Cardiac Rupture during Myocardial Infarction

A Review of 44 Cases

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SUMMARY
Forty-four cases of myocardial infarction with cardiac rupture, 88 cases of unruptured myocardial infarction, and 88 cases without myocardial infarction were studied retrospectively. The incidence of cardiac rupture in cases with acute myocardial infarction alone was 5.5% while in hearts having both healed and acute myocardial infarction the incidence was 2.3%. One instance of cardiac rupture was encountered in a heart having only a healed myocardial infarction with subsequent aneurysmal dilation of the healed infarct. Although females represented only 37% of the population having acute myocardial infarction in this institution, they accounted for 55% of the cases of cardiac rupture. On the average the hearts which ruptured following myocardial infarction were lighter and thinner than in the control group of patients having infarction without rupture. Among the clinical correlates possibly associated with postinfarction rupture the most significant finding in the present study is the presence of postinfarction hypertension. In the group of cases with cardiac rupture this was present in 40% while in the control group not having suffered cardiac rupture the comparable figure was 14%. A history of diabetes was found in 18% of the cases of myocardial infarction not having suffered cardiac rupture. A similar history was found in only 9% of the cases having a postinfarction cardiac rupture. This latter incidence is identical with the frequency of diabetes mellitus in the general autopsy population. There is a suggestion that early and severe atherosclerosis may cause earlier heart disease and when infarction occurs provide some protection against rupture. Of interest among the 44 cases of cardiac rupture, none of the patients had cirrhosis, in striking contrast to the 11% incidence of this condition in our general autopsy population.

Additional Indexing Words:
Postinfarction hypertension    Atherosclerosis

Cardiac rupture as a complication of myocardial infarction was first described by William Harvey in 1647.1 Despite long familiarity with its existence the circumstances which lead to this cardiac catastrophe are still unclear. Some of the more significant studies of this clinical problem have been reported.2-12 Among these reports the frequency of cardiac rupture after acute myocardial infarction has varied from 3.3 to 24%. Some of this variation stems from differences in the populations surveyed. Improved methods for the diagnosis and treatment of myocardial infarction and its complications justify a reappraisal of cardiac rupture. It seemed appropriate to reevaluate the clinical and anatomic parameters of this problem in a group of patients treated in a large municipal general hospital. Hopefully, some improved understanding would be achieved of the setting in which cardiac rupture occurred.

Materials and Methods
All of the autopsy protocols on adult patients in the files of the Mallory Institute of Pathology of...
the Boston City Hospital were reviewed for the period January 1, 1959, to December 31, 1968. A total of 8589 protocols were evaluated. Among these, acute and/or healed myocardial infarction was found in 1845 (21.4%). In the present study a myocardial infarct is defined as any grossly visible area of acute ischemic necrosis or myocardial fibrosis at least 1 cm in diameter. The lesion was designated as acute if the heart showed histologic changes compatible with a lesion of less than 4-weeks' duration using the criteria established by Mallory, White, and Salcedo-Salgar.19 Older lesions were arbitrarily termed for our purposes healed. Rupture of the free walls or septum of the heart was found in 44 cases. Accordingly two control groups were established for comparison. Group A comprised the 88 cases of myocardial infarction with the closest autopsy numbers before or after the cases of cardiac rupture. Group B represents an additional 88 cases selected in the same fashion but not having myocardial infarction. The size and age of infarct and site of rupture, whether free wall or interventricular septum, were determined in the study group.

All cases with incomplete rupture of the myocardium or rupture of the valves, papillary muscles, or chordae tendineae were excluded. Also excluded were all patients with a history of cardiac massage or intracardiac injections in the current terminal heart attack, since such therapy might predispose to rupture. None of the patients in the present study received pump assist or pacemakers, or had surgical interventions in the treatment of their infarcts. All medicolegal cases were excluded from this study, i.e., all patients dead on arrival at the hospital, or those where the death had occurred under questionable circumstances. Undoubtedly these exclusions impose a bias in the overall incidence of myocardial infarction within the time span of the study since they exclude a certain number of sudden cardiac deaths. Nonetheless, comparisons between the cardiac rupture group and the myocardial infarctions without rupture may still be valid.

Results

The results of this study will be presented under various headings relevant to the yearly distribution of the cases with rupture and significant anatomic and clinical features.

Annual Distribution of Cardiac Rupture

Among the 44 cases of cardiac rupture occurring over the 10-year span of the study, 28 cases occurred in the first 5 years and 16 cases in the last 5 years. It should be recalled that in the present study all patients with a history of cardiac resuscitation have been excluded, and this form of treatment was employed more often in the recent past than formerly. The highest incidence of cardiac rupture among all cases having an acute myocardial infarction (4.2%) occurred in 1961, while the lowest incidence (1.3%) occurred in the last year of the study, 1968.

Age and Sex

In table 1, the sex distribution and age range of the cases with cardiac rupture are compared with the two control groups: group A (those having myocardial infarction without rupture), and group B (those having no myocardial infarction). There are no statistically significant differences, as judged by P values, between the mean age of males and females in any of the groups. Moreover the mean ages for the males and the females are the same in the cardiac rupture and group A populations. The group B patients appear to

| Table 1 |
|---|
| **Age and Sex Comparisons of Groups** |

| Group               | Sex | No.  | Age (yr)  | Mean age (± sd) |
|---------------------|-----|------|-----------|-----------------|
| Cardiac rupture     | M   | 20 (45.5%) | 52–84   | 67 (± 11)       |
|                     | F   | 24 (54.5%) | 54–87   | 73 (± 11.8)     |
| Group A*            | M   | 53 (63%)  | 48–95    | 68 (± 9.5)      |
|                     | F   | 33 (37%)  | 52–94    | 74.5 (± 8.8)    |
| Group B†            | M   | 45 (51.1%) | 18–85   | 61.5 (± 14)     |
|                     | F   | 43 (48.9%) | 33–90   | 68.5 (± 15.2)   |

*Patients with myocardial infarction.
†Patients without myocardial infarction.
be younger. Cardiac ruptures appear to be approximately evenly divided between males and females, while in the population of myocardial infarctions without rupture there is a male preponderance in the ratio of approximately 2:1.

Relationship of Cardiac Rupture to Age of Infarct

Among the 1845 cases (21.4%) of myocardial infarction in the 10-year period under study, 609 hearts (33%) had acute lesions (less than 4 weeks of age), 856 (41%) were healed, and 380 hearts (26%) were judged to have infarctions of distinctly different ages meriting the designation acute and healed. The 44 cases with myocardial rupture represent 2.4% of all myocardial infarctions. The control group A of 88 unruptured myocardial infarcts disclosed: 29 (33%) acute lesions, 29 (33%) acute and healed infarctions, and 30 (34%) healed infarctions.

Turning to an analysis of the 44 patients with myocardial infarction and cardiac rupture it is found that 34 (77.3%) occurred in the 609 hearts having only acute myocardial infarction, representing 5.5% of this group. Nine (20.5%) were found in the 380 hearts having both acute and healed lesions, representing 2.3% of this group. When all hearts having any acute lesion, including those with only an acute lesion and those with both acute and healed lesions are considered together, it is found that 4.3% suffered myocardial rupture. In no instance were there ventricular aneurysms associated with the rupture in these hearts with acute infarction. Among the 856 hearts with healed myocardial infarction, one rupture through a ventricular aneurysm was encountered representing 0.11% of this group. These data are presented in table 2.

An attempt was made on morphologic grounds to estimate the age of the acute infarct at the time of rupture. The criteria were those used by Mallory, White, and Salcedo-Salgar. The morphologic estimate of the infarct age was also compared with estimates based on duration of symptoms. The analysis disclosed the following: an infarction of less than 24 hours, one case by morphologic

| Age of MI | Cases with MI | Cases with rupture |
|-----------|---------------|--------------------|
| Acute MI  | 609           | 33                  | 34                  | 5.5* |
| Acute and healed MI | 380         | 26                  | 9                   | 2.3* |
| Healed MI | 856           | 41                  | 1                   | 0.11 |
| Total     | 1845          | 100                 | 44                  |      |

*The difference between the incidence of cardiac rupture in acute MI and acute and healed MI is statistically significant (P < 0.001).

and two cases by estimates based on symptoms; 24–48 hours, nine and 12 cases, respectively; 3–5 days, 21 and 14 cases; 6–15 days, 11 and 10 cases; more than 15 days, two and two cases; and unknown, none and four cases, respectively.

Size and Site of Infarction

The size of the infarct in the cases with myocardial rupture ranged from lesions about 1 cm in diameter to massive involvement of virtually the entire left ventricle, mean 4–5 cm. In 27% of the cases the lesion was less than 3 cm in diameter. The data clearly indicate that very small lesions are capable of rupture. Indeed approximately 16% of the nonrupture

Table 3

| Site         | Cardiac rupture | Group A |
|--------------|-----------------|---------|
|              | No.  | %    | No.  | %    |
| Anterior     | 5    | 10.9 | 11   | 11.8 |
| Anteroseptal | 9    | 19.4 | 15   | 16.2 |
| Anterolateral| 9    | 19.4 | 7    | 7.3  |
| Posterior    | 9    | 19.4 | 19   | 20.6 |
| Posteroseptal| 2    | 4.6  | 11   | 11.8 |
| Posterolateral| 7  | 14.8 | 6    | 6.2  |
| Lateral      | 1    | 2.3  | 6    | 6.2  |
| Septal       | 1    | 2.3  | 4    | 4.1  |
| Massive L V  | 2    | 4.6  | 11   | 11.8 |
| Massive L V + R V | 1 | 2.3 | 2    |      |
| Massive L V + R A | 0 | 2    | 2    |      |
| Total        | 46*  |      | 94†  |      |

*In two cases, two different areas were involved.
†In 10 cases, two different areas were involved.
cases were considered to be massive, while in the cases of rupture only 7% were of this size. The site of the infarct is presented in Table 3. Here the location is compared in the cardiac rupture patients with the lesions in group A, patients having a myocardial infarct but no rupture. The topographic distribution of the lesions in these two groups runs essentially parallel. In Table 4 the site of infarction and rupture is classified according to the predominant location of the lesion, again comparing the series of cardiac rupture with those having no rupture. Here it is seen that the anterior wall was the most common site of infarction, followed closely by the posterior wall of the left ventricle. Also evident is the fact that the anterior wall was the most common location of rupture (36.3%) followed closely by the posterior wall (30%). Rupture through the septum occurred in four (9%) of the series.

**Coronary Artery Disease**

As might be anticipated all of the cases with cardiac rupture had moderate to severe coronary atherosclerosis. In 29 cases a recent occlusion was found in one of the major coronary trunks. Thus occlusion was not identified in approximately 34% of the cases. When occlusions were present they had the following distribution: left anterior descending, 15 (52%); left circumflex, eight (26%); right coronary, six (19%); and one additional occlusion being found in the left marginal branch. In all cases histologic examination disclosed that the final occlusive episode represented thrombosis superimposed upon advanced atherosclerosis. There were no instances where coronary intramural hemorrhage alone caused the final occlusion, although it is not possible to exclude such a complication as the trigger event precipitating the ultimate thrombosis.

**Weight of Heart**

Table 5 presents the mean heart weight among the patients in the cases with cardiac rupture and in those without rupture. For comparison a random group of patients without myocardial infarction is presented as group B. There appear to be some highly interesting differences among these groups. The mean heart weight for males and females in patients suffering a cardiac rupture was 404 g; in those having an infarction without rupture it was 491 g; while in the control patients without cardiac disease the mean heart weight was 376 g. Correlated with these cardiac weights was the left ventricular thickness of 1.37 cm in the cases with rupture, 1.52 cm in the cases of myocardial infarction without rupture, and 1.40 cm in the cases having no myocardial infarction. It would

**Table 4**

| Site of infarct | Cardiac rupture | Group A | Site of rupture |
|----------------|-----------------|---------|-----------------|
|                | No. | %   | No. | %   | No. | %   |
| Anterior       | 26  | 30.3| 48  | 23.4| 16  | 36.3|
| Posterior      | 21  | 26.1| 51  | 28.2| 13  | 30  |
| Lateral        | 20  | 23.2| 34  | 18.6| 8   | 18  |
| Septal         | 15  | 19.2| 43  | 24.6| 4   | 9   |
| RV             | 1   | 1.2 | 2   | 1.1 | 1   | 2.2 |
| RA             | 0   | 2   | 1.1 | 2   | 4.5 |

*Unknown.

**Table 5**

| Cardiac rupture | Group A | Group B |
|-----------------|---------|---------|
| Case            | Weight (g) | Mean weight (±sd) | Weight (g) | Mean weight (±sd) | Weight (g) | Mean weight (±sd) |
| Males           | 230–700  | 408 (±78) | 280–800  | 507 (±129) | 290–600  | 412 (±95) |
| Females         | 200–600  | 400 (±84) | 200–700  | 459 (±123) | 200–600  | 358 (±88) |
| Acute MI        | 250–700  | 407 (±188) | 250–750  | 470 (±125) | 470–580  | 485 (±115) |
| Healed MI       | —        | 450*     | 250–750  | 470 (±125) | —        | —        |
| Acute +         | 250–580  | 382 (±92) | 250–700  | 485 (±115) | —        | —        |

*Only one healed MI sustained a cardiac rupture.
appear that in the group of cases of myocardial infarction with rupture the heart weight closely approximated those of patients having no myocardial infarction. It was, however, significantly lighter than those having an infarct but no rupture. The possible significance of these differences is discussed later.

Clinical Data

Adequate clinical records were not available on all patients because some entered the hospital acutely ill and died soon afterward. Sufficient clinical information was available to extract certain findings in 43 of the patients with cardiac rupture, in 84 patients having myocardial infarction but no rupture, and in 85 patients having no myocardial infarction (control group B). Data on the incidence and severity of hypertension were collated. Hypertension was defined as any diastolic blood pressure over 90 mm Hg present on at least two occasions. On this basis a history of hypertension documented by at least two blood pressure recordings prior to the myocardial infarction was present in 15 or 35% of the cases of cardiac rupture and 30% of the cases of group A who had acute infarcts. Of interest is the fact that females had such a history of prior hypertension twice as frequently as males among cases with myocardial rupture, but in the group A there was no significant sex difference (table 6). Of the 15 cases of cardiac rupture with the history of high blood pressure, 11 remained hypertensive after the infarct. Six additional cases not having had a previous history of hypertension developed elevated blood pressure postinfarction. Among the group A cases of 17 with high blood pressure, five remained hypertensive after the infarct. Three additional cases developed elevated blood pressure postinfarction. From these data it is evident that almost 40% of the patients who suffered a cardiac rupture were hypertensive postinfarction. Once again there was a striking female preponderance in this phenomenon (11 females, six males). Less than 15% of those in group A who did not have a cardiac rupture were hypertensive after infarction. There was no statistically significant difference in the frequency of preinfarction hypertension between the cases with rupture and those in group A, but the incidence of hypertension after infarction in the group with cardiac rupture was significantly higher than in group A (P < 0.01).

It was deemed of interest to determine how many of the patients in the various groups were diabetic and how many had cirrhosis of the liver. Approximately 9% of the patients in the cardiac rupture group were diabetic as compared with 18% in groups A and B. The difference in incidence of diabetes between group A and B was significant (P < 0.05). None of the patients suffering cardiac rupture was found to have cirrhosis of the liver. In contrast, three of 88 in group A were cirrhotic, and 10 among the 88 patients in group B had this form of liver disease. Some aspects of the possible relevance of these observations are discussed later.

There has been a long-standing controversy over the possible effect of treatment of

### Table 6

| History of Hypertension | Cardiovascular rupture (N = 43) | Group A (N = 58) | Group B (N = 88) |
|-------------------------|-------------------------------|----------------|-----------------|
| Preinfarction:          |                               |                |                 |
| Males                   | 5                             | 9              | 3               |
| Females                 | 10                            | 8              | 3               |
| Total                   | 15 (34.8%)                    | 17 (29.5%)     | 6 (6.8%)        |
| Postinfarction:         |                               |                |                 |
| Males                   | 6                             | 4              |                 |
| Females                 | 11                            | 4              |                 |
| Total                   | 17 (39.5%)                    | 8 (13.7%)      |                 |
myocardial infarction on the development of cardiac rupture. In our series, 37% of the patients who sustained a cardiac rupture had been treated with digitalis, while in the nonrupture group A the comparable frequency was 47%. Anticoagulants had been used in 25% of the cardiac rupture group and in only 14% of the nonrupture group.

Discussion

The incidence of cardiac rupture in myocardial infarction as reported in the literature varies widely. Spierkerman, Brandenburg, Achor, and Edwards in 1962 analyzed a series of postmortem examinations derived from a community of 30,000 individuals. In this analysis they encountered 87 adult cases of acute myocardial infarction and cited a frequency of cardiac rupture among these of 24%. No explanation is offered for this remarkably high incidence of cardiac rupture in acute myocardial infarction. In contrast, Yater et al. cited a frequency of 3.3% of cardiac rupture among a group of 454 males studied at the Armed Forces Institute of Pathology. The average age of this sample was 43.2 years. In general, the highest incidences have been reported among the elderly, psychotics, and among series of cases studied by coroners. In the series reported here the incidence of myocardial rupture was 5.5% in patients having only an acute myocardial infarction and 3.8% in those having both old and acute lesions. This frequency is somewhat lower than the average incidence found in the literature, which ranges between 7 and 9%. Mather, Mallory, and Laurenzi made a study of the autopsy population at this same institute in 1956 and cited a significantly higher incidence of 11.5%, almost twice the rate encountered in the present study. This earlier report included cases derived from the medical examiner's files. Our exclusion of these cases undoubtedly accounts in some part for our lower frequency. Moreover, we also excluded five cases of cardiac rupture following the use of intracardiac injections during the terminal phase of treatment of this disease. It was felt that such treatment, in some way, might have potentiated or contributed to myocardial damage, and such cases were excluded. Better medical care could have contributed to the lower frequency of myocardial rupture found in this present analysis as compared with the earlier study of Mather et al. Indeed, among the 44 cases of cardiac rupture reported here 28 occurred in the first 5-year period with only 16 in the last 5 years. Griffith, Hegde, and Oblath have also suggested that better medical management reduces the frequency of myocardial infarction, but a contrary opinion has been expressed by others.

There is general agreement that rupture rarely occurs in a healed myocardial infarction. When it is encountered, it is usually associated with aneurysmal dilation of the healed infarction as occurred in our single case of rupture in a heart having only an old healed lesion and an aneurysm at the site of the scar.

Myocardial infarction is well known to be predominantly a disease of males. Among the 88 cases of myocardial infarction in group A, 37% occurred in females, the remainder in males. Yet, among the 44 cases with cardiac rupture more than half, 24 (54%), occurred in females. The greater risk of cardiac rupture following myocardial infarction in females has been noted by others. Among the many attempts to explain this phenomenon perhaps the most credible is that of Zeman and Rodstein. They stated, "With a retarded age of onset of coronary artery disease, they lack the protective effects provided by the increased collateral circulation of the myocardial fibrosis and the muscular hypertrophy which develop with long standing coronary artery disease."

It should be noted that, on the average, females suffering cardiac rupture are older than males. In the present study the mean age for males suffering rupture was 67 years while that for females was 73.7 years. While this difference does not have a statistically valid P value (>0.2) it may nonetheless suggest a trend that would be borne out by a larger sample. However, the excess of females in the
rupture group as compared with the sex distribution of group A is significant at the level of \( P < 0.005 \).

Several other observations are worthy of note. The average heart weight among the patients with myocardial rupture was 404 g. In group A (myocardial infarction without rupture), the average heart weight was 491 g. Furthermore, the average heart weight of group B (not having myocardial infarction) was 376 g. Patients with ruptured myocardial infarctions have an average heart weight comparable to patients without myocardial infarction and this average is approximately 100 g less than those having myocardial infarction without rupture. This observation is of greater interest since hypertension in our studies was more prevalent in patients with cardiac rupture than in those without such a complication. This surprising concurrence, namely a higher incidence of hypertension and a lower incidence of cardiac hypertrophy in patients with ruptured myocardial infarction, is in agreement with the studies reported by others.\(^3, 7, 18\) The possibility is raised that patients who do not have cardiac enlargement are at a higher risk of rupture following infarction.

Often asked is the question, does the size or location of the infarct materially modify the risk of rupture? The present study would tend to answer such questions negatively. Myocardial rupture was encountered in infarcts ranging in size from 1 cm in greatest diameter to those with massive involvement of almost the entire left ventricle. Indeed massive infarction was more frequent in those who did not sustain a rupture (see table 3). In approximately one quarter of the cases the size of the infarct was less than 3 cm in greatest diameter. Moreover, location did not seem to predispose to rupture since the distribution of the infarcts in our cases with rupture corresponded quite closely to that of myocardial infarctions without rupture. One possible difference is to be noted. While the septum was involved in acute infarction in 19% of our cases, only 9% of ruptures occurred in this site. It has been previously noted that the septum is relatively spared from rupture in acute infarction.\(^21\)

The importance of a history of hypertension in the pathogenesis of cardiac rupture has been noted by many others.\(^3, 4, 7, 16, 19, 20\) Our data do not indicate any significant difference between the incidence of myocardial rupture following infarction in patients having or not having a previous history of hypertension. In the cardiac rupture group of patients 35% had a history of hypertension, while in the group of patients without rupture the incidence was 30%. Of interest is the observation that hypertension following an acute myocardial infarction was present in 40% of the patients suffering cardiac rupture, while it was present in only 14% of those having infarction who did not suffer a rupture, suggesting a possible causal relationship. Postinfarction hypertension as a predisposition to cardiac rupture has been noted by many others.\(^8, 18, 23\) However, others do not concur that the persistence of hypertension is an important contribution to postinfarction rupture.\(^6, 19\) Most of our patients having persistent postinfarction hypertension were females.

The report by Jetter and White in 1944 which called attention to the high incidence (73%) of rupture of the heart in patients in mental institutions having an acute myocardial infarct raised the issue of whether physical activity postinfarction might predispose to cardiac rupture.\(^16\) In our analysis of this problem approximately 50% of the cases of cardiac rupture and an equal percentage of patients having infarction without rupture were admitted to the hospital within the first 24 hours of onset of symptoms. In other words, there was no greater delay in hospital admission after the onset of the disease in the patients who later suffered a myocardial rupture. There was then no evidence from this study which would indicate that delay in hospitalization played an important predisposing role. An additional question has been raised about the role of therapeutic agents such as digitalis and anticoagulants in the pathogenesis of cardiac rupture.\(^5, 24, 25\) In this survey we can only express some broad trends
since the clinical data were not complete on many cases. With respect to digitalis, 37% of the patients who suffered rupture were treated with this agent while 47% of the control group of infarcts without rupture were similarly treated. However 25% of the group with the cardiac rupture were treated with anticoagulants while only 14% of the control group were so treated. The number of cases involved in both the digitalis and anticoagulant groups is too small to have statistical significance, and the data are only reported for interest. Several additional clinical correlates merit comment. Diabetes mellitus and its associated atherosclerosis as a predisposition to myocardial infarction has been documented many times. However, it is of interest that, as in the series of cases reported by London and London, only 9% of our cases with cardiac rupture had diabetes, while in the control group not having cardiac rupture the incidence of diabetes was twice as high (18%). Could the earlier development of more severe atherosclerosis act as a protective mechanism, as has been suggested by Zeman and Rodstein? The relationship of cirrhosis of the liver to the development of atherosclerosis has long been questioned. Some authors have noted any difference in the severity of atherosclerosis between age-matched cirrhotics and noncirrhotics, while others have reported less severe atherosclerosis in cirrhotics. On the proposition that cirrhotics develop less severe atherosclerosis it is of interest that none of the patients with cardiac rupture in our series had cirrhosis of the liver. In contrast, three (3.3%) of the control group with unruptured myocardial infarction had cirrhosis, and 10 (11%) of the nonmyocardial infarction controls had cirrhosis. There is no statistical significance to these small numbers, but it is of interest however that the patients free of myocardial infarction had a much higher incidence of cirrhosis than the groups with infarction whether ruptured or not. However, it should be noted that our patients with cirrhosis but no myocardial infarction were on the average 5 years younger than those with myocardial infarcts. It could be argued that had they lived longer they would have developed a higher incidence of myocardial infarction. Nonetheless in a hospital having about 11% incidence of cirrhosis in the general autopsy population, the rarity of cirrhosis of the liver in our patients with myocardial infarction is of interest.

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