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Original Research

Conduction Disturbances in Acute Myocardial Infarction: A Clinical Study and Brief Review of the Literature

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Introduction: Heart blocks may occur as complications of acute myocardial infarction (AMI) and are accompanied by increased in-hospital mortality. The objective of this investigation was to study heart blocks in patients with AMI and to assess their association with clinical features and therapeutic measures.

Methods: Four hundred consecutive patients (263 men, 137 women, mean age 59.6 ± 8.4 years) who were admitted with the diagnosis of AMI were assessed. The initial ECG, recorded immediately after the patient's admission to the emergency department, was considered as baseline. Any heart blocks occurring over the following days were noted by comparing the relevant ECGs with this baseline ECG.

Results: The overall prevalence of heart blocks was 15.8%. There was no significant statistical correlation between the incidence of heart blocks and the patients' age and sex. Although the prevalence of cigarette smoking, hypertension, hypercholesterolaemia and diabetes mellitus in patients with heart block was greater than in patients without, the differences were not statistically significant. The development of heart blocks was more common among those patients treated with thrombolytic therapy (21.1% vs. 12%, $p=0.01$). Also, the development of heart blocks was associated with a significantly lower left ventricular ejection fraction. It was found that 25% of patients who died following AMI had experienced heart blocks, compared with only 13.6% of those who survived ($p<0.01$).

Conclusions: Development of heart blocks has important prognostic significance. The higher prevalence of heart blocks in anterior wall or Q-wave infarctions indicates that the increased mortality following heart block development is probably not related solely to the conduction disturbance itself, but also to the relatively larger infarcted area.

Heart blocks, such as atrioventricular (AV) and intraventricular blocks, are among the most important electrical disturbances which occur following acute myocardial infarction (AMI).[1-6] In these settings, delayed conduction may occur as a consequence of physiological phenomena (for example, increased vagal tone) or pathological processes with varying incidence rates in different populations (first degree AV block 2-12%, second degree AV block 3-10%, third degree AV block 3-7%).[3-19] It has been confirmed that these conduction disturbances are associated with an increased in-hospital mortality rate.[14] On the other hand, it has been stated that these abnormalities do not predict the long-term mortality in patients who survive and are discharged from the hospital after AMI.[16-17] Hence, the important finding of heart block in patients with AMI has some remarkable prognostic implications.[16]

The objective of this investigation was to study heart blocks in patients with AMI who were admitted to the coronary care unit (CCU), to estimate their prevalence, and also their association with clinical features and therapeutic measures.

Methods

During the 19 months of the investigation (between June 2004 and January 2006), we conducted a study of 400 consecutive patients admitted to the CCU of our hospital. All patients had the diagnosis of AMI (based on ECG findings and serum CK-MB alterations) and were admitted to the hospital <4 hours after the onset of symptoms. The diagnosis was established by the presence of typical precordial pain lasting at least 30 minutes, ST-segment elevation or depression >0.1 mV in more than two ECG leads, and an increase in CK of double the normal value (190 U/L). Only those patients who satisfied all three diagnostic criteria (typical chest pain, ECG changes, and CK rise) were enrolled in the study.

Routine clinical and historical data as well as infarct characteristics (location of the infarction, Q or non-Q type, post-AMI ejection fraction) were recorded for all patients.

Electrocardiographic evaluation

All the patients underwent continuous ECG monitoring while in the CCU. Also, a standard 12-lead ECG was recorded on admission (baseline) and was repeated every 8 hours throughout the hospitalisation period (range 3-9 days, mean 4.7 days). AMI was classified as Q or non-Q depending on the presence or absence, respectively, of pathological Q waves in any of the 12 ECG leads. Transmural infarction was considered as anterior or inferior when Q waves appeared in two contiguous leads from V_1 to V_6, or II, III, and aVF.

Heart blocks were diagnosed based on the ECG findings, which were interpreted by an experienced cardiologist who was blinded to the patients' other clinical data. The diagnosis of either left bundle branch block (LBBB) or right bundle branch block (RBBB) and also hemiblocks was made using standard criteria.[20] Heart blocks were classified according to the time of their appearance: they were considered as a "recent-onset" complication if they appeared after admission and were not present on the baseline ECG, or as "old" if they were present on the baseline ECG. Those patients who had old heart blocks were excluded from the study. The heart blocks were also classified into two other groups: AV block (including first degree, Mobitz type I, Mobitz type II, and third degree), and intraventricular blocks (including LBBB, RBBB, left anterior hemiblock [LAHB], left posterior hemiblock [LPHB]). All patients whose AV blocks developed after the administration of beta-blocking drugs were excluded from the study.

Therapeutic measures and echocardiographic evaluation

All patients suffering from ST-elevation AMI (STEMI, according to baseline and subsequent ECGs) underwent thrombolytic therapy. Thrombolytic therapy was administered by the attending physician if no contraindication was detected. Streptokinase (1,500,000 U IV over 45 minutes) was used for this purpose.

The remaining patients had the diagnosis of non ST-elevation AMI (NSTEMI), which was made based on the elevation of serum CK-MB. These patients were treated using heparin (70 IU/kg as the bolus initial dose, followed by a continuous infusion of 15 IU/kg/hr).

Left ventricular ejection fraction and the presence of wall motion abnormalities (such as hypokinesia, akinesia, and dyskinesia) were evaluated in a subgroup of 364 patients (missing data in 36 patients) before hospital discharge (on days 2 or 3) using echocardiography and Simpson's method. Delayed contraction of the septum in patients with LBBB was not considered as abnormal wall motion in this subgroup of patients.

Our patients were not followed up after hospital discharge; however, the in-hospital mortality (death that occurred during the hospital stay) was recorded for all the patients.

Statistical analysis

A descriptive analysis was performed for all variables in order to obtain a frequency distribution. Quantitative variables were expressed as mean ± standard deviation and range. The relationships between categorical variables were evaluated using contingency tables and the chi square test. Between-group comparisons were studied using ANOVA complemented with means equality contrasts using Student's t-test. A p-value ≤ 0.05 was considered significant.

Results

Four hundred patients were included in the final analysis. Of these, 263 (65.8%) were men and 137 (34.2%)

were women. The mean age of the patients was 59.6 ± 8.4 years (range 37-81). The mean time interval between the initiation of patients' symptoms and the recording of the baseline ECG was 2.5 ± 0.4 hours.

The overall prevalence of heart blocks was 15.8% (63 patients). Table 1 shows the frequency of different heart blocks in our population.

There was no significant statistical correlation between the incidence of heart blocks and the patients' age or sex (both $p > 0.05$). In patients with heart block, the prevalence of major cardiovascular risk factors was greater than in those patients without, but the differences were not statistically significant (cigarette smoking 16.3% vs. 15.5%, $p=0.85$; hypertension 16.6% vs. 15.2%, $p=0.72$; hypercholesterolaemia 20.4% vs. 14.2%, $p=0.14$; diabetes mellitus 19.8% vs. 14%, $p=0.14$).

Of the 400 patients, 166 (41.5%) suffered from STEMI and underwent thrombolytic therapy. The remaining patients had NSTEMI (58.5%) and underwent heparin therapy. The development of heart blocks was more common among those patients treated with thrombolytic therapy (21.1% vs. 12%, $p=0.01$). Also, the development of heart blocks was associated with a significantly lower left ventricular ejection fraction ($p<0.01$, Table 2).

Based on the echocardiographic evaluation, 327 patients (81.8%) with heart blocks had a low ejection fraction (<50%). There was no association between the type of heart block and reduced ejection fraction ($p>0.05$). Moreover, the appearance of heart blocks was more common in patients who had evidence of hypokinesia of at least one segment on echocardiographic images (21.1% vs. 6.6%, $p<0.01$). On the other hand, the appearance of hypokinesia on echocardiographic images was more common in patients who had heart blocks (81.8% vs. 54.5%, $p<0.01$). There was a significant relationship between the number of segments suffering from abnormal wall motion and the calculated ejection fraction ($p<0.01$).

Table 1. The frequency of different heart blocks in our population.

Block type	No. of patients (%)	Permanent/transient
First degree	18 (28.6)	5/13
Mobitz I	1 (1.6)	0/1
Mobitz II	0 (0)	0/0
Third degree	3 (4.8)	3/0
LAHB	19 (30.2)	11/8
LPHB	4 (6.3)	2/2
LBBB	12 (19)	9/3
RBBB	6 (9.5)	3/3
Total	63 (100)	33/30

LAHB – left anterior hemiblock; LBBB – left bundle branch block; LPHB – left posterior hemiblock; RBBB – right bundle branch block.

Table 2. The association between ejection fraction (EF) and the development of heart blocks. The relationship between EF and the presence or absence of heart block was statistically significant ($p<0.01$).

EF (%)	Heart block		Total
	No	Yes	
<25	6	4	10
25-34	15	4	19
35-44	51	11	62
45-54	102	21	123
>54	135	15	150
Total	309	55	364

The incidence of heart blocks was significantly higher in patients with a Q-wave AMI than in those with a non Q-wave AMI (66% vs. 20.5%, $p<0.01$). There was also a significant correlation between the type of AMI and the type of heart block ($p<0.05$). Among those patients who had both Q-wave AMI and heart blocks, 70.4% of patients developed intraventricular blocks (including LAHB, LPHB, LBBB, RBBB), whereas 66.7% of patients with both non Q-wave AMI and heart block suffered from AV blocks (AV block degree I-III).

Of the 400 patients, 278 (69.5%) showed electrocardiographic and echocardiographic evidence of anterior AMI, while 169 (42.25%) suffered from inferior AMI. Hence, 47 patients (11.75%) had evidence of both anterior and inferior infarction. There was a significant association between the location of AMI and the type of heart block ($p<0.01$): 79.5% of the blocks in patients with anterior AMI were of intraventricular type, while 68.4% of the blocks which happened after inferior AMI were AV type blocks.

In-hospital course

During the follow-up period, 36 patients (9%) died. It was found that 9 (25%) of those patients who died following AMI had experienced heart blocks, compared with only 63/364 (13.6%) of those who survived. The difference was statistically significant ($p<0.01$). All patients who suffered from first-degree AV block were discharged alive. Thus, all 9 cases of in-hospital death after development of heart block occurred in patients with other types of heart block: 2 cases with third-degree heart block, 2 cases with RBBB, 3 cases with LBBB, 1 case with LPHB, and 1 case with LAHB.

All 3 patients with third-degree heart block (one with extensive anterior AMI and two with inferior AMI) received temporary pacing; two of these patients subsequently died. The only patient (with extensive anterior AMI) who survived was given a permanent pacemaker and was discharged alive.

Discussion

Based on our study, the development of heart blocks is associated with more post-infarction hypokinesia of cardiac walls, a lower ejection fraction, and greater in-hospital mortality. Hence, these easily detectable abnormalities have important prognostic significance. In fact, our study confirms previously reported findings concerning the adverse prognostic significance of heart blocks in AMI.[2-7,21] As is indicated by the higher prevalence of heart blocks in anterior wall or Q-wave infarctions, such increased mortality is probably not entirely related to the conduction disturbance itself, but also to the relatively larger infarcted area.[21] The above mentioned conclusions are supported by the following observations: all patients with first-degree AV block were discharged alive, while 2/3 of patients who developed complete heart block died. Although at first glance it may seem that our reported value (2/3) is too high, it is not greatly different from previous reports. Nguyen et al found that the overall proportion of patients with AMI who develop complete heart block (CHB) is 4.1% (relatively similar to our finding; Table 1) and emphasised that the incidence of CHB complicating AMI has declined appreciably over time, with the greatest decline in these incidence rates occurring during the most recent years (2.0% of patients hospitalised with AMI in 2005 vs. 5.1% in 1975).[22] In their study, patients with AMI who developed third-degree heart block had greater in-hospital mortality than did those who did not develop CHB (43.2% vs. 13.0%).[22] Moreover, Dissmann et al reported that 42% of patients with post-AMI LBBB died during follow up.[23]

Similarly to previous reports, we believe that such increased mortality was probably not entirely related to the conduction disturbance itself, but was also due to the relatively larger infarcted area.[21] For example, Botvinick et al reported that patients with rhythm and conduction disturbances and without congestive heart failure during AMI may follow an uncomplicated or a complicated late clinical course.[24] However, it should be stressed that anatomical variations in structure or regional blood supply are as important as the degree of myocardial damage in determining the occurrence of this conduction disturbance.[21]

LAHB was the most frequent type of intraventricular block and its relative incidence in our study was similar to that in previous reports.[10,11,21] As stated by Basualdo et al, the posterior division of the left bundle is relatively short and thick and hence is less exposed to mechanical trauma than its anterior counterpart. In addition, the posterior division of the left bundle probably receives a double blood supply from both the left anterior descending and the right coronary arteries.[21] These anatomical considerations explain the fact that LPHB is an infrequent complication of AMI and that it is commonly associated with other conduction disturbances. Elizari et al also noted the presence of a frequent association between anteroseptal myocardial infarction and left anterior hemiblock.[25]

Although in our study there was no significant correlation between the patients' age and the incidence of post-infarction heart blocks, Eriksson and his colleagues[13] reported the opposite finding, as did other studies.[26] Moreover, while in previous studies diabetes mellitus was more common in patients with LBBB,[24] our study did not show any significant relationship between major risk factors for coronary artery disease and the incidence of heart blocks.[18] As the number of patients in each subgroup of heart blocks was very low, regression analysis to detect the association between the type of heart block and other variables (ejection fraction, thrombolytic therapy, in-hospital mortality, major cardiac risk factors, etc.) was not performed. Studies with larger series of patients will be necessary to elucidate this matter further.

There are conflicting results concerning the association between the development of heart blocks and thrombolytic therapy. Although Rathor and Gersh showed that the incidence of heart blocks is higher among those patients who had a history of thrombolytic therapy,[16] the observation was not confirmed in the study of Melgare et al.[21] The findings of our study are in accordance with those of Rathor et al. Our explanation is that, in current practice, thrombolytic procedures are less frequently used for those patients who generally suffer from less extensive infarctions; therefore, the association between thrombolytic therapy and heart blocks in AMI patients is secondary to their more extensive lesions. In fact, based on our findings (greater incidence of hypokinesia and lower ejection fraction in echocardiographic analysis, higher in-hospital mortality rate and also higher prevalence of anterior wall and Q-wave infarction in patients complicated by heart blocks), we conclude that developing heart block is an indirect measure of the severity and extension of the disease.

Moreover, it should be noted that the in-hospital mortality rate in our infarcted population was considerably lower than that observed in developed countries.[27,28] This difference can be explained by the fact that our patients were generally younger than those in developed countries.[27-29] However, more precise studies in larger series of patients are needed to confirm this hypothesis. On the other hand, the overall prevalence of heart block following AMI is more common in our population compared with developed countries (14% in the Scandinavian population);[30] the underlying factors contributing to this need to be investigated in the future.

One of the main drawbacks of our study was the fact that there was approximately 2.5 hours' lag between the onset of the patients' symptoms and the recording of the baseline ECG. There is a strong possibility of developing heart block during this lag period and such heart blocks would have been considered as "old" heart blocks and excluded from the study. The other drawback of the study was the fact that, due to cultural limitations, the patients were not followed up after discharge from the hospital.

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