Incidence and Significance of Left Anterior Hemiblock Complicating Acute Inferior Wall Myocardial Infarction

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SUMMARY The hospital course and serial vectorcardiograms of 56 consecutive patients with acute inferior wall myocardial infarction were reviewed. Left anterior hemiblock (LAH) complicating inferior wall myocardial infarction was diagnosed by vectorcardiographic criteria. Seven patients (12.5%) developed LAH between the first and third hospital day, while 49 patients did not. There was no significant difference between these two groups when compared for age, sex, incidence of congestive heart failure, atrial and ventricular arrhythmias, atrioventricular (A-V) block, hospital mortality, and previous hypertension, diabetes mellitus, and myocardial infarction. We conclude that LAH is a relatively common complication of acute inferior wall myocardial infarction, with no apparent effect on the clinical course.

LEFT ANTERIOR HEMIBLOCK (LAH) may be difficult to diagnose in the presence of inferior wall myocardial infarction due to the frequent leftward shift of the frontal plane QRS forces by the infarcted area.1 Vectorcardiographic criteria have been described by Castellanos,2 Kulbertus,3 and Benchimol4 which enable the diagnosis of LAH to be made in the presence of inferior wall myocardial infarction. It is the purpose of this study, using these criteria, to examine prospectively patients with inferior wall myocardial infarction and to determine the incidence and clinical significance of the complication of acute LAH.

Methods

Data were reviewed on 56 consecutive patients admitted to a coronary care unit with acute inferior wall myocardial infarction. Patients with LAH at the time of admission were not included. The criteria for diagnosis were:

1) History of ischemic chest pain.
2) Significant Q waves and ST-segment elevation in leads II, III, aVF, with reciprocal ST-segment depression in leads I and aVL.
   a) Associated lateral wall involvement was diagnosed by appearance of 0.04 sec Q wave and ST elevation in leads V4 and V5.
3) Serial changes of serum glutamic oxaloacetic transaminase, lactic dehydrogenase, and creatinine phosphokinase.

All patients were admitted to the coronary care unit within the first 24 hours following onset of acute chest pain and were constantly monitored using a nonfade television and display, and a V1 monitor lead. A 12-lead electrocardiogram was recorded daily or more often if indicated clinically. Routine coronary care included a constant intravenous infusion of lidocaine (average 2 mg/min) and heparin 75 mg intravenously every six hours, unless specific contraindications were present in a patient.

Vectorcardiograms using the Frank lead system were recorded on day one and day three and on the basis of these studies the patients were separated into two groups.

Group I: Patients with conventional frontal plane vectorcardiographic criteria for inferior wall infarction.5,6
   a) Superior orientation and clockwise rotation of the initial 25 msec vector.
Group II: Patients with frontal plane vectorcardiographic criteria for inferior wall infarction coexisting with LAH.5,6
   a) Clockwise and superior inscription of the initial 25 msec vector.
   b) Counterclockwise rotation of the returning limb, with or without a figure-of-8 morphology.
   c) Maximal QRS deflection vector located in the left superior quadrant.
   d) QRS loop duration less than 0.12 sec.

Complete history and physical examination were done on admission. Data concerning age, sex, history of previous hypertension, diabetes mellitus, congestive heart failure and myocardial infarction were obtained.

A diagnosis of previous hypertension, diabetes, congestive heart failure and myocardial infarction was accepted only if documentation was available.

The hospital course of these patients was carefully followed with regard to the development of atrial and ventricular arrhythmias, congestive heart failure, pericarditis and atrioventricular block. Congestive heart failure following admission was diagnosed by accepted radiographic and clinical evidence of pulmonary vascular congestion which responded to digitalis and/or diuretic therapy. Pericarditis was diagnosed by the presence of a typical pericardial rub which was heard by at least two observers.

Arrhythmias including sion bradycardia (recorded with the patient awake and not immediately following medication), junctional rhythm, atrial flutter-fibrillation, accelerated ventricular rhythm, ventricular tachycardia and ventricular fibrillation were diagnosed according to standard criteria.5 All agonal rhythms were excluded.

Mortality refers to hospital mortality only.

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Received August 2, 1975; revision accepted for publication December 27, 1975.
Results

Forty-nine (87.5%) of the 56 patients studied had acute inferior wall infarction alone (group I) while seven (12.5%) developed LAH following admission (group II) (fig. 1). Four of the seven group II patients developed LAH during the first 24 hours, while three developed this conduction disturbance within 48 hours of admission. Lateral wall involvement was present in 12 patients in group I and one patient in group II. Six patients had electrocardiographic evidence of LAH at the time of hospital discharge.

Three (5.3%) group I patients had right bundle branch block. Only one was documented to be the result of infarction. This patient had had a previous anterior wall infarction and died in cardiogenic shock shortly after admission. One patient (1.7%), also belonging to group I, developed transient non-rate-related left bundle branch block. None of our patients had right bundle branch block and LAH, right bundle branch block and left posterior hemiblock, or isolated left posterior hemiblock.

Table 1. Comparison of Patient Characteristics with Inferior Wall Myocardial Infarction

<table>
<thead>
<tr>
<th></th>
<th>Total (without LAH)</th>
<th>Group I (with LAH)</th>
<th>Group II (without LAH)</th>
<th>Group II (with LAH)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients</td>
<td>56 (100%)</td>
<td>49 (87.5%)</td>
<td>7 (12.5%)</td>
<td></td>
</tr>
<tr>
<td>Age range (years)</td>
<td>40-77</td>
<td>41-77</td>
<td>40-75</td>
<td></td>
</tr>
<tr>
<td>Mean age (years)</td>
<td>57.3</td>
<td>57.9</td>
<td>56.8</td>
<td></td>
</tr>
<tr>
<td>Male/female</td>
<td>43/16</td>
<td>36/16</td>
<td>7/0</td>
<td></td>
</tr>
<tr>
<td>Prior hypertension</td>
<td>17 (30%)</td>
<td>16 (32%)</td>
<td>1 (14%)</td>
<td></td>
</tr>
<tr>
<td>Prior diabetes</td>
<td>4 (7%)</td>
<td>4 (8%)</td>
<td>0 (0%)</td>
<td></td>
</tr>
<tr>
<td>Prior CHF</td>
<td>2 (3%)</td>
<td>2 (4%)</td>
<td>0 (0%)</td>
<td></td>
</tr>
<tr>
<td>Previous MI</td>
<td>3 (5%)</td>
<td>2 (4%)</td>
<td>1 (14%)</td>
<td></td>
</tr>
</tbody>
</table>

LAH = left anterior hemiblock.

Discussion

Left anterior hemiblock may occur with cardiomyopathy,7 congenital heart disease,10 pulmonary embolism,11 and metabolic abnormalities.12 The most common etiology is arteriosclerotic heart disease.9, 10, 12

The greater vulnerability of the left anterior division of
the left bundle branch to ischemia or necrosis is explained by its anatomy (longer and thinner than the posterior division) and to its unique blood supply which depends mainly on the perforating branches of the left anterior descending coronary artery while the posterior division receives its blood supply from branches of both the right and left coronary arteries.\textsuperscript{10}

Left anterior hemiblock complicating acute anterior wall infarction is related to occlusion of the left anterior descending coronary artery.\textsuperscript{10, 14, 15} However, the reason for the appearance of LAH in acute inferior wall infarction is not well understood. It has been suggested that pre-existing left coronary artery disease may produce bundle branch block or hemiblocks if collaterals from the right coronary artery are interrupted.\textsuperscript{14, 16}

Although vectorcardiography may suggest LAH complicating inferior wall infarction,\textsuperscript{2, 4} definite anatomic confirmation is not available. A large inferobasal infarction might account for the same vectorcardiographic pattern. Selvester et al.,\textsuperscript{17} using computer simulation of myocardial infarction, found that a large transmural inferobasal infarction (6–8 cm in diameter) could be associated with superiorly oriented and counterclockwise terminal forces in the absence of any obvious conduction abnormality. However, our patients who developed the LAH pattern had serum enzymes and a clinical course which were essentially the same as the patients without this conduction defect and provided no evidence of extensive necrosis.

The difficulty in correlating electrocardiographically noted conduction defects with specific histopathological lesions has been emphasized,\textsuperscript{16, 18} especially when the mechanisms involved are related to ischemic events. Ischemia and other reversible factors rather than structural damage of the conduction system may be responsible for the different types of A-V and interventricular block.\textsuperscript{18}

Despite lack of anatomic confirmation, indirect evidence is available suggesting that an abnormality of the left anterior division is responsible for the described vectorcardiographic pattern. This evidence includes observation of cases\textsuperscript{2, 5, 4, 25} with pre-existing LAH who then had inferior wall infarction which resulted in a vectorcardiographic pattern similar to the pattern described in our group II patients. The appearance of this vectorcardiographic pattern after artificially induced atrial premature beats (with LAH aberrancy) in patients with acute inferior wall infarction\textsuperscript{8} provides additional evidence. Transient occurrence of the LAH vectorcardiographic pattern\textsuperscript{2} in patients with inferior wall infarction also suggests a conduction abnormality rather than evolutionary changes of myocardial necrosis.

Left anterior hemiblock is the most common conduction disturbance complicating all types of acute infarction,\textsuperscript{14, 19, 20} with an incidence ranging from 4% in the series of Scheinman and Brenman\textsuperscript{19} to 15.2% in the series of Marriot and Hogan.\textsuperscript{19} Previous studies have not examined the incidence and effects of acute LAH complicating the course of inferior wall infarction alone.

We noted acute LAH complicating inferior wall infarction in 12.5% of our cases. This relatively high incidence is probably due to our systematic use of the vectorcardiogram which made possible its identification in the presence of inferior wall infarction.

Our study also showed no significant difference between the groups with and without LAH with regard to age, sex,
history of previous myocardial infarction, hypertension, diabetes mellitus, and congestive heart failure.

The prognosis of isolated LAH in acute infarction appears to be benign in contrast to the poor prognosis of bifascicular block and pump failure. Our study, which deals specifically with inferior wall infarction, also shows that development of acute LAH does not affect the in-hospital survival or increase the incidence of pericarditis, congestive heart failure, atrial and ventricular arrhythmias or atrioventricular block.

Awareness of the problem posed by concealment of inferior wall infarction by LAH is of clinical importance. Figure 2 illustrates a patient who is not included in our series but does demonstrate this problem. Rosenbaum describes three changes which occur when inferior infarction is associated with LAH: 1) The initial forces of LAH directed at +120° are assumed to be due to early activation of the posteroinferior wall of the left ventricle. Infarction involving these areas may cause disappearance of initial vectors giving rise to a QS in leads II, III and aVF. 2) The infarction does not involve the portions of the ventricle which are first depolarized in LAH. In this situation, the inferior infarction may be entirely concealed but suspected if ST-segment abnormalities are present. 3) The initial forces of LAH are only partially affected, giving rise to a tiny q wave in lead II which may represent the only evidence of inferior wall infarction.

It is concluded that isolated LAH complicating acute inferior wall myocardial infarction is fairly common, occurs early during its evolution, may mask evidence of transmural infarction, and does not appear to affect the clinical course or increase the mortality of the patient.

References

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P Kourtesis, E Lichstein, K D Chadda and P K Gupta

Circulation. 1976;53:784-787
doi: 10.1161/01.CIR.53.5.784

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

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the World Wide Web at:
http://circ.ahajournals.org/content/53/5/784

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