

CLINICAL AND ELECTROCARDIOGRAPHIC VARIABLES ASSOCIATED WITH INCREASED RISK OF COMPLETE ATRIOVENTRICULAR BLOCK IN ACUTE INFERIOR MYOCARDIAL INFARCTION

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Atrioventriküler (AV) blok akut inferior miyokard enfarktüsünün (ME) sık komplikasyonlarında biridir ve bu nispeten sık görülen komplikasyon yüksek mortaliteye neden olması dolayısıyla klinik olarak büyük önem taşır. Bu çalışmada akut inferior ME seyri sırasında görülebilen atrioventriküler tam blok gelişimi ile ilişkili olabilecek klinik faktörler ve elektrokardiyografik parametreler araştırılmıştır.

AV tam blok gelişen 79 hasta ile AV blok izlenmeyen randomize 119 akut inferior ME'li hasta karşılaştırılmıştır. AV tam blok gelişen hastalarda başvurudaki ortalama kan basıncı, trombolitik tedavi oranları ve ejeksiyon oranları düşük, hipertansiyon, senkop, zirve kreatin kinaz (CK-MB) düzeyleri, 3 damar hastalığı, ventriküler aritmi ve hastane içi mortalite oranları daha yüksek olarak bulunmuştur. Akut inferior ME'li 65 ve üzerindeki hastalarda AV tam blok gelişimi 2.2 kat daha yük-

sektir. Ayrıca, RV4'de 1 mm'nin üzerinde ST yükselmesi AV tam blok gelişimi 3.7 kat, DIII'de 2.75 mm'nin üzerinde ST yükselmesi 12.3 kat, DIII'deki ST yüksekliğinin DII'dekine oranının 1.5'den fazla olması 3.7 kat artırırken trombolitik tedavi AV tam blok gelişimi 3.2 kat azaltmaktadır.

Sonuç olarak, başvurudaki basit elektrokardiyografik ölçümler AV tam blok gelişimi için tarama testi olarak ve yüksek riskli hastaların belirlenmesinde kullanılabilir. Bu hastalar daha kararsız klinik durumlar açısından dikkatle izlenmeli veya profilaktik kalp pili yerleştirilmesi açısından değerlendirilmelidir.

Anahtar kelimeler: Atrioventriküler tam blok, Akut inferior miyokard enfarktüsü, Hastane içi mortalite

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INTRODUCTION

Inferior wall acute myocardial infarction (MI) is often complicated by atrioventricular block (AV) with an incidence varying from 8 to 20 %¹⁻³ during the hospital course. Several investigators report that larger infarctions are associated with AV block, a high incidence of in-hospital complications and an increased short term mortality rate occur among the patients with complete atrioventricular block (CAVB)^{1-3,4}. Therefore, it is important to define the patients with a high-risk for the development of CAVB since the clinician managing such patients must decide whether to place a prophylactic pacemaker. Some electrocardiographic (ECG) markers are shown to be useful in identifying the location

of the totally occluded coronary artery⁵ and in predicting in-hospital mortality in acute inferior wall MI⁶.

This study assessed whether there are baseline clinical factors and electrocardiographic patterns of acute inferior MI that are associated with greater risk of developing CAVB.

MATERIAL and METHODS

Study Sample: Consecutive 787 patients admitted to our clinics with their first Q-wave inferior myocardial infarction were included in our study. Blood samples were obtained on admission and every 3 hours during the first 24 hours, every 6 hours for the next two days and daily until discharge. Peak CK-MB level was estimated for each patient. All the patients were monitored for at least 3 days but the patients with rhythm disorders until discharge. CAVB was defined by standart criteria: complete dissociation of atrial and ventricular rates with the atrial rate greater than ventricular rate. Ventricular arrhythmias were classified as Lown crite-

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Table 1: Comparison of patients with and without complete atrioventricular block

Variables	Patients with CAVB (n=79)	Patients without CAVB (n=119)	p
Age (Years)	64.2±10.9	57.7±11.4	0.001
Male Patients (%)	52 (65)	93 (78)	0.06
Mean Blood Pressure on Admission (mmHg)	65±15	89±13	0.001
DM n (%)	27 (34)	35 (29)	0.5
HT n (%)	43 (54)	37 (31)	0.001
HL n (%)	19 (24)	50 (42)	0.01
Smoking (%)	37 (47)	58 (49)	0.8
Previous Angina (%)	42 (53)	56 (47)	0.4
Syncope (%)	40 (51)	2 (2)	0.001
Trombolytic Treatment n (%)	54 (68)	97 (82)	0.02
Temporary Pacemaker n (%)	71 (90)	0	0.001
Permanent Pacemaker (%)	3 (4)	0	0.001
Peak CK-MB levels (IU/ml)	183.9±64.6	142.6±39.3	0.01
EF(%)	44.9±5.8	47.0±6.4	0.02
IRA			
Cx	10 (15)	27 (26)	0.5
RCA	56 (85)	76 (74)	0.09
RCA Stenosis (%)	86.6±20.6	70.6±35.1	0.001
LAD Stenosis >50% n (%)	31 (47)	32 (31)	0.02
3-Vessel Disease	31 (47)	32 (31)	0.02
Proximal RCA lesion (%)	50 (89)	48 (65)	0.01
Well-developed Collaterals (%)	14 (21)	33 (33)	0.1
Ventricular Arrhythmia (%)	23 (30)	10 (9)	0.001
Hospitalization Time (day)	8.2±5.1	6.1±2.2	0.001
In-Hospital Mortality n (%)	21 (26)	6 (5)	0.001

CAVB: Complete Atrioventricular Block, DM: Diabetes Mellitus, HT: Hypertension, HL: Hyperlipidemia, MI: Myocardial Infarction, CK-MB: Myocardial band fraction of Creatine Kinase, EF: Ejection Fraction; IRA: Infarct Related Artery, RCA: Right Coronary Artery, Cx: Circumflex Artery, LAD: Left Anterior Descending Artery.

rias⁷ and an arrhythmia with a grade >3 and ventricular fibrillation were considered significant. The patients with a history of previous MI, percutaneous or surgical revascularization, significant valvular disease and the patients with first-degree, Mobitz type I and II AV block, left anterior and posterior hemiblock, bundle branch block and atrial fibrillation were excluded.

Electrocardiographic Evaluation: All the patients enrolled in this study had an electrocardiogram (ECG) obtained before the development of CAVB and measurements were performed on this ECG. ECGs were also taken at the time of the patients' admission to the hospital and at least at intervals of 6 hours for the first two days at a paper speed of 25 mm/s and an amplification of 10 mm/mV. The ECGs were reviewed blindly by two cardiologist without knowledge of the patient's clinical course and the study design. ST segment elevation was measured 0.08 second after the J point in leads II, III, aVF and aVR, to an accuracy of 0.5 mm. At least 2 consecutive QRS complexes were measured with the PQ

segment used as the isoelectric line and the mean value recorded for each lead. Each patient was analyzed for the presence of ST elevation > 1mm in lead V4R, the ratio of ST elevation in leads III/II, the presence of ST depression in lead aVL > 1mm.

Coronary Angiography: Coronary angiography was performed in 169 patients at the 3-5th day after admission. Coronary artery lesions with > 50% reduction in diameter were considered significant. Angiographically visible collaterals were graded as follows: 0= no collaterals, 1= incomplete slow opacification of the distal vessel, 2= slow but complete opacification of the distal vessel and 3= distal vessel opacified as the normal vessel⁸. Good collateral filling was classified as grade 2 or 3 and poor collateral filling as grade 0 or 1. The proximal right coronary artery lesion was defined as a lesion before the acute marginal branch. Angiograms were analysed by 2 experienced angiographers without the knowledge of clinical and electrocardiographic findings. Culprit lesion or infarct-related artery (IRA) was defined when the lesion was totally occluded or showed

Table 2: Comparison of electrocardiographic findings of the patients with and without complete atrioventricular block on admission

Electrocardiographic Variables	Patients with CAVB (n=79)	Patients without CAVB (n=119)	p
Atrial Rate on Admission (beats/min)	70±12	71±15	0.6
Ventricular Rate on Admission (beats/min)	45±12	71±15	0.001
ST elevation in lead II	2.5±1.2	2.2±3.2	0.3
ST elevation in lead III	4.0±1.7	2.4±1.3	0.001
ST elevation in aVF	3.2±1.5	1.9±1.1	0.001
ST elevation D3/D2	1.7±0.4	1.4±0.5	0.001
ST elevation aVF/D2	1.3±0.3	1.1±0.4	0.001
>1 mm ST depression in aVL (%)	63 (%80)	78 (%65)	0.03
>1mm ST elevation in RV4 (%)	48 (%61)	21 (%18)	0.001

severe stenoses.

Echocardiography: Two-dimensional and M-mode transthoracic echocardiography were performed at the 3rd day after admission and left ventricular systolic functions were evaluated. Left ventricular ejection fraction was estimated from apical four-chamber view using Simpson method⁹.

Statistical Analysis: Continuous variables are presented as mean±SD and categorical variables are expressed as frequencies and percentages. For continuous variables, differences between patients with and without CAVB were tested using Student's t-test, and for categorical variables Chi-square test (or Fisher's exact test) was used. A receiver operating characteristic curve was used to evaluate the various sensitivities and specificities at different cutpoints of some electrocardiographic and clinical variables. Multivariate logistic regression techniques were used to develop a model to predict CAVB and in-hospital mortality. A p value <0.05 was considered statistically significant.

RESULTS

Seventy-nine consecutive patients (52 male, 27 female with an average age of 64.2±10.9 years) with CAVB and 119 randomized patients (93 male, 16 female with an average age of 57.7±11.4 years) without CAVB were enrolled in this study. Sixty-six (83.5%) of the patients with CAVB and 100 (84%) of the control group underwent coronary angiography. There were no significant differences between the 2 groups as to sex, DM, smoking, previous angina, infarct-related arteries (IRA). The patients with CAVB were older. The mean blood pressure on admission, hyperlipidemia, thrombolytic treatment and ejection fraction (EF) were lower, hypertension (HT), syncope, pacemaker implantation, peak CK-MB levels, 3-vessel disease, left anterior descending artery (LAD) stenosis >50%, right coronary artery (RCA) stenosis, proximal RCA lesion, hospitalization time,

ventricular arrhythmias, in-hospital mortality were higher in the patients with CAVB compared to those without atrioventricular block (Table 1). ST segment elevation in leads III and aVF, the ratio of ST elevation in III:II and aVF:II, ST segment elevation >1mm in RV4 and ST depression >1mm in aVL were significantly higher in the patients with CAVB on admission (Table 2). Temporary endocardial ventricular pacemakers were installed in 71 patients (90 %) but permanent pacemaker implantation were performed only in 3 patients (4%).

Of the 79 patients, 68 (86%) developed CAVB during the first 24 hours after the onset of symptoms and they constituted the early block group. Eleven patients (13.9%) developed CAVB after 24 hours and they compromised the late block group. There were no significant difference in age, sex, cardiac risk factors, presence of previous angina history, ST-segment elevation in leads II, III, aVF, RV4, ST depression in aVL, thrombolytic treatment, peak CK-MB levels, EF, the need for pacemaker implantation, 3-vessel disease, RCA stenosis, infarct related artery, collateral development, in-hospital mortality. Only the ratio of ST elevation in lead III:II was greater (1.8±0.5 vs 1.5±0.4, p=0.002) and proximal RCA lesion was higher (92 % vs 75 %, p=0.002) in the early block compared to the late block group.

The patients who died during the hospitalization were older (71.6±8.3 year vs 58.5±11.1 year, p=0.001), higher ST elevation in lead III (3.9±1.9 vs 2.9±1.6, p=0.009), higher CK-MB levels (233.1±58.1 vs 138.1±36.9, p=0.001), lower left ventricle EF (39.7±4.6 vs 47.2±5.8, p=0.001) than those who survived. Those patients had also higher CAVB ratio (74% vs 34%, p=0.001), right ventricle involvement (81% vs 28%, p=0.001), 3-vessel disease (77% vs 34%, p=0.006), higher ventricular arrhythmia rate (56% vs 7%, p=0.001). ST-segment elevation in lead III >3.25 mm in the admission ECG separated the

Table 3: Factors affecting mortality in the patients with acute inferior wall myocardial infarction

Variables	β	S.E	R.R	P
Age >65	2.1	0.5	8.3	0.001
CAVB	3.0	0.5	6.8	0.001
ST elevation in lead III >3.25	1.3	0.4	3.8	0.002
ST elevation D3/D2 >1	1.1	0.6	3.1	0.001
>1mm ST elevation in RV4	1.3	0.8	3.5	0.001
Peak CK-MB>166	4.8	1.0	3.2	0.001
EF<35%	3.1	0.9	24.2	0.001
RCA Stenosis >95%	1.2	0.5	3.6	0.003
LAD Stenosis >50%	1.7	0.7	3.8	0.002
Thrombolytic treatment	-0.8	0.6	-3.3	0.001
Ventricular Rate on Admission <36 beats/min	1.0	0.6	2.8	0.04

Table 4: The factors affecting the development of complete atrioventricular block in the patients with acute inferior wall myocardial infarction

Variables	β	S.E	R.R	P
Age>65	0.8	0.4	2.2	0.03
HT	0.4	0.5	1.5	0.4
HL	-0.9	0.2	-2.5	0.08
Trombolytic Treatment	-0.9	0.6	-3.2	0.02
>1 mm ST depression in aVL	-0.6	0.6	-2.0	0.3
>1mm ST elevation in RV4	1.3	0.5	3.7	0.01
ST elevation in lead III>2.75 mm	2.5	0.4	12.3	0.001
ST elevation D3/D2>1.5	1.3	0.4	3.7	0.001
ST elevation in aVF >2.75 mm	1.6	0.3	5.1	0.001
ST elevation aVF/D2>1.0	0.7	0.3	2.0	0.04
Proximal RCA lesion	1.9	0.8	2.9	0.001
LAD Stenosis >50%	1.1	0.6	1.6	0.02

patients who died during hospitalization from those who saved by a sensitivity of 63%, specificity of 70% and a positive predictive accuracy of 66%. ST-segment elevation in lead III >3.25 mm increased the mortality by 3.8 folds, a ratio of ST-segment elevation lead III:II>1 by 3.1 folds, ST segment elevation >1mm in RV4 by 3.5 folds, peak CK-MB>166 by 3.2 folds, EF<35% by 24.2 folds, RCA stenosis>95% by 3.6 folds, LAD stenosis>50% by 3.8 folds, age>65 years by 8.3 folds, ventricular rate<36 beats/min on admission by 2.8 folds, CAVB by 6.8 folds on contrary thrombolytic treatment decreased the mortality by 3.3 folds (Table 3).

A ratio of ST-segment elevation in lead III:II >1.5 separated the patients with CAVB from control subjects with a sensitivity of 70%, specificity of 68% and a positive predictive accuracy of 70%. Regression analysis revealed that an age>65 years increased the development of CAVB during acute inferior MI by 2.2 folds, >1mm ST elevation in RV4 by 3.7 folds, ST

elevation >2.75 mm in lead III by 12.3 folds, ST elevation >2.75 mm in aVF by 5.1 folds, ratio of elevation in lead III:II>1.5 by 3.7 folds and ratio of elevation in aVF:II>1.0 by 2 folds but the thrombolytic therapy decreased the development of CAVB by 3.2 folds (Table 4). Intra and interobserver variability for all electrocardiographic measurements were less than 3%.

The patients with right ventricular (RV) involvement had higher ST elevation in lead III (1.9±0.2 vs 1.3±0.2, p=0.001) and aVF (1.6±0.2 vs 1.1±0.2, p=0.001), higher lead III/II ST elevation ratio (1.7±0.3 vs 1.5±0.2, p=0.002), higher aVF/II ST elevation ratio (1.7±0.4 vs 1.5±0.5, p=0.03), higher CK-MB levels (173.7±68.1 vs 139.1±35.2, p=0.001), lower EF (44.3±5.9 vs 47.2±6.2, p=0.001) than those without RV involvement. Moreover, they also had ventricular arrhythmias (25% vs 8%, p=0.002) and mortality (32% vs 4%, p=0.001). Regression analysis revealed that a ST elevation>2.75 in lead III (β =1.6, RR=3.2,

$p=0.007$) is a significant predictor for RV involvement in the patients with acute inferior MI.

DISCUSSION

The main findings of this study; 1) ST elevation in III>II and a ratio of ST segment elevation in leads III/II >1.5; 2) >1mm ST depression in RV4; 3) ST elevation in lead III>2.75 mm; 4) ST elevation in aVF >2.75 mm; 5) ST elevation aVF/DII>1.0; 6) Proximal RCA lesion; 7) LAD stenosis; 8) Older age (>65 years) are significant predictors for complete atrioventricular block in the patients with acute inferior wall MI.

Complete atrioventricular block is a frequent complication of inferior wall acute MI that is associated with a high incidence of in-hospital morbidity and mortality^{1-4,10,11}. Therefore the clinician managing such patients must decide whether to place a prophylactic pacemaker or observe a patient in a potentially unstable condition. Conduction defects complicated acute MI have a graded impact on short-term prognosis although a decline in the rate of severe conduction defects compared with previous reports possibly reflecting the beneficial effects of thrombolytic therapy^{3,13,14}. But also in the thrombolytic era, AV in the setting of ST elevation MI is common and associated with higher mortality^{15,16}. Although Chen S et al¹⁷ showed that thrombolytic therapy can reduce the incidence of severe AV block, shorten its duration and decrease mortality, Ben Ameer Y et al¹⁸ noted that thrombolysis does not affect the incidence of AV block but improves the outcomes of these patients. Despite the initial successful reperfusion, the patients with acute inferior MI and CAVB have a higher rates of in-hospital complications and mortality². In our study, we found that thrombolytic therapy significantly decreased both the mortality and the development of CAVB. But still, CAVB was shown to increase in-hospital mortality by 6.8 folds independent to the thrombolytic therapy.

A number of studies have assessed the in-hospital significance of AV block in acute inferior MI^{1-4,15-19}. In-hospital mortality rates are varying from 8 to 45%. The hospital mortality in the patients who developed CAVB block was 26% in our study and much higher compared to those without CAVB (5%). The increased risk of early mortality in these patients may be related to several factors: larger infarct, ischemia at a distance, increased electrical instability and more severe right or left ventricular dysfunction²⁰. Previous studies have shown that the patients with CAVB and inferior MI have a large infarct size and increased in-hospital mortality despite thrombolytic treatment¹⁹⁻²¹. In our study, the peak CK-MB levels were higher and

left ventricle EF was lower in the patients with CAVB compared to those without CAVB. In addition, an EF<35 % was found to increase the mortality by 24.2 folds, a peak CK-MB levels>166 increased the mortality by 3.2 folds. However, Kimura et al¹⁴ found that peak CK activity and QRS score at discharge are similar in the patients with and without CAVB. In another study, although early CAVB is shown to be related to a more extensive area at risk, the clinical features are found to depend on the atrial rate during CAVB²². We found that a ventricular rate <36 beats/min at admission increase the mortality by 2.8 folds in acute inferior MI.

Some electrocardiographic risk factors such as first-degree, Mobitz type I and II AV block, bundle branch block, left anterior and posterior hemiblock were defined to predict the occurrence of CAVB¹². Previous 2 reports noted that patients with J-point/R-wave ratio >0.5 in >2 inferior leads (II, III and aVF), female patients and the patients with higher Killip class on admission (>2) have an increased risk for development of high-degree AV block in inferior wall acute MI^{23,24}. In our study, we found that ST elevation in III>II and a ratio of ST segment elevation in leads III/II >1.5, >1mm ST depression in RV4, ST elevation in lead III>2.75 mm, ST elevation in aVF >2.75 mm, ST elevation aVF/DII>1.0 are significant predictors for CAVB in the patients with acute inferior wall MI. As to clinical variables, the patients older than 65 years have higher risk of CAVB development. Similarly, Meine TJ et al¹⁵ reported that significant independent predictors of AV block are inferior MI, older age, worse Killip class at presentation, female sex, current smoking, hypertension, and diabetes. The prevalence of stenosis in LAD was much higher in the patients with CAVB. We found that LAD stenosis is one of the predictors for CAVB development in the patients with inferior wall acute MI. Similar to our results, Bassan R et al²⁵ showed that patients with AV block during acute inferior wall MI has a significantly higher prevalence of LAD obstruction. These findings also support the observations that the proximal AV conduction system usually has a dual arterial blood supply from both the right and left anterior descending coronary arteries. Proximal RCA lesion is found to be associated with a higher risk of high-degree AV block development²⁶. In most of the patients with acute inferior MI, there is a total occlusion of the proximal RCA²¹. Greater ST elevation in lead III than II is a sensitive and specific marker of RCA occlusion²⁷. Moreover, Zimetbaum et al⁵ demonstrated that the presence of ST-segment elevation in lead III>II is a powerful predictor of occlusion of the proximal or

mid portion of the RCA in the patients with acute inferior MI. Accordantly in our study, proximal RCA lesion and a ratio of ST segment elevation in leads III/II >1.5 were independent predictors of CAVB and increased the development of CAVB by 2.9 and 3.7 folds, respectively.

Right ventricular involvement in the patients with acute inferior MI is reported to have a higher rate of major complications and in-hospital mortality²⁸. Similarly, we found that ST segment elevation >1mm in RV4 increased the mortality in the patients with acute inferior MI by 3.5 folds. Moreover, right ventricular involvement identifies high risk developing atrioventricular nodal conduction disturbances in the patients with inferior MI³⁰. In our study, >1mm ST elevation in RV4 increased the development of CAVB by 3.7 folds. Although Turhan et al²⁹ demonstrated that ST-segment depression >1 mm in lead aVL, obtained on admission is useful in identifying RV involvement, ST-segment depression >1 mm in aVL did not increase the risk of mortality and development of CAVB in our study.

Ventricular arrhythmias were much more common among the patients with CAVB than among those without those complication in our study similar to the results of Kaul et al³¹. However, Dubois et al²⁰ found that frequent premature ventricular complexes and ventricular tachycardia were similar in the patients with and without CAVB.

As a result, these simple electrocardiographic measurements at admission can be used as a screening test for development of CAVB. And the clinician managing such patients should either observe more cautiously for a potentially unstable condition or place a prophylactic pacemaker.

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