Brain Natriuretic Peptide Levels in Acute Inferior Myocardial Infarction

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Abstract

Background: Our objective was to evaluate the relationship between initial serum brain natriuretic peptide (BNP) levels and right ventricular functions in inferior myocardial infarction (MI) with and without right ventricular involvement.

Methods: The study included 61 patients, who presented with acute inferior MI. Twenty-seven patients had right ventricular involvement. Blood samples for BNP were obtained from each patient on admission. Echocardiographic assessments were performed and recorded during the first 12 h. Right ventricular involvement was determined by electrocardiography, conventional and tissue Doppler echocardiography (TDI).

Results: In inferior MI with right ventricular involvement, tricuspid annulus planimetric systolic excursion (TAPSE) and right ventricular fractional area change were lower, and left ventricular E/E' ratio was higher. In the group with BNP levels above 400 pg/mL, left ventricular end-diastolic diameter and left ventricular end-systolic diameter were higher, and left ventricular ejection fraction and TAPSE, indicator of right ventricular systolic function, were lower. The elevated BNP levels were negatively correlated with RSm and TAPSE, while they were positively correlated with the E/E' ratio. The systolic blood pressure and left ventricular end-diastolic diameter during admission were independent predictors of BNP levels.

Conclusions: In acute inferior MI, initially increased BNP levels may be valuable in predicting the right ventricle involvement. Higher rates of hypotension, right ventricular dysfunction and increased left ventricle diameters are observed in patients with BNP levels ≥ 400 pg/mL.

Keywords: BNP; Doppler echocardiography; Inferior myocardial infarction; Right ventricle

Introduction

Both systolic and diastolic left ventricular functions are impaired following acute myocardial infarction (AMI). The extent of impairment in left ventricular function is one of the most important factors of morbidity and mortality [1]. In cases of myocardial infarction (MI) associated with left ventricular dysfunction, the right ventricular dysfunction is an independent predictor of mortality [2]. Approximately 50% of acute inferior MIs are accompanied by right ventricular myocardial infarction (RVMI) [3]. RVMI is associated with suppression of the right ventricular functions, resulting in right heart failure and decreased heart rate. Although elevated jugular venous pressure, clear lung fields and hypotension are specific to RVMI, their sensitivity is less than 25% [4]. ST segment elevation in the right precordial derivation V4 (V4R) indicates RVMI, but it has a disadvantage of being temporary [5]. The brain natriuretic peptide (BNP) is synthesized from the ventricular myocardium as a result of increased cardiac wall stress due to pressure overload or volume expansion. BNP can decrease right atrial pressure, systemic vascular resistance, stimulation of sympathetic nerves, aldosterone secretion and cell hypertrophy while increasing the excretion of sodium [6]. Elevated BNP levels were found to be poor outcome in heart failure, and in patients who had a first AMI and underwent primary percutaneous transluminal coronary angioplasty (PTCA), elevated BNP levels was found to be related with poor outcome [7]. In the present study, our objective was to evaluate the relationship of right ventricular dysfunction as determined by conventional and tissue Doppler echocardiography (TDI) with the serum BNP levels in inferior MI with and without right ventricular involvement.

Materials and Methods

This prospective study was developed and adapted in accordance with the guidelines and ethical principles defined in the Declaration of Helsinki and all patients approved and consented to participate in the study. We enrolled 61 patients who had a first acute inferior MI with/without right ventricular involvement with a normal sinus rhythm and a heart rate...
Serum troponin I (TnI) levels were detected by fluorometry using a Siemens troponin kit (cTnI direct fluorometric, Siemens, New York, USA). The cutoff BNP value was 100 pg/mL.

BMI: body mass index; DBP: diastolic blood pressure; DM: diabetes mellitus; GFR: glomerular filtration rate; HR: heart rate; HT: hypertension; OTP: onset time of chest pain; SBP: systolic blood pressure. *Independent groups t-test, †Mann-Whitney U test, ‡Significant at p < 0.05. Data are shown in mean ± standard deviation, median or number (%).

The patients underwent echocardiographic evaluation during the first 12 h after admission. After ECG monitoring, an echocardiographic evaluation was performed from parasternal long axis, parasternal short axis, apical four chamber, and subcostal views using an echocardiographic unit (GE Vingmed Ultrasound, Horten, Norway) and 2.5-3.5 MHz transducers in the left lateral decubitus position. The patients underwent M-mode, two-dimensional echocardiography, pulse wave (PW) and continuous wave (CW) Doppler and colored tissue Doppler echocardiographic evaluation. All Doppler measurements were recorded at the end of the expirium while patients were holding their breath. An average of three consecutive measurements were calculated. An echocardiographic evaluation was performed using the parasternal long axis, short axis, and apical four- and two-chamber images to analyze the left ventricular functions. Following the evaluation of aortic and left ventricular wall thickness, the left ventricular ejection fraction (LVEF) was calculated by the Teicholz method. Wall motion was evaluated from the parasternal short axis view. Biplane ejection fraction was determined from the apical four-chamber and two-chamber views using the Simpson method. Sample volume was placed on the ventricular surface of the mitral valve to evaluate diastolic functions. Right ventricle fractional area change (RVFAC), tricuspid annular plane systolic excursion (TAPSE), and right ventricular systolic wave (RSm) were used to evaluate right ventricular systolic functions, and right ventricle myocardial performance index (RVMPI), tissue Doppler imaging for global functions. We compared the groups with and without right ventricular involvement. After this as-

Table 1. Baseline Characteristics of Patients

| variables | Inferior MI + RVMI (n = 27) | Isolated inferior MI (n = 34) | P  
|-----------|-----------------------------|-----------------------------|-----
| age, years | 59.48 ± 11.46 | 61.06 ± 11.83 | 0.60
| sex, M/F  | 9/18 | 10/24 | 0.74
| BMI, kg/m² | 27.47 ± 4.28 | 25.57 ± 4.28 | 0.86
| DM, n, % | 6, 22.2 | 3, 8.8 | 0.17
| HT, n, % | 11, 40.7 | 16, 47.1 | 0.62
| smoking, n, % | 15, 55.6 | 17, 50 | 0.67
| familial history, n, % | 11, 40.7 | 19, 55.9 | 0.24
| SBP, mm Hg | 134.12 ± 38.25 | 129.28 ± 20.10 | 0.99
| DBP, mm Hg | 76.25 ± 29.28 | 80.75 ± 11.17 | 0.88
| HR, /min | 74.37 ± 12.33 | 71.72 ± 12.92 | 0.36
| OTP, h | 4.26 ± 3.03 | 6.59 ± 5.11 | 0.04
| GFR (mL/min) | 107.10 ± 38.95 | 92.33 ± 37.21 | 0.50
| Gensini score | 32.87 ± 28.59 | 30.77 ± 27.24 | 0.91
Tables 2. Left Ventricular Echocardiographic Parameters

| Variables       | Inferior MI + RVMI (n = 27) | Isolated inferior MI (n = 34) | P    |
|-----------------|-----------------------------|------------------------------|------|
| LA, mm          | 38.92 ± 3.99                | 40.31 ± 4.28                 | 0.46‡|
| LVEDD, mm       | 50.50 ± 4.54                | 50.59 ± 3.56                 | 0.81#|
|                 | 50.50                       | 51.00                        |      |
| LVESD, mm       | 35.12 ± 4.70                | 34.78 ± 3.82                 | 0.43‡|
| LVEF, %         | 57.62 ± 7.05                | 57.50 ± 6.71                 | 0.38‡|
| Mitral E wave, cm/s | 0.80 ± 0.20                  | 0.73 ± 0.24                  | 0.47‡|
| Mitral A wave, cm/s | 0.75 ± 0.24                  | 0.77 ± 0.17                  | 0.66‡|
| DT, ms          | 218.71 ± 60.64              | 217.00 ± 62.63               | 0.65#|
|                 | 205.50                      | 217.00                       |      |
| E/A ratio       | 1.26 ± 0.79                 | 1.02 ± 0.44                  | 0.33#|
|                 | 1.00                        | 0.86                         |      |
| E/E’ ratio      | 12.55 ± 6.2                 | 9.68 ± 3.14                  | 0.05‡|
| Lateral Sm, cm/s| 7.71 ± 1.83                 | 7.28 ± 1.99                  | 0.77#|
|                 | 7.50                        | 7.50                         |      |
| Lateral IVCT, ms | 66.71 ± 16.60              | 66.12 ± 17.44                | 0.54‡|
| Lateral IVRT, ms | 77.79 ± 16.13              | 74.06 ± 16.04                | 0.54‡|
| Lateral ET, ms  | 264.87 ± 19.52             | 262.59 ± 40.03               | 0.99#|
|                 | 265.50                      | 264.00                       |      |
| DT: deceleration time; E/A ratio: ratio of early to late diastolic filling; E/E’: ratio of early filling velocity to tissue Doppler early filling velocity; ET: ejection time; IVCT: isovolumetric contraction time; IVRT: isovolumetric relaxation time; LA: left atrium; LVEDD: left ventricular end-diastolic diameter; LVEF: left ventricular ejection fraction; LVESD: left ventricular end-systolic diameter; RVMI: right ventricular myocardial infarction. ‡Independent groups t-test. #Mann-Whitney U test. Data are shown in mean ± standard deviation, and median.

Results

The study included a total of 61 patients. Of these patients, 27 (44%) had acute inferior MI with right ventricular involvement (mean age, 59.48 ± 11.46 years) and 34 (56%) had acute inferior MI (mean age, 61.06 ± 11.83 years), and 42 (69%) were males, and 19 (31%) were females. As shown in Table 1, no significant difference was found between two groups in baseline characteristics except for onset time of chest pain.

Echocardiographic parameters

Table 2 shows left ventricular echocardiographic parameters. There was no significant difference except E/E’ ratio, which is an indicator of diastolic dysfunction. Right ventricular echocardiographic parameters are shown in Table 3. RVFAC and TAPSE were significantly lower in patients with inferior MI with right ventricular involvement. No significant difference was found in groups between BNP levels. The TnI levels were higher in patients who presented with isolated inferior MI (P = 0.01) (Table 4). When patients were divided into two groups according to BNP levels, left ventricular end-diastolic diameter (LVEDD) and left ventricular end-systolic diameter (LVESD) were higher, and LVEF was lower in patients with BNP level ≥ 400 pg/mL. The right atrial diameter was larger and TAPSE was lower in the group with BNP level ≥ 400 pg/mL (Table 5). Correlation analysis between BNP levels and echocardiographic parameters showed a relationship between the BNP levels and the RVFAC, TAPSE and left ventricular mass index (LVMI) (Table 6). The systolic blood pressure and left ventricular end-diastolic diameter are independent predictors of BNP levels (Table 7).

Discussion

In this study, we found that RVFAC and TAPSE were lower in...
patients with inferior MI with right ventricular involvement. In the BNP level ≥ 400 pg/mL group, LVEDD and LVESD were larger than BNP level < 400 pg/mL group. The systolic blood pressure and left ventricular end-diastolic diameter are independent predictors of BNP levels. The incidence of acute inferior MI accompanied with right ventricular involvement is ranging from 10% to 50% [9, 10]. The incidence of death, shock and arrhyth-

| Table 3. Right Ventricular Echocardiographic Parameters |
|-----------------------------------------------|
| Variables                        | Inferior MI + RVMI (n = 27) | Isolated inferior MI (n = 34) | P     |
|-----------------------------------------------|
| RSm, cm/s                      | 11.00 ± 1.50 | 13.94 ± 2.17 | 0.90* |
|                                | 13.00       | 12.50       |       |
| RVMPI                          | 0.58 ± 0.14 | 0.61 ± 0.16 | 0.62* |
|                                | 0.57       | 0.57       |       |
| TAPSE, mm                      | 17.04 ± 1.64 | 21.23 ± 2.35 | 0.00**|
|                                | 19.00      | 21.00      |       |
| RVFAC, %                       | 46.37 ± 9.86 | 55.50 ± 7.77 | 0.00**|

RSm: right ventricular systolic wave; RVFAC: right ventricular fractional area change; RVMI: right ventricular myocardial infarction; RVMPI: right ventricular myocardial performance index; TAPSE: tricuspid annular plane systolic excursion. *Independent groups t-test. **Mann-Whitney U test.

| Table 4. Brain Natriuretic Peptide Levels |
|------------------------------------------|
| Variables                  | Inferior MI + RVMI (n = 27) | Isolated inferior MI (n = 34) | P     |
|------------------------------------------|
| BNP, pg/mL                     | 372.92 ± 324.75 | 430.13 ± 320.39 | 0.52* |
|                                | 245.19       | 281.66       |       |
| TnI, ng/mL                     | 1.78 ± 6.13  | 7.11 ± 13.21 | 0.01* |
|                                | 0.12        | 1.38        |       |

Data are shown as median ± standard deviation, and median. BNP: brain natriuretic peptide; RVMI: right ventricular myocardial infarction; TnI: troponin I. *Mann-Whitney U test.

| Table 5. Echocardiographic Parameters With BNP Levels |
|-----------------------------------------------|
| Variables                        | BNP ≥ 400 pg/mL (n = 27) | BNP < 400 pg/mL (n = 38) | P     |
|-----------------------------------------------|
| LA, mm                        | 40.16 ± 4.60 | 39.67 ± 3.85 | 0.599**|
| LA vol index                    | 24.65 ± 8.88 | 25.72 ± 7.70 | 0.594* |
|                                | 21.24      | 25.86      |       |
| LVEDD, mm                      | 52.53 ± 2.53 | 49.51 ± 4.21 | 0.001*|
|                                | 53        | 49        |       |
| LVESD, mm                      | 36.71 ± 4.15 | 33.92 ± 3.94 | 0.004**|
| LVEF, %                        | 55.23 ± 7.28 | 58.92 ± 6.36 | 0.018**|
| LVMI, g/m²                     | 118.34 ± 25.23 | 106.39 ± 23.71 | 0.120**|
| E/E' ratio                     | 11.39 ± 5.63 | 10.70 ± 4.68 | 0.634* |
|                                | 11.43     | 9.12      |       |
| DT, ms                         | 201.35 ± 52.94 | 222.89 ± 64.50 | 0.564**|
| E/A ratio                      | 1.32 ± 0.83 | 1.05 ± 0.50 | 0.634* |
|                                | 0.98 | 0.91 |       |
| RA diameter, mm                | 43.59 ± 4.89 | 40.97 ± 6.98 | 0.059**|
| TAPSE, mm                      | 18.41 ± 2.67 | 20.00 ± 2.96 | 0.042**|
| RVFAC, %                       | 48.86 ± 8.35 | 52.16 ± 10.67 | 0.219**|

Data are shown as median ± standard deviation, and median. BNP: brain natriuretic peptide; DT: deceleration time; E/A: ratio of early to late diastolic filling; E/E’: ratio of early mitral inflow filling to tissue Doppler early filling velocity; LA: left atrium; LA vol index: left atrial volume index; LVEDD: left ventricular end-diastolic diameter; LVEF: left ventricular ejection fraction; LVESD: left ventricular end-systolic diameter; LVMI: left ventricular mass index; RA: right atrium; RVFAC: right ventricular fractional area change; TAPSE: tricuspid annular plane systolic excursion. *Mann-Whitney U test. **Independent groups t-test.
and RVFAC).

Inferior MI with right ventricular involvement (TAPSE, RSm, and LVMI. In addition to that, TAPSE and RSm values were higher in patients with right ventricular EF < 40% compared to patients with inferior MI without right ventricular involvement. We found no significant difference in BNP level between right ventricular function and BNP levels, while no relationship was found between right ventricular diastolic diameter and BNP levels [20]. It may be attributed to the higher pulmonary arterial pressure in these patients. In the present study, there was no significant difference in right ventricular global functions between groups with higher and lower BNP levels. Unlike the study by Nagaya et al, our patients had no pathology of pulmonary hypertension, which contributed to the right ventricular dysfunction. We observed no difference in right ventricular diastolic diameter between groups with higher and lower BNP level. The systolic and diastolic blood pressures were lower in our group with BNP level ≥ 400 pg/mL. We thought that this reduction might be associated with right ventricular MI. Mayer et al evaluated the relationship of BNP levels with LVEF, left atrial diameter, LVEDD, posterior wall and septum thickness in heart failure patients with functional mitral regurgitation. Consequently, they found a positive correlation between LVEDD and BNP levels [21]. In the group with BNP level ≥ 400 pg/mL, we determined 33.3% right ventricular involvement and in the group with BNP level < 400 pg/mL we found right ventricular involvement 66.6%. In the present study, LVEDD and LVEDD were larger, and LVEF and TAPSE were lower in patients with inferior MI with and without right ventricular involvement and BNP level ≥ 400 pg/mL. like as shown in the study which has been presented by Radwan et al [22].

In our study, we concluded that initial BNP levels ≥ 400 pg/mL in patients presenting with acute inferior MI might predict right ventricular dysfunction. In addition, higher rates of hypotension, right ventricular dysfunction and increase in left ventricle diameters were observed in patients with BNP levels ≥ 400 pg/mL.

### Study limitations

Evaluation of right ventricular function only by echocardiography is the first limiting factor of this study. Right ventricular dysfunction is higher in patients with right ventricular involvement [2, 3]. In V4R ST elevation has a sensitivity of 88%, specificity of 78% in the diagnosis of RVMI [11]. Because this is a temporary finding, some patients with acute inferior MI might actually have a nondiagnosed RVMI. BNP is secreted from the ventricles in response to volume expansion or increased pressure load. The BNP level increases in 2 - 4 h after the onset of ischemic symptoms, with a tendency to stabilize over the first 24 h [6]. In the present study, no significant difference was found in BNP level between inferior MI with right ventricular involvement and isolated inferior MI groups. The most important factor that caused this finding was the time difference between onset of symptoms and hospital admission. Serum BNP levels may be varied substantially during this time period. Although BNP levels were higher in patients with inferior MI and right ventricular involvement in a study by Kaya et al, in our study there was no difference in BNP levels since patients with right ventricular involvement were more symptomatic, and they presented earlier [12]. Studies in patients with unstable angina pectoris and MI have demonstrated the prognostic value of BNP along with troponin level [13-15]. In the present study, TnI levels were significantly higher in the group with isolated inferior MI during admission compared to the group with right ventricular involvement. We considered that lower TnI levels in patients with RVMI were associated with the fact that the patients were more symptomatic, and they presented earlier without giving any chance to an increase in TnI level. A study compared BNP levels of patients with NYHA class 2 and 3 heart failure and LVEF of < 45%, and they found a negative correlation between BNP levels and LVEF [16]. Another study showed that BNP levels were higher in patients with right ventricular EF < 40% compared to patients with EF > 40% [17]. We found no correlation between BNP levels and LVEF. However, the higher BNP group had higher LVEDD and LVMI. In addition to that, TAPSE and RSm values were lower. We also found right ventricular systolic dysfunction in inferior MI with right ventricular involvement (TAPSE, RSm, and RVFAC).

Skrzypek et al showed that there was a significant correlation between systolic and diastolic dysfunction with the levels of N terminal probrain natriuretic peptide (NT-proBNP) [18]. Besides another study comparing BNP levels with left ventricular diastolic function parameters, Ono et al showed that mitral inflows (E and A waves) were correlated with BNP levels [19]. And also, they found no significant relationship between BNP levels and isovolumetric relaxation time (IVRT), the earliest abnormality parameter of diastolic function. Similarly, we found no relationship between BNP levels and diastolic dysfunction. In a study by Nagaya et al which compared right ventricular dysfunctions and BNP levels in patients with pulmonary hypertension, there was a relationship between right ventricular functions and BNP levels, while no relationship was found between right ventricular diastolic diameter and BNP levels [20]. It may be attributed to the higher pulmonary arterial pressure in these patients. In the present study, there was no significant difference in right ventricular global functions between groups with higher and lower BNP levels. Unlike the study by Nagaya et al, our patients had no pathology of pulmonary hypertension, which contributed to the right ventricular dysfunction. We observed no difference in right ventricular diastolic diameter between groups with higher and lower BNP level. The systolic and diastolic blood pressures were lower in our group with BNP level ≥ 400 pg/mL. We thought that this reduction might be associated with right ventricular MI. Mayer et al evaluated the relationship of BNP levels with LVEF, left atrial diameter, LVEDD, posterior wall and septum thickness in heart failure patients with functional mitral regurgitation. Consequently, they found a positive correlation between LVEDD and BNP levels [21]. In the group with BNP level ≥ 400 pg/mL, we determined 33.3% right ventricular involvement and in the group with BNP level < 400 pg/mL we found right ventricular involvement 66.6%. In the present study, LVEDD and LVEDD were larger, and LVEF and TAPSE were lower in patients with inferior MI with and without right ventricular involvement and BNP level ≥ 400 pg/mL. like as shown in the study which has been presented by Radwan et al [22].

In our study, we concluded that initial BNP levels ≥ 400 pg/mL in patients presenting with acute inferior MI might predict right ventricular dysfunction. In addition, higher rates of hypotension, right ventricular dysfunction and increase in left ventricle diameters were observed in patients with BNP levels ≥ 400 pg/mL.

### Table 6. Correlation Between Brain Natriuretic Peptide Levels and Echocardiographic Parameters

| Variables | Correlation level | P     |
|-----------|-------------------|-------|
| RSm       | -0.347 x          | 0.045 |
| TAPSE     | -0.402 x          | 0.018 |
| LVMI      | 0.353 x           | 0.048 |

LVMI: left ventricular mass index; RSm: tricuspid lateral annulus S wave; TAPSE: tricuspid annular plane systolic excursion. xPearson’s correlation analysis. *Significant at < 0.05.

### Table 7. Stepwise Logistic Regression Analysis for Brain Natriuretic Peptide Levels

| Variables | B        | Odds ratio | 95% confidence interval | P     |
|-----------|----------|------------|-------------------------|-------|
|           |          |            | Lower limit             | Upper limit |
| SBP       | -0.029   | 0.972      | 0.947                   | 0.996 | 0.025 |
| LVEDD     | 0.281    | 1.324      | 1.090                   | 1.607 | 0.005 |

SBP: systolic blood pressure; LVEDD: left ventricular end-diastolic diameter.
functions can also be evaluated by magnetic resonance and nuclear imaging modalities. However, their high cost, risks posed to the patients as well as unavailability of them as a bedside modality, restricts their routine use. The second limiting factor is the diagnosis of RVMI only with RV4 lead ST elevation which is temporary. Thrombolytic treatment rather than primary percutaneous coronary intervention was preferred treatment modality in our clinic during this study. This is another limitation of this study.

Conflict of Interest

None.

References

1. Gaudron P, Eilles C, Kugler I, Ertl G. Progressive left ventricular dysfunction and remodeling after myocardial infarction. Potential mechanisms and early predictors. Circulation. 1993;87(3):755-763.
2. Zornoff LA, Skali H, Pfeffer MA, St John Sutton M, Rouleau JL, Lamas GA, Plappert T, et al. Right ventricular dysfunction and risk of heart failure and mortality after myocardial infarction. J Am Coll Cardiol. 2002;39(9):1450-1455.
3. Kinch JW, Ryan TJ. Right ventricular infarction. N Engl J Med. 1994;330(17):1211-1217.
4. Dell’Italia LJ, Starling MR, O’Rourke RA. Physical examination for exclusion of hemodynamically important right ventricular infarction. Ann Intern Med. 1983;99(5):608-611.
5. Braat SH, Brugada P, de Zwaan C, Coenegracht JM, Wellsen HJ. Value of electrocardiogram in diagnosing right ventricular involvement in patients with an acute inferior wall myocardial infarction. Br Heart J. 1983;49(4):368-372.
6. Daniels LB, Maisel AS. Natriuretic peptides. J Am Coll Cardiol. 2007;50(25):2357-2368.
7. Kuklińska AM, Sobkowicz B, Mroczko B, Sawicki R, Gąsiorowska M, Kołodziejska M, et al. Prognostic significance of the admission plasma B-type natriuretic peptide measurement in patients with first ST-elevation myocardial infarction in comparison with C-reactive protein and TIMI risk score. Clin Chim Acta. 2007;382(1-2):106-111.
8. Thygesen K, Alpert JS, Jaffe AS, Simoons ML, Chaitman BR, White HD, Joint ESC/ACCF/AHA/WHF Task Force. Third universal definition of myocardial infarction. J Am Coll Cardiol. 2012;60(16):1581-1598.
9. GISSI-3: effects of lisinopril and transfemoral glyceryl trinitrate singly and together on 6-week mortality and ventricular function after acute myocardial infarction. Gruppo Italiano per lo Studio della Sopravvivenza nell’infarto Miocardico. Lancet. 1994;343(8906):1115-1122.
10. Spencer FA, Allegrone J, Goldberg RJ, Gore JM, Fox KA, Granger CB, Mehta RH, et al. Association of statin therapy with outcomes of acute coronary syndromes: the GRACE study. Ann Intern Med. 2004;140(11):857-866.
11. Somers MP, Brady WJ, Bateman DC, Mattu A, Perron AD. Additional electrocardiographic leads in the ED chest pain patient: right ventricular and posterior leads. Am J Emerg Med. 2003;21(7):563-573.
12. Kaya MG, Özdogru I, Kalay N, Dogan A, Inanc T, Gul I, Gunebakmaz O, et al. Plasma B-type natriuretic peptide in diagnosing inferior myocardial infarction with right ventricular involvement. Coron Artery Dis. 2008;19(8):609-613.
13. de Lemos JA, Morrow DA, Bentley JH, Omland T, Sabatine MS, McCabe CH, Hall C, et al. The prognostic value of B-type natriuretic peptide in patients with acute coronary syndromes. N Engl J Med. 2001;345(14):1014-1021.
14. Lindahl B, Lindback J, Jernberg T, Johnston N, Stridsberg M, Venge P, Wallentin L. Serial analyses of N-terminal pro-B-type natriuretic peptide in patients with non-ST-segment elevation acute coronary syndromes: a Fragmin and fast Revascularisation during In Stabiliy in Coronary artery disease (FRISC)-II substudy. J Am Coll Cardiol. 2005;45(4):533-541.
15. Katlandur H, Kalay N, Yilmaz Y, Gur M, Sarli B, Oguz F, Kasapkara A, et al. Relationship between B-type natriuretic peptide and coronary flow in patients with myocardial infarction of ST segment elevation receiving thrombolytic therapy. Eur J Health Sci. 2015;1:54-57.
16. Kjaer A, Hildebrandt P, Appel J, Petersen CL. Neurohormones as markers of right- and left-sided cardiac dimensions and function in patients with untreated chronic heart failure. Int J Cardiol. 2005;99(2):301-306.
17. Mariano-Goulart D, Eberle MC, Boudousq V, Hejazi-Moughari A, Piot C, Caderas de Kerleau C, Verdier R, et al. Major increase in brain natriuretic peptide indicates right ventricular systolic dysfunction in patients with failure. Eur J Heart Fail. 2003;5(4):481-488.
18. Skrzypek A, Nessler P. Asymptomatic ischemic heart dysfunction, echocardiographic changes and NT-proBNP during 2-years observation. Przegl Lek. 2014;71:378-383.
19. Ono M, Tanabe K, Asanuma T, Yoshitomi H, Shimizu H, Ohta Y, Shimada T. Doppler echocardiography-derived index of myocardial performance (TEI index): comparison with brain natriuretic peptide levels in various heart diseases. Jpn Circ J. 2001;65(7):637-642.
20. Nagaya N, Nishikimi T, Okano Y, Uematsu M, Satoh T, Kyotani S, Kuribayashi S, et al. Plasma brain natriuretic peptide levels increase in proportion to the extent of right ventricular dysfunction in patients with pulmonary hypertension. J Am Coll Cardiol. 1998;31(1):202-208.
21. Mayer SA, De Lemos JA, Murphy SA, Brooks S, Roberts BJ, Grayburn PA. Comparison of B-type natriuretic peptide levels in patients with heart failure with versus without mitral regurgitation. Am J Cardiol. 2004;93(8):1002-1006.
22. Radwan H, Selem A, Ghazal K. Value of N-terminal pro brain natriuretic peptide in predicting prognosis and severity of coronary artery disease in acute coronary syndrome. J Saudi Heart Assoc. 2014;26(4):192-198.