Cardiac Hypertrophy in Acute Myocardial Infarction

A Study Based on 100 Autopsied Cases

By EDWARD C. ZAINO, M.D., AND SIDNEY H. TABOR, M.D.

The literature presents conflicting views regarding the role of coronary artery disease in the etiology of cardiac hypertrophy.1-5 A presumptive clinical diagnosis of cardiac hypertrophy is often based on information derived from radiographic, electrocardiographic, and angiocardiographic studies, and while these modalities give considerable assistance, they are subject to some degree of error.6,7 Autopsy findings provide the basic incontrovertible features of gross evaluation of heart weight and thickness of chamber walls, and histology.

Utilizing autopsy material derived from accidental and operative deaths, Smith8 found that the average heart weight of all adult age groups was 294 grams for the male, 250 grams for the female. He noted that the weight of the heart increases with body weight. Gross et al.2 considered 400 grams or more as indicative of cardiac hypertrophy.

The purpose of the study reported herein is to record our findings from examination of autopsied material of patients who had acute myocardial infarction.

Material and Methods

The weight of the heart, clinical history of blood pressure readings, age, and sex were analyzed from the hospital and autopsy records of 100 consecutive patients with fatal acute myocardial infarction who had been hospitalized and autopsied at Meadowbrook Hospital during the period 1955 to 1961. Cases were selected for this study solely on the autopsy finding of acute myocardial infarction. Other pathologic conditions that cause cardiac hypertrophy, such as rheumatic and syphilitic cardiovalvular disease, as well as congenital lesions were excluded.

Using heart weight as a measure of cardiac hypertrophy, hearts weighing more than 400 grams were considered hypertrophied. After careful scrupulous study of the case histories, patients were diagnosed as hypertensive if the systolic blood pressure readings were 150 mm. Hg or more, or the diastolic readings were 100 mm. Hg or higher.

The classification of patients according to age and sex is given in Table 1.

Results

Cardiac hypertrophy was found in 70 patients in our series, as shown in Table 2, thus confirming the premise that cardiac hypertrophy is frequently associated with acute myocardial infarction. Similar findings were presented in a recent report by Kannel et al.9 on the Framingham study; "A pattern of left ventricular hypertrophy by electrocardiogram was noted to be associated with a two- or three-fold increase in risk of development of CHD [coronary heart disease] at given hypertensive blood pressure levels in men."

Of our group manifesting cardiac hypertrophy, 41 cases (59 per cent) were normotensive; this is especially noteworthy considering that hypertension frequently exists without cardiac hypertrophy. Hypertension in 56 per cent of the females in our series and in 30 per cent of the males may mean that hypertension is a more significant factor in the development of myocardial infarction among women than men; this corroborates the results of the Framingham Study9 where-in hypertension was noted to represent a

Table 1

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Males</th>
<th>Females</th>
</tr>
</thead>
<tbody>
<tr>
<td>30-39</td>
<td>6</td>
<td>1</td>
</tr>
<tr>
<td>40-49</td>
<td>10</td>
<td>4</td>
</tr>
<tr>
<td>50-59</td>
<td>18</td>
<td>13</td>
</tr>
<tr>
<td>60-69</td>
<td>18</td>
<td>14</td>
</tr>
<tr>
<td>70-80</td>
<td>9</td>
<td>7</td>
</tr>
<tr>
<td>Totals</td>
<td>61</td>
<td>39</td>
</tr>
</tbody>
</table>
greater risk factor in women than in men in the development of coronary heart disease. Since the major incidence of coronary heart disease in females is in the older age group, cognizance should be taken of the fact that hypertension occurs more frequently with the advance of age. Friedberg\(^2\) found that hypertension is encountered in 40 to 60 per cent of people older than 50. In the 60- to 80-year age group of our series 7 out of 27 (26 per cent) males manifested hypertension, whereas 14 out of 21 (67 per cent) females presented that finding.

Cardiac failure was observed as a frequent complication, a finding previously described by Davis and Blumgart\(^10\) (table 3). As was noted in our previous study,\(^11\) the preponderance of coronary artery disease in the young male is reaffirmed in this present series.

**Table 2**

<table>
<thead>
<tr>
<th>Heart Size</th>
<th>Male</th>
<th>Female</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>9</td>
<td>10</td>
<td>18</td>
</tr>
<tr>
<td>Enlarged</td>
<td>34</td>
<td>7</td>
<td>41</td>
</tr>
<tr>
<td>Total</td>
<td>43</td>
<td>17</td>
<td>60</td>
</tr>
</tbody>
</table>

**Table 3**

<table>
<thead>
<tr>
<th>Sex</th>
<th>Decompensated</th>
<th>Compensated</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Acute</td>
<td>Chronie</td>
</tr>
<tr>
<td>Female</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Male</td>
<td>11</td>
<td>15</td>
</tr>
<tr>
<td>Total</td>
<td>13</td>
<td>19</td>
</tr>
</tbody>
</table>

Discussion

Sundry mechanisms have been proposed to explain the etiology of cardiac dilatation and hypertrophy. Starling’s law of the heart suggests that the output of the heart is determined by the diastolic fiber length. Bacaner et al.,\(^12\) in commenting on Starling’s law, state that stroke output and cardiac size may vary both independently and inversely under normal physiological conditions. They felt that Starling’s law is not applicable to the regulation of the in vivo mammalian heart. Their own work has shown that the coronary blood flow influences the heart size, the strength of contraction, and the adaptive mechanisms to increased work loads. Hass and his co-workers\(^13\) found that hypertrophy is usually accompanied by an increase in the blood supply. This is particularly noticeable in young people when massive generalized or local cardiac hypertrophy occurs with comparably enlarged coronary arteries. It was suggested that myocardial hypertrophy in the older age groups may be restricted by the limited capacity of diseased arterial channels to enlarge, and the encroachment of athero-arteriosclerotic processes on the existing and newly established collateral circulation.

When patients with hypertension and other types of heart disease were excluded, McCain et al.\(^14\) found that myocardial infarction and coronary arteriosclerosis produced cardiac hypertrophy. Using 400 grams as the dividing line between normal heart size and cardiac hypertrophy in their study of autopsied material, they noted that 82 per cent of a group of 281 cases of acute myocardial infarction had cardiac hypertrophy, and 74 per cent of the supposedly purely arteriosclerotic group had hypertrophied hearts.

Our findings revealed heart failure, either acute or chronic, present in 32 of the 41 cases of cardiac hypertrophy among the normotensive group. Davis and Blumgart\(^10\) reported that in 16 out of 17 cases of congestive heart failure associated with advanced coronary arteriosclerosis the heart weight was above 400 grams. They concluded that the failure was the cause and not the result of hypertrophy.
The role of hypertension as a concomitant factor in producing cardiac hypertrophy is particularly difficult to evaluate. Not infrequently, patients who manifest hypertension will have blood pressure levels within normal limits after myocardial infarction. Plotz stated, “The absolute decrease is greater in patients with antecedent hypertension. . . . A blood pressure which seems normal may in fact represent a significant drop from an earlier high level.” Since the onset of hypertension is rarely known, the effect of the duration upon the degree of left ventricular hypertrophy is difficult to determine.

Summary and Conclusion

Cardiac hypertrophy occurred in 70 per cent of 100 consecutive autopsied cases of acute myocardial infarction without rheumatic, syphilitic, or congenital cardiovalvular disease.

Coronary artery disease is a common cause of cardiac hypertrophy with or without concomitant hypertension.

References


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