ORIGINAL ARTICLE

N-terminal pro-brain natriuretic peptide and electrocardiographic variables associated with increased risk of complete atrioventricular block and mortality in patients with acute inferior myocardial infarction

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Abstract

Background: *Although brain natriuretic peptide (BNP) levels are shown to be an important prognostic factor in patients with acute myocardial infarction (MI), the relationship between arrhythmias and BNP levels is not known. This study assessed whether baseline clinical factors, N-terminal-proBNP (NT-proBNP) levels and electrocardiographic patterns of acute inferior MI are associated with greater risk of developing complete atrioventricular block (CAVB) and mortality.*

Methods and Results: *Seventy-nine consecutive patients (52 male, 27 female with an avarage age of 64.2 ± 10.9 years) with CAVB and 119 control patients (93 male, 16 female with an average age of 57.7 ± 11.4 years) without CAVB were enrolled. Regression analysis revealed that NT-proBNP levels > 104 pg/mL increased the development of CAVB by 16.7 folds, > 1 mm ST elevation in RV4 by 2.7 folds, ratio of elevation in lead III:II > 1.5 by 10.1 folds but the thrombolytic therapy decreased the development of CAVB by 2.8 folds. NT-proBNP > 92 pg/mL increased the mortality by 8.9 folds, a ratio of ST-segment elevation in lead III:II > 1 by 3.1 folds, ST segment elevation > 1 mm in RV4 by 3.5 folds, ejection fraction < 35% by 24.2 folds, age > 65 years by 8.3 folds and CAVB by 6.8 folds, on contrary thrombolytic treatment decreased the mortality by 3.3 folds.*

Conclusions: *Simple electrocardiographic measurements and NT-proBNP levels at admission can be used as a screening test for development of complications such as CAVB, right ventricular involvement and mortality during acute inferior wall MI.* (Cardiol J 2012; 19, 5: 479–486)

Key words: complete atrioventricular block, ST-segment elevation inferior myocardial infarction, brain natriuretic peptide

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Introduction

Inferior wall acute myocardial infarction (MI) is often complicated by atrioventricular (AV) block 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 AV block (CAVB) [1–4]. 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 artery [5] and in predicting in-hospital mortality in acute inferior wall MI [6]. Although higher brain natriuretic peptide (BNP) levels are shown to be an important prognostic factor for short- and long-term in patients with acute MI [7–10], the relationship between arrhythmias and BNP levels is not known.

This study assessed whether baseline clinical factors, N-terminal-proBNP (NT-proBNP) levels and ECG patterns of acute inferior MI are associated with greater risk of developing CAVB and mortality.

Methods

Study sample

Consecutive 787 patients admitted to our clinics with their first ST elevation inferior MI were included in our study. A written consent was obtained from all patients and our local ethics committee approved the study [11]. Blood samples were obtained on admission and every 3 h during the first 24 h, every 6 h for the next 2 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 standard criteria: complete dissociation of atrial and ventricular rates with the atrial rate greater than ventricular rate. Ventricular arrhythmias were classifed as Lown criterias [12] 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 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 h for the first 2 days at a paper speed of 25 mm/s and an amplification of 10 mm/mV. The ECGs were reviewed blindly by two cardiologists without knowledge of the patient's clinical course and the study design. ST segment elevation was measured 0.08 s 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 > 1 mm in lead V4R, the ratio of ST elevation in leads III/II, the presence of ST depression in lead aVL > 1 mm.

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 vessel [13]. Good collateral filling was classified as grade 2 or 3 and poor collateral filling as grade 0 or 1. The proximal right coronary artery (RCA) lesion was defined as a lesion before the acute marginal branch. Angiograms were analyzed by 2 experienced angiographers without the knowledge of clinical and ECG findings. Culprit lesion or infarct-related artery (IRA) was defined when the lesion was totally occluded or showed severe stenoses (> 90–95% obstruction).

Echocardiography

Two-dimesional and M-mode transthoracic echocardiography were performed at the 3rd day after admission and left ventricular systolic functions were evaluated. Left ventricular ejection fraction (LVEF) was estimated from apical four-chamber view using Simpson method [14].

Measurement of NT-proBNP plasma levels

Peripheral venous blood samples were collected into EDTA-containing tubes for each subject at rest at admission. The samples were centrifuged within 20 min at +4°C. The plasma was stored at –80°C until analysis. Serum NT-proBNP was mea-

sured by a double antibody sandwich technique using electrochemiluminescence immunoassay kit (Roche Diagnostics). The results were reported as picogram per milliliter [pg/mL]. The clinicians involved in the study were blinded to the NT--proBNP values obtained.

Statistical analysis

Continous variables are presented as mean ± ± standard deviation and discrete variables are expressed as frequencies and percentages. For continous variables, differences between patients with and without CAVB were tested using Student's *t*-test, and for categorical variables χ^2 (or Fischer's exact test) was used. A receiver operating characteristic curve was used to evaluate the various sensitivities and specificities at different cutpoints of some ECG and clinical variables besides NT-proBNP. Multivariate logistic regression techniques were used to developed 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 control 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. The mean blood pressure on admission, hyperlipidemia, thrombolytic treatment and EF were lower, hypertension (HT), syncope, pacemaker implantation, peak CK-MB levels, NT-pro-BNP levels, 3-vessel disease, left anterior descending artery (LAD) stenosis > 50%, RCA stenosis, proximal RCA lesion, hospitalization time, ventricular arrhythmias, in-hospital mortality were higher in the patients with CAVB compared to those without AV 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 > 1 mm in RV4 and ST depression > 1 mm in aVL on admission were significantly higher in the patients with CAVB (Table 2). Temporary endocardial ventricular pacemakers were installed in 71 (90%) patients, but permanent pacemaker implantation was performed only in 3 (4%) patients.

Of the 79 patients, 68 (86%) developed CAVB during the first 24 h after the onset of symptoms and they constituted the early AV block group. Eleven (13.9%) patients developed CAVB after 24 h

and they compromised the late AV 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, IRA, collateral development, in-hospital mortality. The ratio of ST elevation in lead III:II was greater (1.8 ± 0.5 vs. 1.5 ± 0.4, p = 0.002), NT-proBNP levels on admission were higher (133.1 ± 62.9 vs. 128.3 ± ± 32.5, p = 0.001) and proximal RCA lesion was higher (92% vs. 75%, p = 0.002) in the early AV block compared to the late AV block group. The patients with late CAVB had also higher NT-proBNP levels than those without CAVB (128.3 ± 32.5 vs. 86.4 ± 30.1, p = 0.008).

Correlation analysis showed that NT-proBNP levels were correlated with ST-elevations in leads III and aVF. Moreover, NT-proBNP levels were correlated with peak CK-MB levels (r = 0.4, p = = 0.001) and LVEF (r = –0.4, p = 0.001). Other correlations were shown in 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%. An NT-proBNP level > 104 separated the patients with CAVB from control subjects with a sensitivity of 72%, specificity of 79% and a positive predictive accuracy of 70%. Regression analysis revealed that NT-proBNP levels > 104 increased the development of CAVB by 16.7 folds, proximal RCA lesion by 3.9 folds, LAD stenosis > 50% by 1.2 folds, > 1 mm ST elevation in RV4 by 2.7 folds, ratio of elevation in lead III:II > 1.5 by 10.1 folds but the thrombolytic therapy decreased the development of CAVB by 2.8 folds (Table 4).

The patients who died during the hospitalization were older (71.6 ± 8.3 vs. 58.5 ± 11.1, p = = 0.001), had 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 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 seperated the patients who died during the hospitalization from those who survived by a sensitivity of 63%, specificity of 70% and a positive predictive accuracy of 66%. NT-proBNP > 92 pg/mL

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 [mm Hg]	65 ± 15	89 ± 13	0.001
Diabetes mellitus	27 (34%)	35 (29%)	0.5
Hypertension	43 (54%)	37 (31%)	0.001
Hyperlipidemia	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
Thrombolytic treatment	54 (68%)	97 (82%)	0.02
Temporary pacemaker	71 (90%)	0	0.001
Permanent pacemake	3 (4%)	0	0.001
Peak CK-MB levels [IU/mL]	183.9 ± 64.6	142.6 ± 39.3	0.01
Pro-BNP [pg/mL]	131.7 ± 55.7	86.4 ± 30.1	0.001
Ejection fraction (%)	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%	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	21 (26%)	6 (5%)	0.001

CAVB — complete atrioventricular block; CK-MB — myocardial band fraction of creatine kinase; BNP — brain natruretic peptide; IRA — infarct related artery; RCA — right coronary artery; CX — circumflex artery; LAD — left anterior descending artery

Table 2. Comparison of electrocardiographic findings of the patients with and without complete atrioventricular block (CAVB) on admission.

Electrocardiographic variables	Patients with CAVB (n = 79)	Patients without CAVB (n = 119)	P
Atrial rate on admission [bpm]	70 ± 12	71 ± 15	0.6
Ventricular rate on admission [bpm]	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
> 1 mm ST elevation in RV4 (%)	48 (61%)	21 (18%)	0.001

increased the mortality 8.9 folds (sensitivity 88%, specificity 62%). A ratio of ST-segment elevation in leads III:II > 1 by 3.1 folds, ST segment elevation > 1 mm in RV4 by 3.5 folds, peak CK-MB > 166 IU/mL by 3.2 folds, EF < 35% by 24.2 folds, RCA stenosis > 95% by 3.6 folds, LAD stenosis

Table 3. Correlations between electrocardiographic parameters and laboratory.

Variables	ΣST in II	ΣST in III	ΣST in aVF	ΣST III/II	Basal HR
NT-proBNP	r = 0.03 p = 0.7	**p = 0.3** **p = 0.001**	**p = 0.3** **p = 0.001**	p = 0.1 p = 0.09	r = 0.2 p = 0.3
LVEF	**r = –0.2** **p = 0.01**	**p = –0.5** **p = 0.001**	**p = –0.5** **p = 0.001**	p = –0.02 p = 0.8	r = 0.01 p = 0.9
CK-MB	**r = 0.3** **p = 0.001**	**p = 0.5** **p = 0.002**	**p = 0.5** **p = 0.002**	p = 0.03 p = 0.7	r = 0.2 p = 0.3
Stenosis degree in RCA	r = 0.03 p = 0.7	p = 0.02 p = 0.08	p = 0.01 p = 0.9	p = 0.1 p = 0.2	r = 0.2 p = 0.3

NT-proBNP — N-terminal-pro-brain natriuretic peptide; LVEF — left ventricular ejection fraction; CK-MB — myocardial band fraction of creatine kinase; RCA — right coronary artery; HR — heart rate

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

Variables	β	SE	RR	P
Age > 65	0.02	0.04	1.1	0.6
Hypertension	0.8	0.9	1.5	0.5
Hyperlipidemia	–0.5	0.8	–2.7	0.6
Thrombolytic treatment	–0.8	0.9	–2.8	0.02
> 1 mm ST depression in aVL	1.2	1.2	2.1	0.3
> 1 mm ST elevation in RV4	1.1	0.8	2.7	0.02
ST elevation in lead III > 2.75 mm	2.1	0.6	2.3	0.01
ST elevation D3/D2 > 1.5	4.6	1.7	10.1	0.007
Proximal RCA lesion	1.4	0.6	3.9	0.02
LAD stenosis > 50%	1.0	0.6	1.2	0.03
Collaterals to IRA	–1.1	0.9	3.3	0.3
NT-proBNP > 104	2.7	1.1	16.7	0.006

LAD — left anterior descending artery; IRA — infarct related artery; NT-proBNP — N-terminal-pro-brain natriuretic peptide

Table 5. Factors affecting mortality in the patients with acute inferior wall myocardial infarction.

Variables	β	SE	RR	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
> 1 mm ST elevation in RV4	1.3	0.8	3.5	0.001
Peak CK-MB > 166	4.8	1.0	3.2	0.001
Ejection fraction < 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 bpm	1.0	0.6	2.8	0.04
NT-proBNP > 92 pg/mL	2.2	0.8	8.9	0.005

CAVB — complete atrioventricular block; CK-MB — myocardial band fraction of creatine kinase; RCA — right coronary artery; LAD — left anterior descending artery; NT-proBNP — N-terminal-pro-brain natriuretic peptide

> 50% by 3.8 folds, age > 65 years by 8.3 folds, ventricular rate < 36 bpm on admission by 2.8 folds, CAVB by 6.8 folds on contrary thrombolytic treatment decreased the mortality by 3.3 folds (Table 5).

Discussion

The main findings of this study: 1) A ratio of ST segment elevation in leads III/II; 2) > 1 mm ST elevation in RV4; 3) ST elevation in lead III; 4) Proximal RCA lesion; 5) LAD stenosis; 6) Older age; 7) NT-proBNP are significant predictors for CAVB and mortality in the patients with acute inferior wall MI.

CAVB is a frequent complication of inferior MI that is associated with a high incidence of in-hos-

pital morbidity and mortality [1–4, 15, 16]. Conduction defects complicated acute MI have a graded impact on short-term prognosis [3, 17–21]. Although Chen et al. [22] showed that thrombolytic therapy can reduce the incidence of severe AV block, shorten its duration and decrease mortality, Ben Ameur et al. [23] 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 rate of in-hospital complications and mortality [2]. In our study, 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, 20–24]. In-hospital mortality rates are varying from 8 to 45% [1–4, 20–24]. The hospital mortality in the patients who developed CAVB 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 dysfunction [25]. Previous studies have shown that the patients with CAVB and inferior MI have a large infarct size and increased in-hospital mortality despite thrombolytic treatment [24–26]. In our study, the peak CK-MB levels were higher and LVEF 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 and a peak CK-MB levels > 166 IU/mL by 3.2 folds. However, Kimura et al. [15] found that peak CK activity and QRS score at discharge are similar in the patients with and without CAVB. An NT-proBNP level > 92 pg/mL increased the mortality by 8.9 folds in our study. This may be explained by two proposals. First, high level of BNP is a powerful marker of LV systolic dysfunction and poor prognosis after MI [7–10]. In patients with acute MI, increases in plasma BNP concentration during the early phase reflect MI size, and thereby may predict later LV function [7–10, 27]. Accordingly, NT-proBNP levels were found to be correlated with LVEF and peak CK-MB levels in our study. Secondly, higher BNP level may reflect right ventricular involvement in patients with inferior MI [28]. Similarly, we found that the patients (n = 74) with ST elevation > 1 mm in RV4 had higher NT-proBNP levels (138.5 ± 45.9 pg/mL vs. 79.3 ± 22.9, p = 0.001) compared to those without. NT-proBNP level > 95 pg/mL separated the patients with right ventricular involvement by a sensitivity of 98% and a specificity of 77%. NT-proBNP level > 95 pg/mL increased the risk of right ventricular involvement by 12.9 folds (β = 4.9, p = 0.001). Right ventricular involvement in the patients with acute inferior MI is reported to have a higher rate of major complications and in-hospital mortality [29]. Similarly, we found that ST segment elevation > 1 mm in RV4 increased the mortality in the patients with acute inferior MI by 3.5 folds. Moreover, right ventricular involvement identifies high risk developing AV nodal conduction disturbances in the patients with inferior MI [30]. In our study, > 1 mm ST elevation in RV4 increased the development of CAVB by 3.7 folds. 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 CAVB [31, 32]. We found that a ventricular rate < 36 bpm 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 CAVB [16]. 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 [33, 34]. In our study, we found that ST elevation in lead III greater than in II and a ratio of ST segment elevation in leads III/II > 1.5, > 1 mm ST elevation 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 et al. [20] 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 et al. [35] 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 development [36]. In most of the patients with acute inferior MI, there is a total occlusion of the proximal RCA [26]. Greater ST elevation in lead III than II is a sensitive and specific marker of RCA occlusion [37]. Moreover, Zimetbaum et al. [5] 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. Accordingly 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.

Although higher BNP levels are shown to be an important prognostic factor for short- and long--term in patients with acute MI [7–10], the relationship between arrhythmias and NT-proBNP levels is not known. Higher BNP levels are useful for identification of hypertrophic cardiomyopathy patients [38], heart failure patients [39] who have a risk of atrial fibrillation. The plasma levels of NT-proBNP in patients with bradyarrhythmia increased in proportion to aggravation of AV asynchrony [40]. Koch et al. [41] found that high degree AV block can induce elevated plasma BNP levels and the loss of AV synchrony induce a further increase of BNP. Therefore, the increase in NT-proBNP in patients with CAVB during acute inferior MI is not surprising. But the patients with late CAVB had also higher NT-proBNP levels at admission than control group suggesting that NT-proBNP levels may be useful for identification of the patients with acute inferior MI who have a high risk for high degree AV block.

Limitations of the study

Patients with their first ST elevation inferior MI who are managed with medical therapy/thrombolytics in the acute setting were evaluated. Observations cannot be generalized to patients with previous MI or those whom performed a primary percutaneous coronary intervention.

Conclusions

As a result, these simple electrocardiographic measurements and NT-proBNP levels at admission can be used as a screening test for development of complications such as CAVB, right ventricular involvement and mortality during acute inferior wall myocardial infarction. And the clinician managing such patients should either observe more cautiously for a potentially unstable condition or should apply much more invasive procedures such as percutaneous coronary interventions or a prophylactic pacemaker implantation.

Conflict of interest: none declared

References

1. Tans AC, Lie KI, Durrer D. Clinical setting and prognostic significance of high degree atrioventricular block in acute inferior myocardial infarction: A study of 144 patients. Am Heart J, 1980; 99: 4–8.
2. Clemmensen P, Bates ER, Califf RM et al. Complete atrioventricular block complicating inferior wall acute myocardial infarction treated with reperfusion therapy. Am J Cardiol, 1991; 67: 225–230.
3. Archbold RA, Sayer JW, Ray S et al. Frequency and prognostic implications of conduction defects in acute myocardial infarction since the introduction of thrombolytic therapy. Eur Heart J, 1998; 19: 893–898.
4. Rotman M, Wagner GS, Waugh RA. Significance of high degree atrioventricular block in acute posterior myocardial infarction. The importance of clinical setting and mechanism of block. Circulation, 1973; 47: 257–262.
5. Zimetbaum PJ, Krishnan S, Gold A et al. Usefulnes of ST-segment elevation in lead III exceeding that of lead II for identifying the location of the totally occluded coronary artery in inferior wall myocardial infarction. Am J Cardiol, 1998; 81: 918–920.
6. Saw J, Davies C, Fung A et al. Value of ST elevation in lead III greater than II in inferior wall acute myocardial infarction for predicting in-hospital mortality and diagnosing right ventricular infarction. Am J Cardiol, 2001; 87: 448–450.
7. Omland T, Persson A, Ng L et al. N-terminal pro-B-type natriuretic peptide and long-term mortality in acute coronary syndromes. Circulation, 2002; 106: 2913–2918.
8. Sabatine MS, Morrow DA, de Lemos JA et al. Multimarker approach to risk stratification in non-ST elevation acute coronary syndromes: Simultaneous assessment of troponin I, C-reactive protein, and B-type natriuretic peptide. Circulation, 2002; 105: 1760–1763.
9. Yamashita T, Seino Y, Ogawa A et al. N-terminal pro-BNP is a novel biomarker for integrated cardio-renal burden and early risk stratification in patients admitted for cardiac emergency. J Cardiol, 2010; 55: 377–383.
10. Güneæ Y, Okçün B, Kavlak E et al. Value of brain natriuretic peptide after acute myocardial infarction. Anadolu Kardiyol Derg, 2008; 8: 182–187.
11. Coats AJ. Ethical authorship and publishing. Int J Cardiol, 2009; 131: 149–150.
12. Lown B, Graboys TB. Management of patients with malignant ventricular arrhythmias. Am J Cardiol, 1977; 39: 910–918.
13. Rentrop KP, Cohen M, Blanke H et al. Changes in collateral channel filling immediately after controlled coronary artery occlusion by an balloon angioplasty in humanbeings. J Am Coll Cardiol, 1982; 5: 587– 592.
14. Schiller NB, Shah PM, Crawford M et al. Recommendations for quantitation of the left ventricle by two-dimensional echocardiography. American Society of Echocardiography. Committee on standards, subcommittee on quantitation of two-dimensional echocardiograms. J Am Soc Echocardiography, 1989; 2: 358–367.
15. Berger PB, Ruocco NA, Ryan TJ et al. Incidence and prognostic implications of heart block complicating inferior myocardial infarction treated with thrombolytic therapy: Results of TIMI II. J Am Coll Cardiol, 1992; 20: 533–540.
16. Goldberg PJ, Zevallos JC, Yazebski J et al. Prognostic of acute myocardial infarction complicated by complete heart block (the Worcester Heart Attack Study). Am J Cardiol, 1992; 69: 1135–1141.
17. Lamas GA, Muller JE, Turi ZG et al. A simplified method to predict occurence of complete heart block during acute myocardial infarction. Am J Cardiol, 1986; 57: 1213–1219.
18. McNeill AJ, Roberts MJ, Purvis JA. Thrombolytic therapy administered to patients with complete heart block complicating acute myocardial infarction. Coron Artery Dis, 1992; 3: 223––229.
19. Kimura K, Kosuge M, Ishikawa T et al. Comparison of early reperfusion in patients with inferior wall acute myocardial infarction with and without complete atrioventricular block. Am J Cardiol, 1999; 84: 731–733.

20. Meine TJ, Al-Khatib SM, Alexander JH et al. Incidence, predictors, and outcomes of high-degree atrioventricular block complicating acute myocardial infarction treated with thrombolytic therapy. Am Heart J, 2005; 149: 670–674.

21. Ruiz-Bailén M, de Hoyos EA, Issa-Khozouz Z et al; ARIAM Group. Clinical implications of acute myocardial infarction complicated by high grade atrioventricular block. Med Sci Monit, 2002; 8: 138–147.

22. Chen S, Feng S, Zhang Q. Atrioventricular block complicating inferior acute myocardial infarction treated with thrombolytic therapy. Chin Med J (Engl), 2001; 114: 1039–1042.

23. Ben Ameur Y, Mghaieth F, Ouchallal K et al. Prognostic significance of second and third degree atrioventricular block in acute inferior wall infarction. Ann Cardiol Angeiol, 2003; 52: 30–33.

24. Sclarovsky S, Strasberg B, Hirshberg A et al. Advanced early and late atrioventricular block in acute inferior wall myocardial infarction. Am Heart J, 1984; 108: 19–24.

25. Dubois C, Pierard LA, Smeets JP et al. Long-term prognostic significance of atrioventricular block in inferior acute myocardial infarction. Eur Heart J, 1989; 10: 816–820.

26. Berger PB, Ryan TJ. Inferior myocardial infarction: High-risk groups. Circulation, 1990; 81: 401–411.

27. Arakawa N, Nakamura M, Aoki H et al. Relationship between plasma level of brain natriuretic peptide and myocardial infarct size. Cardiology, 1994; 85: 334–340.

28. Kaya MG, Ozdogru I, Kalay N et al. Plasma B-type natriuretic peptide in diagnosing inferior myocardial infarction with right ventricular involvement. Coron Artery Dis, 2008; 19: 609–613.

29. Zehender M, Kasper W, Kauder E et al. Right ventricular infarction as an independent predictor of prognosis after acute inferior myocardial infarction. N Engl J Med, 1993; 328: 981–988.

30. Braat SH, Zwaan C, Brugada P et al. Right ventricular involvement with acute inferior myocardial infarction identifies high risk developing atrioventricular nodal conduction disturbances. Am Heart J, 1994; 107: 1183–1187.

31. Kosuge M, Ishikawa T, Morita S et al. Posterior wall involvement attenuates predictive value of ST-segment elevation in lead V4R for right ventricular involvement in inferior acute myocardial infarction. J Cardiol, 2009; 54: 386–393.

32. Kosuge M, Kimura K, Ishikawa T et al. Clinial features of patients with reperfused inferior wall acute myocardial infarction complicated by early complete atrioventricular block. Am J Cardiol, 2001; 88: 1187–1191.

33. Birnbaum Y, Sclarovsky S, Herz I et al. Admission clinical and electrocardiographic characteristics predicting in-hospital development of high-degree atrioventricular block in inferior wall acute myocardial infarction. Am J Cardiol, 1997; 80: 1134––1138.

34. Solodky A, Assali A, Herz I et al. Early development of high--degree atrioventricular block in inferior acute myocardial infarction is predicted by a J-point/R-wave ratio above 0.5 on admission. Cardiology, 1998; 90: 274–279.

35. Bassan R, Maia IG, Bozza A et al. Atrioventricular block in acute inferior wall myocardial infarction: harbinger of associated obstruction of the left anterior descending coronary artery. J Am Coll Cardiol, 1986; 8: 773–778.

36. Tsuka Y, Sugiura T, Hatada K et al. Clinical significance of ST--segment elevation in lead V1 in patients with acute inferior wall Q-wave myocardial infartction. Am Heart J, 2001; 141: 615–620.

37. Chia BL, Yip JWL, Tan HC et al. Usefulness of ST elevation II>III ratio and ST deviation in lead I for identifying the culprit artery in inferior wall acute myocardial infarction. Am J Cardiol, 2000; 86: 341–343.

38. Matsura H, Murakami T, Hina K et al. Association of elevated plasma B-type natriuretic peptide levels with paroxysmal atrial fibrillation in patients with nonobstructive hypertrophic cardiomyopathy. Clin Biochem, 2008; 41: 134–139.

39. Mabuchi N, Tsutamoto T, Maeda K et al. Plasma cardiac natriuretic peptides as biochemical markers of recurrence of atrial fibrillation in patients with mild congestive heart failure. Jpn Circ J, 2000; 64: 765–771.

40. Pan W, Su Y, Hu K et al. Effect of bradyarrhythmia on the plasma levels of N-terminal pro-brain natriuretic peptide. Int J Cardiol, 2009; 136: 105–107.

41. Koch A, Zink S, Dittrich S. Plasma levels of B-type natriuretic peptide in children and adolescents with high degree atrioventricular block. Int J Cardiol, 2009; 134: 429–430.