Left Anterior Hemiblock Obscuring the Diagnosis of Right Bundle Branch Block in Acute Myocardial Infarction

SAMUEL Sclarovsky, M.D., RUBEN F. LEWIN, M.D., BORIS STRASBERG, M.D. AND JACOB AGMON, M.D., F.A.C.C.

SUMMARY Thirty cases in which transient left anterior hemiblock (LAHB) obscured the diagnosis of right bundle branch block (RBBB) appearing during the first days of an acute myocardial infarction (AMI) are presented. Twenty-eight of the patients with AMI had a clear septal wall involvement, while the remaining two had an anterolateral and lateral wall AMI, respectively. These intraventricular conduction defects developed 2–120 hours (mean 64.9 ± 26 hours) after the acute event, and persisted for 24 hours to 7 days (mean 63.1 ± 35 hours).

The ECG was characterized by a pure LAHB with wide QRS complexes and the presence of RBBB was shown by recording high V₁ and right-sided chest leads. The vectorcardiogram was also useful in several cases.

The clinical course of this type of bifascicular block was transient and benign, with an in-hospital mortality of 6.7%. No patient developed trifascicular or complete atrioventricular block and, therefore, we conclude that prophylactic installation of a temporary pacemaker is not indicated in this type of bifascicular block. The possible role of extracellular potassium released during acute myocardial necrosis in the pathophysiological mechanism of these blocks is discussed.

Materials and Methods

From 2500 patients admitted to our Intensive Coronary Care Unit (ICCU) with definite diagnosis of AMI, 249 patients had LAHB on admission or during hospitalization. From this group, 30 patients (12%) developed LAHB, obscuring the diagnosis of RBBB.

The diagnosis of AMI was established by a history of severe chest pains, appearance of new pathological Q waves and a typical elevation of cardiac enzymes. A 12-lead ECG was taken at least once daily during the hospitalization and the position of the precordial leads was marked on the patient’s chest on arrival to the ICU.

LAHB was defined electrocardiographically as marked left-axis deviation (LAD) of −30° or more, with a Q₃S₂ pattern and small R waves in leads II, III and aV₃. LAHB obscuring RBBB was also defined according to the criteria of Rosenbaum et al.: 1) LAHB with increased QRS duration without any other explicatory cause; and 2) a marked terminal delay oriented slightly to the right in the transverse plane (R' in aV₁, S in V₅, R' in high V₁, or RV₃). This late vector measured more than 0.02 second. The presence of RBBB was confirmed by recording a high V₁ lead and right-sided chest leads. In several cases, we used vectorcardiographic (VCG) recordings to confirm the presence of RBBB. The study began at the time of initial appearance of chest pain.

We performed statistical analysis using the t test.

Results

Our findings are summarized in table 1. Of the 30 patients studied, 19 (63%) were male and 11 (27%) female. Age ranged from 32–80 years (mean 59.4 years). The AMI location was anteroseptal in 20 patients and anterior wall in eight. Of the other two patients, one developed an anterolateral and the second a lateral wall AMI. Nineteen patients (63.3%) had no cardiac symptoms before the onset of the acute event; six were diabetics and five had a duodenal ulcer. One patient had hypertension, another had ischemic cerebrovascular disease and a third had severe chronic obstructive lung disease.

The frontal QRS axis before the appearance of the LAHB obscuring RBBB ranged from −45° to +120° (mean 4.5 ± 38.8). During the presence of LAHB obscuring RBBB, the QRS axis ranged from −30° to −90° (mean −58 ± 17.5) (figs. 1 and 2). In eight patients, a mean frontal axis of at least −30° was

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present before the appearance of the AMI, but all of them had a further left-axis deviation during the LAHB obscuring RBBB (fig. 2). One patient had an incomplete RBBB. After the disappearance of the LAHB obscuring RBBB, the mean QRS frontal axis was in the range of $75^\circ$ to $-75^\circ$ (mean $-7 \pm 34.7^\circ$). The difference between the QRS mean frontal axis before and after the appearance and disappearance of LAHB obscuring RBBB was not statistically significant ($p \geq 0.05$).

The QRS complex measured 0.05–0.08 second (mean 0.066 ± 0.009 second) before the LAHB obscuring RBBB; 0.1–0.14 second (mean 0.111 ± 0.0097 second) during LAHB obscuring RBBB; and 0.06–0.1 second (mean 0.072 ± 0.01 second) after LAHB obscuring RBBB disappeared. In all patients, QRS duration increased 0.02 second or more during LAHB obscuring RBBB. The difference of the QRS width before the appearance and after the disappearance of the LAHB obscuring RBBB was statistically significant ($p \leq 0.025$).

The interval between initial symptoms and the appearance of the LAHB obscuring RBBB was 2–120 hours (mean 64.9 ± 26 hours). The LAHB obscuring RBBB persisted for 24 hours to 7 days (mean 63.1 ± 35 hours). Three patients each developed this type of conduction defect twice.

A commonly observed pattern of disappearance of the LAHB obscuring RBBB consisted of a narrowing of the QRS in all the cases followed by the disappearance of the signs of LAHB in some of them (fig. 2).

In all 30 patients, the demonstration of concealed RBBB was easily obtained with a high $V_1$ recording (fig. 3), and the VCG gave additional proof of the origin of the wide QRS (fig. 4). After the QRS narrowed, it was not possible to show any degree of RBBB in unipolar high and right-sided chest leads or in the VCG.

There was no statistically significant difference between the heart rates before, during or after the appearance of the LAHB obscuring RBBB ($p \geq 0.05$). Carotid massage performed in some cases resulted in slowing of the sinus rate without change in the QRS width or frontal axis.

The in-hospital mortality of this group was 6.7%. One patient died from cardiogenic shock 96 hours after admission to the ICCU and 24 hours after the appearance of the LAHB obscuring RBBB. The second patient died suddenly, probably from ventricular fibrillation, on the twentieth day of hospitalization. Apart from pericarditis in two patients and congestive heart failure in 10, there were no other complications. No patient developed signs of trifascicular block or complete atrioventricular block.

Discussion

In 1973 Rosenbaum described three cases in which the presence of LAHB obscured the diagnosis of RBBB. Another case was described by Heydorn et al. Rosenbaum et al. suggested that an increased QRS duration in the presence of LAHB should be the main clue for suspecting hidden or concealed RBBB; recording additional chest leads one interspace above the conventional level or slightly to the right of $V_1$ may readily uncover the concealed RBBB (fig. 3). A VCG may also be helpful in the diagnosis by showing a

![Figure 1. Anteroseptal anterior myocardial infarction (first strip) complicated by a transient left anterior hemiblock (LAHB) obscuring right bundle branch block (second strip). Characteristic pattern of disappearance: first the QRS narrows (third strip), then the LAHB disappears (fourth strip).](http://circ.ahajournals.org/doi/10.1161/01.CIR.71.2.27)
marked terminal delay oriented slightly to the right in the transverse plane (fig. 4). To the best of our knowledge, such an ECG evolution of LAHB obscuring RBBB during the first days of an AMI has not been reported.

As pointed out by Rosenbaum et al., several other conditions capable of producing the same ECG finding should be excluded: 1) severe left ventricular hypertrophy. This was definitely not present in any of our patients. 2) Focal blocks (infarction or fibrosis of the anterolateral wall of the left ventricle), perinfarction or intra-infarction block. These terms are not clearly defined and are used as another terminology for hemiblocks. However, the transient nature of the LAHB obscuring RBBB in our patients does not support the diagnosis of focal blocks, which seem to be a more permanent phenomenon. In LAHB the passage of the electrical wave from the posterior to the anterior wall may be through the septum; in cases with anteroseptal AMI (28 of our patients) the conduction through the septum could be delayed, causing the wide QRS. However, as shown on the VCG (fig. 4), the actual delay is in the last vector, without the participation of the infarcted septal portion in the last wave front. Another characteristic of this group was the typical interval between the onset

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Abbreviations: A = after RBBB; AH = arterial hypertension; An/La = anterolateral wall; AP = angina pectoris; ASW = anteroseptal wall; B = before RBBB; CHF = congestive heart failure; COPD = chronic obstructive pulmonary disease; CVA = cerebrovascular accident; D = during RBBB; DI = diabetes mellitus; DU = duodenal ulcer; Ex/An = extensive anterior; LW = lateral wall; No = no previous disease; OMI = old myocardial infarction; RA = rheumatoid arthritis.
of the AMI and the LAHB obscuring RBBB, which most of the patients developed after 24–48 hours of the hyperacute stage of the AMI.

Most of the bifascicular blocks occurring in AMI tend to appear during the first hours, and are considered to be of ischemic origin. Intraventricular conduction defects appearing relatively late seem to be less severe and their mortality is lower.9

The pathophysiology of these late blocks is still unclear. Jackrel et al.10 reported that irreversible atrioventricular conduction disturbances after ligation of the anterior septal artery are caused by the presence of total cross-sectional necrosis of the His bundle, and that reversible atrioventricular conduction disturbances are caused by the effect of increased extracellular potassium released by necrotic myocardial cells on viable conducting fibers. The distal His bundle and proximal bundle branches are more susceptible to the depolarizing effect of extracellular potassium than the more proximal conducting tissue. The potassium release from necrotic myofibers is greatest 24 hours after the AMI and the necrotic area gradually loses potassium until the fourth day after the AMI. This phenomenon has been described by Hackel and co-workers.11 The transient nature of these blocks may be explained on the basis of this pathophysiological mechanism and, therefore, they may be considered as metabolic blocks.

The mortality in our series (6.7%) is relatively low, even lower than the mortality reported with acute LAHB alone.12–14 This is in agreement with the relatively lower mortality observed in transient late

### Table 1. (Continued)

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<th>Time of onset (hours)</th>
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bifascicular blocks and in contrast to the considerably higher mortality of the bifascicular blocks appearing early in the course of AMI. These findings support the assumption that LAHB obscuring RBBB is benign and has a better prognosis than other types of acute blocks appearing in the early phase of AMI. Furthermore, because of the lack of progression to trifascicular block (bifascicular block and PR prolongation) or complete atrioventricular block and the transient nature of these blocks, prophylactic pacemaker installation is not indicated in these cases.

**Figure 2.** Evolution of anteroseptal anterior myocardial infarction showing a progressive left-axis deviation (−45°, −60°) (strips 1–4, from the left). The fourth strip shows an electrical axis of −60° with a QRS width of 0.11 second, representing left anterior hemiblock (LAHB) obscuring right bundle branch block (RBBB). In the fifth strip, LAHB persists while the RBBB has disappeared (narrow QRS).

**Figure 3.** Right bundle branch block. The precordial lead is located one interspace above the normal V1 (V1 high) in two patients.

**Figure 4.** Vectorcardiogram (VCG) in a patient with left anterior hemiblock (LAHB) obscuring right bundle branch block (RBBB) showing: top) anterior wall infarction with normal axis without RBBB; center) 36 hours later, appearance of LAHB (frontal (F) plane) and terminal slow vector slightly oriented to the right (horizontal (H) plane) compatible with RBBB; bottom) LAHB and RBBB disappear. S = sagittal plane.
Addendum

Since the manuscript was submitted we have observed four additional cases of LAHB obscuring RBBB. One of the patients was studied with simultaneous two- and three-channel ECGs. Normal and high V1 were recorded simultaneously, showing from the fifth beat a shift of the QRS width and terminal left-superior forces obscuring RBBB. No R' wave was observed before the QRS of the fifth beat (fig. A1). Simultaneous precordial leads V1, V3, V6 were also recorded (fig. A2). The first beat shows a wide QRS with a terminal S wave in V6; the second and third beats show a narrowed QRS without the characteristic pattern of LAHB obscuring RBBB. The fourth and fifth beats show RBBB again and then the block disappears. There is no differences in PR or RR intervals during the appearance or the disappearance of LAHB obscuring RBBB. The same ECG picture was noted in all leads.

Figure A1. Normal and high V1 recorded simultaneously in a patient with left anterior hemiblock obscuring right bundle branch block.

Figure A2. Simultaneous precordial leads V1, V3, and V6 from the same patient as in figure A1.
References

Left anterior hemiblock obscuring the diagnosis of right bundle branch block in acute myocardial infarction.

S Sclarovsky, R F Lewin, B Strasberg and J Agmon

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