Beni-Suef University Journal of Basic and Applied Sciences 7 (2018) 306–309

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Beni-Suef University
Journal of Basic and Applied Sciences

journal homepage: www.elsevier.com/locate/bjbas

Review Article

BNP as a promising marker in prediction of malignant arrhythmias in pts with LV systolic dysfunction after an acute MI

Ahmed Yassin*, MohamedAbo Hamila, Ahmed Battah, Amal Rizk, SheriefMokhtar

Critical Care Department, Cairo University, Cairo, Egypt

1. Introduction

Natriuretic peptides are released from the heart, in situations of pressure and volume overload of the ventricles. During the last decade, B-type natriuretic peptide (BNP) has been proposed as a useful marker for the determination of acute and chronic left ventricular dysfunction and the severity of systolic left ventricular dysfunction (Bassan et al., 2005, Potocki et al., 2009).

These peptides are powerful markers for prognosis, diagnosis, and risk stratification in heart failure (HF) patients In almost of all Previous studies (Maisel, 2001; Gardnera et al., 2003; Koglin et al., 2001; Harrison, 2002), Few Studies only (Tapanainen et al., 2004; Heeschen et al., 2004; Omland et al., 2002; Galvani et al., 2004) had investigated that role of plasma BNP or NT-pro BNP levels are associated with an increased risk of ventricular arrhythmia in patients with acute coronary syndrome.

And despite of all advances in management of ventricular arrythmia by pharmacological treatment and effectiveness of implantable cardioverter defibrillators (ICDs) as effective therapeutic measure for such problem, ventricular arrhythmia remains major cause of sudden cardiac death in these group of patients (Ray and Reddy, 2005, Josephson and Wellens, 2004, Huikuri et al., 2003).

So, improving risk stratification in order to identify patients at increased risk for SCD and patients who would not get benefit and exposed to potential adverse effects (Ray and Reddy, 2005, Josephson and Wellens, 2004, Huikuri et al., 2003). Would achieve the maximum benefit from ICD therapy,

Since a relationship between B-type natriuretic peptides and ventricular arrhythmia has been suggested (Berger et al., 2002; Hansen et al., 1990; Franz et al., 1992; Reiter, 1996; Zhu et al., 1997).

Our study aimed to evaluate the importance of BNP levels in predicting the occurrence of malignant arrhythmias in patients with Left ventricular dysfunction .Early after an acute myocardial infarction, BNP is released as a result of ischemia and necrosis of myocardial cells. Afterwards, BNP rises as a result of systolic or diastolic dysfunction and increased wall stress of the left ventricle (Huikuri et al., 2003, Maisel, 2001; Berger et al., 2002).

2. Aim of the work

Evaluate the role of B-type natriuretic peptide (BNP), in predicting ventricular arrhythmia within 90 days from the onset of MI in pts developed LV dysfunction in this period.

The present study is a prospective observational study conducted on 60 patients diagnosed to have acute STEMI complicated by LV systolic dysfunction & admitted to critical care department of Kasr El-Aini Hospital, Cairo University.

2.1. Our patients included

Cases who diagnosed to have STEMI (46 pts) and underwent primary intervention by PCI, and those received thrombolytic therapy (14 pts).

2.2. Inclusion criteria

Adult patients Diagnosed to have an acute myocardial infarction (STEMI), only if (at least 2 criteria of the following) were met:

1- Typical retro sternal chest pain (relieved by nitrates).
2- ST segment elevated greater than 0.1 mV in limb leads or 0.2 mV in precordial leads or new or indeterminate LBBB.
3- Elevated cardiac biomarker suggestive of myocardial injury in early few hours.

Echocardiographic evidence of LV systolic dysfunction (LVEF < 50%).

2.3. Exclusion criteria

1. Renal impairment defined as serum creatinine level more than 1.5 mg/dl that causes a non specific elevation of troponin.
2. Systemic sepsis that also causes a non specific elevation of troponin.
3. Cardiac arrest before taking samples.
4. Patients with cerebro vascular stroke.
5. Advanced malignancy.

* Corresponding author.
E-mail address: manalyoussef@yahoo.com (A. Yassin).

https://doi.org/10.1016/j.bjbas.2018.02.008
Received 15 February 2018; Accepted 28 February 2018
Available online 10 March 2018

All patients were subjected to the following:

(1) Informed consent from the patient or the closest family member.
(2) Detailed medical history taken from the patient or a family member
(3) Clinical assessment
(4) Surface 12 leads ECG
(5) Transthoracic echocardiographic examination:
(6) Biochemical measurement

Clinical assessment in the form of:

- Full physical examination.
- Clinical scoring systems: (New York Heart functional classification).

Surface 12 leads ECG:

Twelve lead ECG with consistent chest leads positioning performed daily for 5 days.

ECG was considered abnormal if the following was detected:

- ❖ Elevation in ST segment greater than 0.1 mV in limb leads, 0.2 mV in precordial leads.
- ❖ Arrhythmias.

Transthoracic echocardiographic examination:

We examined each patient in our studied population, in the left lateral position according to the recommendations of the American Society of Echocardiography (ASE). The study was conducted using an ATL HDI 5000 colored echocardiographic machine, using a 3.5 MHz transducer. 2D and M-mode for assessment of;

- Left Ventricular End diastolic & End systolic dimensions (EDD, ESD),
- Left Ventricular Ejection Fraction (EF),
- Regional Wall Motion Abnormalities (RWMAs), and
- Valvular affection.

Biochemical measurement:

- ❖ Full laboratory investigation.
- ❖ Cardiac biomarkers (Cardiac troponin I, CK MB, CPK, were measured on admission).
- ❖ Plasma Samples withdrawn from every patient in our study in the first 48 h, to assess **BNP**

3. Results

Our study is prospective observational study conducted on 60 adult patients admitted to critical care department (Cairo university) as having acute MI and post MI L.V systolic dysfunction throughout the period from September 2014 to March 2015.

3.1. Demographic and descriptive data

Our study was conducted on sixty patients with LV dysfunction, complicating the course of acute MI. Mean age of studied patients was 57.6 ± 8.4 years old (Range 35–80). Males constituted 73.3% of our study population (44 males and 16 females).

Forty six pts were subjected to primary percutaneous coronary intervention (76.7%) while fourteen pts received medical treatment (23.3%).

3.1.1. Clinical examination

Risk category stratification in our patients, showed mean NYHA 2.8, Killip class of 2.9 and TIMI risk score of 8.3 as shown in Table 1:

Table 1
Clinical examination in NYHA, Killip Class.

Clinical examination	
NYHA	2.8 ± 0.8
KILLIP class	2.8 ± 0.8
TIMI risk point	7.8 ± 2.4

Table 2
Initial echocardiographic examination.

Echocardiographic examination	
LVEF	41.9 ± 8%
RWMA	13.3 ± 3.3

3.1.2. Follow up clinical examination

Follow-up clinical examination was done for survivors and showed a mean NYHA: 1.9 ± 0.7.

Follow-up was done at 90 days. Forty eight patients survived (80%) from those seven patients documented in life-threatening arrhythmias (11.7%) and sudden cardiac death were developed in 12 pts in our population.

3.1.3. Initial echocardiographic examination

Echocardiographic examination was done for all patients and showed a mean EF of 41.9 ± 8% with RWMA: 13.3 ± 3.3 as shown in Table 2 (see Fig. 1).

Twenty seven patients had initial EF value higher than or equal to 45%, while 20 patients had EF values ranging between 35 and 45% and only 13 patients had values lower than 35%.

3.1.4. Follow up echocardiographic examination

Follow-up echocardiographic examination was done for survivors and showed a mean EF of 46.7 ± 8.8% with RWMA: 8.7 ± 4.1 as shown in Table 3 (see Tables 4 and 5 Fig. 2):

Plotting ROC curve to estimate the best predictive cutoff points as predictors of sudden cardiac death pro-BNP revealed the following (see Fig. 3):

3.2. Examining the prognostic accuracy of pro-BNP using survival analysis

Kaplan Meier analysis shows that rise of pro-BNP above 3.2 ng/ml has a significant predictive impact upon SCD, (OR 0.748 [CI 95%: 0.07–0.932], P value .039) (Fig. 4).

4. Discussion

In spite of recent diagnostic and therapeutic improvements; AMI is a major mortality cause of morbidity and mortality rate. Recently, B-type natriuretic peptide (BNP) has been recognized in many studies as a sensitive marker for prognostication of acute and chronic left ventricular failure. In patients developed an acute ST elevation myocardial infarction (STEMI), and had higher levels of BNP would have worse outcome (Harrison et al., 2002, Tapanainen et al., 2004, Heeschen et al., 2004, Omland et al., 2002; Galvani et al., 2004).

ICD proved to be the treatment of choice for patients with high risk for occurrence of potentially fatal ventricular arrhythmias.1–3, and It has been increasingly recognized that ICDs are superior to anti arrhythmic drugs in survivors of cardiac arrest or unstable VT (Yoshimura et al., 1993; Maeda et al., 1998; Maisel et al., 2002).

In certain high-risk groups ICDs, also approved to be more beneficial than drug therapy for primary prevention of SCD (Morrison et al., 2002; Yang et al., 2007; Blangy et al., 2007; Fazlinezhad et al., 2011).

For patients with severe ischemic cardiomyopathy, the population of our study. This is particularly true and Thus, a better risk identification of patients with depressed ventricular function who could benefit from an ICD or, definitely, more importantly, those who are unlikely to get any benefit would be beneficial as regard in cost and also in their