Left Anterior Hemiblock of the 2:1 Type in the Presence of Inferior Wall Myocardial Infarction*

Federico Moleiro, M.D.,** and Iván Mendoza, M.D.†

A case of inferior wall myocardial infarction (IWMI) with left anterior hemiblock (LAH) is presented. The LAH became type 2:1 in the course of the disease. Electrocardiographic signs of LAH in the presence of IWMI are outlined, and the behavior and etiology of the T-wave changes are analyzed.

Left anterior hemiblock (LAH), a conduction defect often associated with ischemic heart disease,† has been described in detail from a clinical and experimental point of view. However, recognition of LAH in the presence of inferior wall myocardial infarction (IWMI) becomes a difficult task, because the latter alone can produce left axis deviation.‡ The present report concerns a single patient with IWMI in progress who developed LAH during the course of the disease. During the evolution of IWMI, association with LAH was intermittent and alternative, a fact which permitted us to reevaluate the electrocardiographic findings of IWMI in the presence of LAH.

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Electrocardiographic features of myocardial ischemia signaled by T-wave changes were also analyzed. Special comments are made on the changes of shape and polarity of myocardial ischemia in the presence or absence of the conduction defect, a phenomenon previously described by others, but one which has not been sufficiently emphasized.

CASE REPORT

An 80-year-old woman was admitted to the coronary care unit with acute myocardial infarction, a diagnosis supported by typical clinical symptoms, electrocardiographic abnormalities, and enzymatic alterations. The ECG on admission (Fig 1) showed sinus rhythm at a rate of 75 beats per minute and diagnostic Q waves in leads 2, 3, and aVF with marked ST-segment elevation and T-wave inversion.

An ECG taken seven days later (Fig 2) showed sinus rhythm at a rate of 85 beats per minute with QRS duration changing from 0.08 second to 0.10 second and the axis shifting in the frontal plane from +30° to -45°. Simultaneously, ST-segment elevation diminished, and the T wave became positive in leads 2, 3, and aVF (cf Fig 1 with Fig 2). The abnormal axis deviation leftward and upward in the presence of a Q1-S3 pattern, and the QS with slurring in leads 2, 3, and aVF are very probably due to an associated LAH-type conduction defect.

Figure 3 shows an ECG 14 days after admission. Here, the changes observed in the second ECG (Fig 2) alternate with the pattern that appears on the first (Fig 1). The upper division of the left bundle carries one of every two oncoming impulses. The QRS complexes with the LAH pattern (A in Fig 3) alternate with complexes without the LAH pattern (B in Fig 3). From Figure 3, it is obvious that when LAH is present, the QRS complex in lead 3 has a larger wave than in the complexes with only the IWMI pattern. The T wave is positive in leads 2, 3, and aVF when LAH is present and negative (ischemic type) when the pattern is solely that of IWMI. Fifteen days after admission, the LAH disappeared.

DISCUSSION

The electrocardiographic findings of LAH in the presence of IWMI are abnormal deviation of the electrical...
axis leftward and upward (further than −30°) in the frontal plane, QS complexes in lead 2, and a QT-S pattern. The presence of an initial small and slurred R wave, instead of the classic low-voltage QS complexes in lead 2 and S waves of high voltage in leads 2 and 3, has been described when IWMI is associated with LAH.

In our case, as shown in Figure 1, the low-voltage QR complexes in leads 2, 3, and aVF changed to a slurred QS morphology, abnormal left axis deviation, a wider QRS complex (from 0.08 to 0.10 second), and higher voltage of the S wave in leads 2, 3, and aVF (Fig 2). These findings are in agreement with previous reports.

The absence of an initial small R wave in leads 2, 3, and aVF when IWMI is associated with LAH is due to the fact that the area activated during the first 20 msec in the inferior wall becomes involved with the necrotic tissue. Figure 3 shows the QRS morphology changing from beat to beat; IWMI complexes with LAH are labelled A, and complexes labelled B are those with IWMI only. The sequence occurred seven days after the ECG shown in Figure 2, when the patient was clinically better and repolarization disorders in the ECG had diminished. The LAH appeared in the initial days of the infarction and disappeared three weeks later.

Undoubtedly, there is a relationship between improvement of clinical symptoms, reduction of repolarization disorders, and disappearance of the conduction defect. The block decreased as ischemic signs disappeared, suggesting that the etiology of LAH was ischemic, the so-called phase-3 block. It is interesting to point out that every time that LAH was present, the T wave was positive in leads 2, 3, and aVF, concealing signs of inferior subepicardial ischemia. This occurred in a beat-to-beat sequence when LAH was of the 2:1 type. It is logical to assume that in the presence of delayed activation in the anterolateral wall, as a consequence of LAH, the posteroinferior areas are activated earlier and, therefore, repolarized earlier, too, originating positive T waves in leads 2, 3, and aVF in the same way as secondary T-wave changes occur in complete bundle-branch block. Thus, positive polarity of the T wave in leads 2, 3, and aVF in the presence of LAH is interpreted here as a secondary change due to LAH and not to the
Intra-His Bundle Block Complicating Acute Inferior Myocardial Infarction*

Antoine T. Nasrallah, M.D., and Earl F. Beard, M.D.

The unexpected findings of atrioventricular block or delay in the His bundle and proximal branches are described in a 51-year-old man with acute inferior myocardial infarction with narrow QRS complexes. With the information from the His bundle electrogram, the site of atrioventricular block is precisely localized.

It has been suggested that heart block in inferior myocardial infarction may be due to the effect of increased vagal tone on atrioventricular conduction, to ischemia of the atrioventricular node and His bundle, or to both.\(^1\) In first, second, or complete atrioventricular block with normal-width (<0.10 second) QRS complexes, the conduction delay is generally above the His bundle. Unfortunately the standard electrocardiogram sometimes fails to localize the site of delay or block precisely.

The purpose of this report is to describe the physiologic site of atrioventricular block in a 51-year-old man with acute inferior myocardial infarction with narrow QRS complexes as delineated by recording the His bundle electrogram.

METHOD

His bundle electrograms were obtained in this patient as part of a prospective study of patients with conduction disturbances. The His bundle electrograms (USCI No. 5) were performed under local anesthesia using a bipolar catheter introduced percutaneously via the right femoral vein using conventional techniques.\(^3,4\) The catheter was positioned across the tricuspid valve under fluoroscopic control and manipulated to achieve optimal recording of the His deflection. All records were obtained on a multichannel-oscilloscope photographic recorder (Electronics for Medicine VR-6) at a paper speed of 100 mm/sec with filter frequencies of 0.1 to 250 Hz for ECG leads and 30 to 250 Hz for electrogram recording. The low right atrium-to-His bundle (LRA-H) potential interval was measured from the onset of the low right atrial depolarization to the first rapid deflection of the His bundle potential. The His-to-ventricle (H-V) interval was measured from the first rapid deflection of the bundle of His potential to the earliest ventricular activity either on surface or intracardiac recording. The right bundle-to-ventricle (Rb-V) interval was measured from the initial rapid deflection of the first potential after the His deflection to the earliest ventricular activity. When split His bundle potentials were recorded with intact atrioventricular conduction, measurements were made of the LRA-H, the H-V, and the Rb-V intervals. Normal range for the LRA-H interval during sinus rhythm in our laboratory is 80 to 150 msec; for the H-V interval, 35 to 55 msec; and for the Rb-V interval, 15 to 29 msec.

CASE REPORT

A 51-year-old man was admitted to the coronary care unit of St. Luke's Episcopal Hospital for acute sudden onset of chest pain of 24 hours' duration. His ECG on admission showed acute inferior myocardial infarction. Three hours after admission, the patient had Wenckebach or Mobitz type I atrioventricular block which progressed to transient complete atrioventricular block with a ventricular rate of 40 beats per minute. Atropine was given intravenously with good response. On the second hospital day, the patient's standard ECG demonstrated a sinus rhythm at a rate of 80 beats per minute with a prolonged P-R interval of 0.32 seconds. The QRS complexes were narrow and measured 0.08 seconds. His bundle electrograms on the second hospital day showed two high-frequency potentials recorded between the atrial and ventricular electrograms representing "split" His potentials (Fig 1) as defined by Narula et al.\(^5\) Each atrial depolarization was followed by a biphasic proximal His bundle potential (H\(_1\)) with LRA-H\(_1\) interval of 100 msec, H\(_1\)-H\(_2\) interval of 95 msec, and H\(_2\)-V interval of 127 msec. In addition, a separate biphasic spike was recorded between the H\(_2\) and V, probably representing a right bundle potential of Rb-V in-