Plasma Brain Natriuretic Peptide Levels in Rheumatic Mitral Stenosis and Regurge

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Abstract: Rheumatic heart disease (RHD) is still an important cause of heart failure in developing countries. Although there are a few studies of Brain natriuretic peptide (BNP) levels in patients with mitral stenosis and regurge, to our knowledge, no previous study has systematically assessed the levels of BNP and the factors stimulating BNP secretion in rheumatic heart disease. BNP is a cardiac hormone secreted from the ventricular myocardium as a response to ventricular volume expansion and pressure overload. The objective of this work was to measure the levels of BNP in patients with rheumatic mitral stenosis and regurge and its clinical and echocardiographic correlation. We studied 45 patients, 15 patients with rheumatic mitral stenosis, 15 rheumatic mitral regurge and 15 healthy subjects as a control group. The results of this study showed that there was statistically significant difference between the BNP plasma level in mitral stenosis and regurge patients, and group control subjects. Also there was positive correlation between the plasma level of BNP and the severity of MVD, left ventricular end systolic dimension, left ventricular end diastolic dimension, left atrial size and that there was reverse significance correlation between the plasma level of BNP and left ventricular ejection fraction, but there was no correlation with the gender of the subjects or the presence or absence of pulmonary hypertension. The conclusion from our results suggested that in rheumatic, chronic MVD, BNP activation is present and biologically active and reflects essentially the hemodynamic, ventricular and atrial consequences. Thus, BNP emerge as a biomarker of severity of MVD consequence.

Keywords: Plasma; Brain; Natriuretic Peptide; Rheumatic Mitral Stenosis; Regurge

1. Introduction

Rheumatic heart disease is a nightmare of the modernized developing countries as it carries a lot of disability limitations to the youngsters and middle aged population needed as an arm for building and work. As known many years ago, rheumatic heart disease is the disease of the mitral valve as it carries the highest statistical mitral valve affection preceding the degenerative causes of mitral valve disease. (Bartoli et al., 1984)

Unlike ANP, whose major storage sites include the atria and the ventricle, the major source of plasma BNP is cardiac ventricles (Fishban et al., 1996). This suggests that the BNP may be a more sensitive and specific indicator of ventricular disorders than other natriuretic peptides (Nagagawa et al., 1995). Unlike ANP, BNP has a minimal presence in storage granules (Shimamoto et al., 2007). Recent studies have demonstrated that plasma BNP is also elevated in patients with valvular lesions. This observation is important because in various cardiac diseases, BNP activation reflects hemodynamic alteration, detects LV dysfunction; and provides prognostic information and thus was toughed as an important clinical tool. (Rodeheffer et al., 2004). Although Echocardiography is the standard method used to evaluate the severity of mitral valve affection and to assess left ventricular systolic function, however it may be difficult to obtain an accurate quantitative assessment. (Zehra et al., 2004). In fact, BNP is really an indicator of raised intra-cardiac pressure, irrespective of whether the raised intra-cardiac pressure is caused by LV hypertrophy (LVH), LV Systolic dysfunction, valve disease or even fast atrial fibrillation. (Meret et al., 1999). The plasma levels of BNP, N-BNP, and ANP rose with increasing severity of MR and MS and with increasing LA dimensions. The correlation coefficients were similar for BNP, N-BNP, and ANP. (Otto et al., 2001). The natriuretic peptide levels of the symptomatic patients with MS after adjustment for age, gender and body surface area; the level of BNP was on average higher in symptomatic patients with MS than in normal control, attributed to the diastolic dysfunction associated with the MS. The difference between symptomatic and asymptomatic patients with MS remained after adjustment as also compared to LV end-systolic dimension (Zehra et al., 2004).

2. Patients and Methods

We studied 45 patients classified into 3 groups, group I included 15 patients with rheumatic mitral stenosis, group II include 15 patients with rheumatic mitral regurge and 15 normal subjects as a control.
group. The patients were selected from those cases admitted to the Cardiology Department at Bab-El Sharia'a University Hospital from October 2011 to June 2012.

**Inclusion criteria:**
1- Chronic mitral rheumatic heart disease stenosis or regurgite.
2- Ejection fraction were within normal range (above 50) to exclude heart failure as a reason for BNP elevation.
3- Normal sinus rhythm.

**Exclusion criteria**
1- Previous history of myocardial infarction
2- Patients with Atrial Fibrillation.

**Control subjects**
Fifteen normal volunteers were studied. Control subjects have no evidence of chronic illness and all had satisfactory echocardiographic imaging results.

Full history taking, general clinical examination, local cardiac examination searching for manifestations of mitral regurgite resting dyspnea, orthopnea, and possibly signs of diminished forward flow, S1 usually diminished with chronic MR murmur usually holosystolic blowing in quality heard best at apex, or stenosis (accentuation of first and second heart sounds, presence of opening snap with disappearance of the splitting.

Standard twelve - lead electrocardiogram (12 – lead ECG) to exclude atrial fibrillation or myocardial infarction.

**Laboratory investigations:**

**Sampling:**
Six ml of venous blood were drawn from each subject by venipuncture under complete sterile conditions after the patient’s permission and used as the following:

One ml in EDTA tube for CBC.

Two ml in EDTA tube were centrifuged and the separated plasma kept frozen at -20C until used for assay of BNP.

The rest of the sample was collected in plain tube, incubated and left to be clotted then centrifuged and the separated serum used for routine lab investigations including: liver functions, kidney functions, and random blood glucose.

**Laboratory testing:**
Complete blood count using automated haematology cell counter Cell Dyn 1700 (Abott, USA).

Liver functions, kidney functions, and random blood glucose were determined by fully automated clinical chemistry auto-analyzer Hitachi 912 (Roche diagnostics).

**Measurement of BNP:**

Detection range of the BNP assay is 10-1300 pg/ml.

- The normal range is between 3-50 pg/ml.
- The grey zone ranges between 51-600 pg/ml.
- Manifested heart failure ranges between 601-1300 pg.

Our study was subjected to the grey zone, which is the pre heart failure area, to detect the early increase in BNP in the patients susceptible for myocardial impairment, either due to rheumatic myocardial affection (fibrosis & myocarditis), or due to the valve affection of the haemodynamics of the heart chambers.

**Echocardiographic study**
Both patients and control subjects were studied by transthoracic echocardiography using a commercially available echocardiographic machines equipped with 2.5 MHZ phased array transducers. The study was performed by experienced operator (at least 5 years of high volume practice).

Two dimensional (2D), M- mode and Doppler echocardiography techniques were applied for obtaining a complete echocardiographic recordings as follows:

A) 2-dimensional echocardiographic technique were performed in the standard method in parasternal long, short axis views and apical 4- and 2- chamber views to detect the following:

1- The overall ventricular performance.
2- Segmental wall motion abnormalities.
3- Mitral annulus diameter.
4- Valvular thickening, abnormal mobility or overlying masses.
5- Intracardiac masses or thrombi
6- Pericardial effusion or thickening.

Mitral stenosis grading, the rate of decline in pressure gradient (from the early peak velocity which is altered by obstruction) can be expressed in terms of the pressure half-time which is the time needed for the initial peak diastolic gradient to decline.

B) M-mode echocardiography was used to detect chamber dimensions. Ejection fraction was calculated by the M-mode method. Normal ventricular function was defined as normal when LV end diastolic diameter was between (4 to 5.8 cm), LV end systolic diameter was between (2.2 to 4 cm), no major segmental wall motion abnormalities. Left atrial dilatation was defined as LA size > 4 cm by M- mode echocardiography. Left ventricular hypertrophy was defined as LV wall thickness of septum and posterior wall of > 1.1 cm.

C) Color Doppler echocardiography allows diagnosis of mitral regurgite by means of visualization.
of regurgitant jet or jets entering the left atrium and allows the assessment of severity by comparing the regurgitant jet area with the left atrial area. Mitral valve regurge was graded from (I) to (IV) as follows:

Grade (I) regurgitant jet area/left atrial area ratio 5-25%.
Grade (II) ratio = 26-50%
Grade (III) ratio = 51-75%
Grade (IV) ratio = 76-100%. (Essop et al.,1991)

D) Doppler echocardiography of mitral inflow.

Patients were diagnosed as having mitral regurge or stenosis when are fulfilled the criteria of it. Pulsed wave doppler is used for localization of the regurgitant jet rather than finding it. Also measuring the mitral inflow by continuous wave Doppler is giving more information about minimal regurge without localization.

**Statistical Analysis:**

Data were collected and submitted to statistical analysis. The following statistical tests and parameters were used:

Table (1):Gender of subjects under study

<table>
<thead>
<tr>
<th>Groups</th>
<th>Gender</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Male</td>
<td>Female</td>
</tr>
<tr>
<td>Group I</td>
<td>7</td>
<td>8</td>
</tr>
<tr>
<td>%</td>
<td>46.67</td>
<td>53.33</td>
</tr>
<tr>
<td>Group II</td>
<td>8</td>
<td>7</td>
</tr>
<tr>
<td>%</td>
<td>53.33</td>
<td>46.67</td>
</tr>
<tr>
<td>Group III</td>
<td>9</td>
<td>6</td>
</tr>
<tr>
<td>%</td>
<td>60.00</td>
<td>40.00</td>
</tr>
<tr>
<td>Chi-square</td>
<td>X²</td>
<td>0.133</td>
</tr>
<tr>
<td>P-value</td>
<td></td>
<td>0.936</td>
</tr>
</tbody>
</table>

2-Laboratory findings:

**Gender in relation to BNP (Table 2):**

The thirty (30) patients were divided equally into fifteen (15) males and fifteen (15) females. The BNP measurements showed no significant pattern of differentiation between males and females whether in stenosis or regurge. The male BNP studies ranged between (62-433 pg) with mean of (238 pg) and (SD) of 99.7, while the female measurements ranged between (106-416 pg) with mean of (260 pg) and (SD) of 120.72. P-value showed no significance (0.584).

Table (2): Relation between BNP and sex of the patients.

<table>
<thead>
<tr>
<th>Sex</th>
<th>BNP Range</th>
<th>Mean ± SD</th>
<th>T-test</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Male</td>
<td>62.00 - 433.00</td>
<td>238.533 ± 99.752</td>
<td>-0.554</td>
<td>0.584</td>
</tr>
<tr>
<td>Female</td>
<td>106.00 - 416.00</td>
<td>260.933 ± 120.728</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Sensitivity & specificity of BNP measurement (Table 3):

Showing that BNP showed 100% specificity and 100% sensitivity between the control group & the other 2 groups collectively (patients with mitral stenosis & mitral regurge). As the cutoff value of BNP rising was demarcated at (>50 pg), all the control individuals results was under the cutoff value [Maximum = 41 pg] with 100% accuracy.
Table (3): ROC curve for BNP between patients and control groups

<table>
<thead>
<tr>
<th>Cutoff</th>
<th>Sens.</th>
<th>Spec.</th>
<th>PPV</th>
<th>NPV</th>
<th>Accuracy</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 50</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
<td>100.0</td>
</tr>
</tbody>
</table>

Difference between BNP level between mitral stenosis and regurge (Table 4):

BNP elevation in cases with mitral regurge was of higher significance than that of mitral stenosis, as we determined the cutoff value here (>360) as the centre of the grey zone (between 50 pg & 600 pg), which is the area between normal subjects and subjects with proven heart failure. The test here was highly specific (91.3%) with positive predictive value of 85.6 and accuracy of 0.566.

Table (4): ROC curve for BNP between Mitral stenosis and regurge

<table>
<thead>
<tr>
<th>Cutoff</th>
<th>Sens.</th>
<th>Spec.</th>
<th>PPV</th>
<th>NPV</th>
<th>Accuracy</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 360</td>
<td>46.7</td>
<td>91.3</td>
<td>85.5</td>
<td>63.6</td>
<td>0.566</td>
</tr>
</tbody>
</table>

BNP and the severity of mitral regurge (Table 5):

In this study, fifteen patients were tested with mitral regurge varying from moderate to severe affection, moderate is grades I-II/IV, and severe are gradeIII/IV. We excluded patients with grade IV/IV from the study as all showed symptoms of heart failure. The table below is showing increase of BNP value from (198.8 pg) with standard deviation (SD) of (64.37) in moderate cases, to (259.12 pg) with standard deviation (SD) of (111.4) in severe cases, with P-value of 0.217, which implies a slight significance.

Table (5): Relation between BNP and the severity of mitral regurge

<table>
<thead>
<tr>
<th>Regurge</th>
<th>BNP</th>
<th>T-test</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Range</td>
<td>Mean ± SD</td>
</tr>
<tr>
<td>Moderate</td>
<td>60.00 - 260.00</td>
<td>196.857 ± 60.375</td>
</tr>
<tr>
<td>Severe</td>
<td>108.00 - 400.00</td>
<td>259.125 ± 110.389</td>
</tr>
</tbody>
</table>

3-Echocardiographic data (table 6):

All variables seen in the echocardiographic study were put in comparison to the levels of the BNP measured for the patients with both mitral regurge and stenosis.

Table (6): the variables of echocardiographic analysis with BNP value

<table>
<thead>
<tr>
<th>Variables</th>
<th>BNP</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>PG</td>
<td>0.441</td>
<td>0.100</td>
</tr>
<tr>
<td>LA</td>
<td>0.675</td>
<td>&lt;0.001*</td>
</tr>
<tr>
<td>LVED</td>
<td>0.532</td>
<td>0.002*</td>
</tr>
<tr>
<td>LVES</td>
<td>0.535</td>
<td>0.002*</td>
</tr>
<tr>
<td>EF%</td>
<td>-0.545</td>
<td>0.002*</td>
</tr>
<tr>
<td>Pulm. gradient</td>
<td>0.103</td>
<td>0.595</td>
</tr>
</tbody>
</table>

Left atrium diameter & BNP (Table 7):

Showing that the patients were divided according to LA size as estimated by echocardiography into 2 subgroups, the first subgroup whose LA size was < 4 cm (n = 5/30) and the second subgroup with LA size > 4 cm (n = 25/30). The plasma levels of BNP was statistically significant positive correlation in comparing the two subgroups (p = 0.05).

Table (7): Mean values of BNP (pg/ml) according to LA size (cm) in the studied groups

<table>
<thead>
<tr>
<th>LA &lt; 4 cm (n = 5)</th>
<th>LA &gt; 4 cm (n = 25)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>5.96 ± 1.79 (170 ± 63)</td>
<td>7.97 ± 1.74 (244 ± 58)</td>
<td>p &lt; 0.001</td>
</tr>
</tbody>
</table>

p < 0.001 = significant.
LV end systolic diameter & BNP

By doing correlation between the plasma level of BNP and the LVES, it was divided the patients group with MVD according to echocardiography of LVES into 2 groups, the 1st group had LVES< 4 cm (n=10), and found that the plasma level of BNP of this group was 8.0 ± 1.68 pg/ml and the 2nd group had LVES ≥ 4 cm (n=20) also, found that the plasma level of BNP of this group was 8.0 ± 1.81 pg/ml so no significant correlation between the plasma level of BNP and the LVES.

Table (8): Mean values of BNP (pg/ml) according to LVES (cm) in the studied groups.

<table>
<thead>
<tr>
<th>LVES &lt; 4 cm (n=10)</th>
<th>LVES ≥ 4 cm (n=20)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>8.62 ± 1.83 (270±43)</td>
<td>8.79 ± 1.6 (290±49)</td>
<td>P&lt;0.002</td>
</tr>
</tbody>
</table>

P<0.002= very significant. ** P<0.01 relative to LVES < 4 cm

LV end diastolic diameter & BNP (Table 9):

According to LVED, it was found in patients with MVD and had LVED< 6 cm (n=25) that their plasma level of BNP was 7.17 ± 1.6 pg/ml and in patients with MVD and had LVED ≥ 6 cm (n=5) that their plasma level of BNP was 8.23 ± 1.89 pg/ml, so there was positive correlation between the plasma level of BNP and the LVED.

Table(9): Mean values of BNP (pg/ml) according to LVED (cm) in the studied groups.

<table>
<thead>
<tr>
<th>LVED &lt; 6 cm (n=14)</th>
<th>LVED ≥ 6 cm (n=16)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>7.17 ± 1.6 (309±40)</td>
<td>8.23 ± 1.89 (400±71)</td>
<td>P&lt;0.002</td>
</tr>
</tbody>
</table>

P<0.002= significant.

Ejection fraction & BNP (Table 10):

According to LVEF%, there was reverse significant correlation between the plasma level of BNP and the LVEF% as the patients divided into 2 groups, the 1st had LVEF% ≤ 55 (n=5), and the BNP concentration was 8.89 ± 1.39 pg/ml and the 2nd group had LVEF% > 55 (n=25), we found the BNP was 7.58 ± 1.84 pg/ml. This correlation meant that whenever the ejection fraction increases, the BNP levels decreased proportionately.

Table 1: Mean values of BNP (pg/ml) according to LV EF (%) in the studied groups.

<table>
<thead>
<tr>
<th>LV EF (%) ≤ 55 (n=5)</th>
<th>LV EF (%) &gt; 55 (n=25)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td>8.89 ± 1.39(440±35)</td>
<td>7.58 ± 1.84(212±69)</td>
<td>&lt;0.002 (RS)</td>
</tr>
</tbody>
</table>

RS= Reverse significant. r=-0.545

4. Discussion

Rheumatic heart disease may be viewed as a disease of the mitral valve, other valves may be involved both anatomically & functionally, but anatomically, mitral valve is always affected. Aschoff bodies have never been formed in hearts without anatomic disease of the mitral valve (Roberts, 1983)

Rheumatic heart disease is a nightmare of the modernized developing countries as it carries a lot of disability limitations to the youngsters and middle aged population needed as an arm for building and work. (Bartoli et al., 1984)

Unlike ANP, whose major storage sites include the atria and the ventricle, the major source of plasma BNP is cardiac ventricles (Yoshimura et al., 1993 and Fishman et al.,1996). This suggests that the BNP may be a more sensitive and specific indicator of ventricular disorders than other natriuretic peptides (Nagagawa; 1995). Unlike ANP, BNP has a minimal presence in storage granules (Shimamoto et al., 2007). BNP release appears to be directly proportional to ventricular volume expansion and pressure overload (Meada, 1998). BNP is an independent predictor of high LV end-diastolic pressure and is more useful than ANP or epinephrine for assessing mortality in patients with chronic congestive heart failure. BNP has been proposed as a tool to distinguish dyspnea of congestive heart failure from other causes of dyspnea. (Yoshimura, 1993).

In this study patients were divided into 3 groups: Fifteen patients (group I) who had mitral stenosis of different degrees of severity, fifteen patients (group II) having mitral regurge with different grades of severity and fifteen control normal subjects (group III). Echocardiography was performed after clinical history and examination to detect the patients who
had mitral regurgitation or stenosis and to estimate its severity, those found to have heart failure or AF were excluded and the rest of patients were included. Then a sample of blood was drawn immediately after the echocardiography.

In the present study, the studied patients were equally chosen between males and females (15/30) and their age ranged from 20 to 36 years. All cases were rheumatic in etiology. But in the study done by Timothy et al., in 2003 most patients were males and the mean age was 55 ± 19 years as mitral valve prolapse was the main cause.

There was no significant change of BNP with age and gender which disagreed with the study done by Micah et al., in 2004, showing that the BNP was significantly higher in females (1926 ± 440 vs. 1147 ± 186 pg/ml, P=0.03) and was positively correlated with age (P=0.44, P<0.01). That’s because our study involved small cut section of age and gender signifying the golden age of rheumatic heart disease while Micah et al. study involved older scale of age (mean age was 43), larger number of candidates (122 patient, 70% were females), with variety of etiologies (Ischemic and degenerative causes more than rheumatic causes).

In the present study, the mitral valve regurgitation was assessed by the jet area and pulsed wave doppler of mitral inflow as these procedures are easy to perform and sensitive for estimation of severity of MR. Also, mitral stenosis was assessed using quantification of stenotic area by 2D echocardiography and measurement of mitral valve area by pressure half-time method using Doppler study.

There was statistically significant increase in BNP plasma level in groups (I) and (II) patients with MS and MR compared to group (III) the control group (P<0.01). This agreed with the study done by Zehra Golapsy et al., (2004), that showed statistically significant increase in BNP plasma levels in patients with rheumatic heart disease (All varieties of valve lesions, isolated and combined) as compared to control subjects (p<0.0001).

In the present study, the patients were divided according to LA size as estimated by echocardiography into 2 subgroups, the first subgroup whose LA size was < 4 cm (n= 5/30) and the second subgroup with LA size > 4 cm (n= 25/30). The plasma levels of BNP was statistically significant positive correlation in comparing the two subgroups (p<0.05). There was statistically significant positive correlation between the plasma levels of BNP and the LA size(r= 0.675; p= 0.001**). This also agreed with the study done by Timothy et al., in 2003 that showed that the level of each natriuretic peptide rose with increases in left atrial (LA) dimensions.

In another study done by Sutton et al., in 2003, showing that the level of each natriuretic peptides rose with increases in left atrial (LA) dimensions that agreed with our study.

In the present study, the patients were divided according to LVESD into 2 subgroups, the 1st subgroup had LVESD< 4 cm and the 2nd subgroup had LVESD ≥ 4 cm. It was found that there was significant increase in the plasma levels of BNP in 2nd subgroup in compared with the 1st subgroup.

It was found that there was positive correlation between the plasma level of BNP and the LVESD in the mitral valve regurgitation group this agreed with the study done by Timothy et al., in 2003, that showed no significant correlation between any natriuretic peptide and LV dimensions.

Another study done by Sutton et al., in 2003 that agreed with this study was that there was no significant correlation between natriuretic peptides and LV dimension.

In the present study, the patients were divided according to LVEDD into 2 subgroups, 1st subgroup patients had LVEDD< 6 cm and 2nd subgroup patients had LVEDD ≥ 6 cm. It was found that there was significant increase in the plasma levels of BNP in 2nd subgroup in compared with the 1st subgroup. And found there was positive correlation between the plasma level of BNP and the LVEDD In the study done by Zilda et al., 2006) the BNP levels had a positive correlation with left ventricular end-diastolic dimensions.

The current study was contradicted with the study done by Timothy et al., in 2003, showing no statistically significant correlation between, LV end-diastolic dimension and natriuretic peptide levels. The study done by Micah et al., in 2004 showed that there was no significant change of BNP and LVEDD and in the study done by Sutton et al., 2003, no significant correlation between any natriuretic peptides and LV dimension was demonstrated and so the study of Brookes et al., 1997.

In the present study, there was reverse significance between the plasma level of BNP in the patients with LVEF% ≤ 55 and in patients with LVEF% > 55. (r= -0.545; p= 0.002).

A study done by Susan et al., in 2004, hypothesized that MR is associated with higher BNP levels. When taking into account only LV ejection fraction and degree of MR (moderate or severe) they were significantly positive correlation associated with BNP levels. When MR was grouped as any MR versus no MR, only LV ejection fraction and any degree of MR was significantly positive correlation associated with BNP plasma level.

In the study done by Zilda et al., 2006, that showed that BNP levels have an inverse correlation
with left ventricular function. This study agreed with the result of the current study showing significant inverse correlation between the plasma level of BNP and the LVEF%. On the contrary, Sutton et al. in (2003) showed that there was no significant correlation between any natriuretic peptides and EF.

In the present study, it was found that there was significant inverse correlation between the plasma level of BNP and LVEF and this disagreed with study done by Lgor et al. in (2004), showing that the BNP concentration in the studied groups was non significant as compared with LVEF. In the study done by Timothy et al., in (2003), that showed no significant correlation between any natriuretic peptide and the EF.

The present study found that there was a positive correlation between the plasma level of BNP and degree of MR. There was statistically significant increase in the plasma level of BNP in patients with MR grade II and grade III as compared to patients with MR grade I this agreed with the study done by Timothy et al., in (2003), that showed the level of natriuretic peptide rose with increasing severity of MR (p< 0.05 relative to type I).

The study done by Sutton et al. in (2003), showed that the level of natriuretic peptides rose with increasing severity of MR.

The study done by Brookes et al. (1997) showed that the BNP levels increased according to the severity of mitral regurgitation and were greater in symptomatic patients.

The study by Delphine et al. (2005) showed that the LV end-systolic volume index, LA volume, atrial fibrillation, clinical symptoms and conversely MR degree are not independently associated with BNP.

In the present study, there was positive correlation between the plasma level of BNP and the presence of MS (To lesser extent correlated with the severity). This results were matched partially with the study done by Robert et al. (2006) which stated that BNP can be useful as a prognostic predictor for mitral stenosis patients as it increases with severity.

A study by Ceyhan et al. (2007) may shed a light on why MS is associated with increased level of BNP while the LV dimensions are more or less preserved. In this study it was found that the levels of NT pro-BNP levels were elevated in patients with LV diastolic dysfunction, even in the early stages of it. The candidates of this study were the hypertensive patients of mean age of (52 ±2 years) without symptoms or signs of heart failure. The results were highly significant using the ratio of early diastolic transmitral E wave velocities to tissue Doppler mitral annulus early diastolic E’ wave velocities (E/E’).

Conclusion

In rheumatic, chronic MVD, BNP activation is present and biologically active and reflects essentially the hemodynamic, ventricular and atrial consequences. Thus, BNP emerge as a biomarker of severity of MVD consequences and of poor clinical outcome in patients with it. BNP measurement should be considered in patients with moderate to severe MVD to support the clinical decision-making process and to foresee the prognosis of the surgery.

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References


