

Do serum B-type natriuretic peptide levels predict short-term mortality and angiographic success?

Original article Grabowski M *et al.* (2004) Serum B-type natriuretic peptide levels on admission predict not only short-term death but also angiographic success of procedure in patients with acute ST-elevation myocardial infarction treated with primary angioplasty. *Am Heart J* 148: 655–662

SYNOPSIS

KEYWORDS B-type natriuretic peptide, primary percutaneous coronary intervention, ST-elevation myocardial infarction

BACKGROUND

Serum levels of the cardiac neurohormone B-type natriuretic peptide (BNP) can predict mortality in patients with acute coronary syndromes. Grabowski *et al.* explored the link between patients' BNP levels immediately after admission for ST-elevation myocardial infarction (STEMI), short-term mortality and outcome of angiographic intervention.

OBJECTIVES

To find out whether levels of BNP at the time of STEMI can be used to predict the success of primary percutaneous coronary intervention and short-term risk of death from any cause.

DESIGN

Between November 2002 and February 2003, men and women admitted for STEMI at the Central University Hospital, Warsaw, were included in this prospective study. Patients were assessed by electrocardiogram immediately after presentation at hospital. Eligible patients had ST-segment elevation of at least 1 mm in at least two leads on the electrocardiogram. Medical history and demographic information were recorded for all patients.

INTERVENTION

Patients gave peripheral blood samples for BNP measurement using the Triage BNP Test (Biosite Incorporated, San Diego, CA, USA) within 15 min of collection. Immediately after assessment, all patients underwent angiography and primary percutaneous coronary intervention. The success of intervention was evaluated

by blood flow grade (Innova 2000 system, General Electrics Company, Fairfield, CT, USA) and coronary blood flow pattern (using the Thrombolysis In Myocardial Infarction [TIMI] blood flow scale). Whether patients were alive or had died from any cause was recorded 42 days after intervention.

OUTCOME MEASURES

The outcomes were death from any cause, failure of percutaneous coronary intervention defined by the no-reflow phenomenon, and BNP levels.

RESULTS

In total, 126 patients (mean age 58.8 years, 28.6% female) with STEMI received angiographic intervention. Patients' median BNP concentration was 100 pg/ml, the mean serum BNP concentration was 183.5 ± 241.3 pg/ml. At 42 days, 10 (7.9%) patients had died. These patients had significantly higher baseline BNP levels than those who survived (584.4 ± 269.1 vs 148.9 ± 206.1 pg/ml, $P < 0.0001$). More patients with BNP concentrations above 100 pg/ml but low TIMI score died than those with concentrations below 100 pg/ml with low TIMI score (1 vs 9, respectively; $P = 0.027$). Patients with high TIMI risk scores (≥ 4) and BNP concentrations > 100 pg/ml had the highest number of deaths (25.8%). Despite successful intervention, the no-reflow phenomenon was seen in 18 patients, of whom 15 had BNP levels > 100 pg/ml (427.3 ± 362.9 vs 142.8 ± 188.3 pg/ml, $P < 0.0001$). From the survival analysis a BNP concentration of 331 pg/ml at admission was shown to have 90% specificity and 87.9% sensitivity for predicting death.

CONCLUSION

Serum BNP concentrations upon admission for STEMI can predict the outcome of percutaneous coronary intervention and early death. Grabowski *et al.* suggest that BNP assays could be used as a tool for risk assessment in patients hospitalized for STEMI.

COMMENTARY

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Following acute myocardial infarction (AMI), BNP levels increase either in a monophasic (peak within first 24 h) or biphasic pattern (peaks within the first 24 h and between 4 and 7 days later).¹ After plaque rupture and thrombus formation, the resultant myocardial ischemia causes alterations in regional myocardial stretch properties and release of BNP. Large areas of ischemia can result in myocardial stunning and either left-ventricular systolic dysfunction or, more commonly, stiffening of the myocardium with an increase in diastolic intraventricular pressure, which leads to active BNP secretion. Therefore, the first peak might represent an acute-phase response to the extent and magnitude of ischemia.² The biphasic pattern is associated with anterior myocardial infarction, larger infarct size, heart failure and ventricular dysfunction. The second peak is, therefore, probably related to infarct expansion and subsequent ventricular remodeling.^{1,3}

Elevated BNP in patients with AMI, measured at various time points (<6 h, 2–7 days and 3–4 weeks) has been linked with increased mortality.^{4,5} In AMI patients treated with fibrinolytic therapy, elevated BNP levels within 6 h of symptom onset are associated with impaired reperfusion and increased short-term mortality.⁴ The elevated BNP levels are probably related to the extent and magnitude of the initial ischemic insult, explaining the association with increased mortality. Elevated BNP 3–4 weeks after an AMI is associated with increased long-term mortality,⁵ probably related to residual infarct size, ventricular remodeling and left-ventricular dysfunction.

Grabowski *et al.* provide important data on the significance of BNP elevations in AMI patients treated with primary percutaneous coronary intervention. Raised BNP level on admission was a powerful independent predictor of both no-reflow after percutaneous coronary intervention and short-term mortality. Patients with BNP elevations had a significantly longer time from symptom onset to treatment (4.4 vs 7.3 h, $P=0.019$) than those with normal BNP levels, which can increase the thrombus burden and influence myocardial reperfusion, thus affecting angiographic success. Patients with elevated BNP were also

more likely than those with BNP ≤ 100 pg/ml to have anterior MI, culprit lesion in the left anterior descending coronary artery and be in Killip class III or IV, suggesting that a larger area of myocardium was at risk. All these factors can influence angiographic outcomes.

The data should, however, be interpreted with caution. The study sample size was small and only 18 patients had no-reflow after percutaneous coronary intervention, limiting the validity of the findings. In addition, only 73% of patients with elevated BNP received stents, compared with 91% patients with BNP < 100 pg/ml. This discrepancy is not clearly explained, despite stents being well known to influence angiographic success in AMI. Finally, since the magnitude of BNP elevation is related to the time of measurement after AMI, the length of time from symptom onset to BNP measurement might have influenced BNP levels in the group with raised BNP values. Despite these limitations the data are provocative and should be studied further.

Where do we stand with BNP measurements in AMI? Although assays are readily available and BNP levels on admission do seem to provide important prognostic information for AMI, the value of this marker in guiding therapeutic decisions is unclear. An appropriate threshold needs to be defined. Before routine BNP measurement in all patients presenting with AMI is recommended, the therapeutic implications of elevated BNP need to be better defined.

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Competing interests

The author declared he has no competing interests.

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PRACTICE POINT

Elevated BNP levels on admission are a powerful and independent predictor of short-term mortality in patients with STEMI, irrespective of reperfusion strategy; further studies are needed to define the therapeutic implications of this finding