveal itself immediately in the only continuously monitored parameter, the ECG? This problem will be dealt with in another paper, 2) does a sudden circulatory arrest in association with a sustained QRS configuration for some minutes give any false positive diagnoses of rupture and tamponade? In this series of 529 patients with acute myocardial infarction there was one false positive diagnosis, which had two special aspects: the patient had a big aneurysm, and it was also the first patient in whom a clinical diagnosis of rupture was made in this CCU; 3) is it possible to intervene surgically on patients with rupture and tamponade with any success? This problem will be dealt with subsequently.

REFERENCES


II. Electrocardiographic Changes*

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The possibility of surgical intervention in cardiac rupture with tamponade prompted a study of the value of the continuous electrocardiogram (ECG) for an immediate diagnosis. The ECG was recorded continuously in a coronary care unit (CCU) during a time period when 32 deaths occurred. At autopsy seven patients had myocardial rupture with tamponade and their ECGs all showed sudden bradycardia terminally. Another patient had tamponade from dissecting aortic aneurysm and also showed similar ECG changes. Two further patients showed nonpenetrating lacerations of the myocardium, and their ECG did not show sudden bradycardia. In all cases the bradycardia was of nodal origin within the first minutes. It is concluded that abrupt onset of bradyaryrhythmias in patients with acute myocardial infarction showing sudden unconsciousness should give rise to a suspicion of cardiac tamponade.

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