University Heart Journal
Vol. 4 No. 2 July 2008

Changes in plasma brain natriuretic peptide in conventional or direct stenting

Saidur Rahman Khan[1], Nuri Kurtoglu[2], Fehime Benli[2], Ahmet Toprak[2], Mehmet Agirbasli[2], and Ahmet Oktay[2]

[1]Department of Cardiology, Ibrahim Cardiac Hospital and Research Institute, Shahbagh, Dhaka.

[2]Department of Cardiology Marmara University School of Medicine, Istanbul, Turkey.

Address for Correspondence:

Dr. Saidur Rahman Khan, Department of Cardiology, Ibrahim Cardiac Hospital and Research Institute, Shahbagh, Dhaka

Email : dr_mishu71@hotmail.com

Abstract

Direct coronary stenting without balloon predilatation may cause lower postprocedural minor myocardial injury in comparison to conventional stenting with balloon predilatation.Plasma brain natriuretic peptide (BNP) is increased in acute myocardial ischaemia. 40 patients with single vessel and single lesion coronary artey disease who underwent elective stent implantation were divided into Group I (n=20) treated with direct coronary stenting (DS) and Group II (n=20) with conventional stenting (CS). Blood samples for Troponin I and CK-MB measurement were obtained at baseline, just after the intervention procedure, 6 hours after and 12 hours after stenting and for BNP levels at baseline, 6 hours and 12 hours after stenting. In Group II there was a significant increase in Troponin I level 12 hours after the procedure (baseline 0.033 ± 0.028 ng/ml vs. 0.084 ± 0.085 ng/ml; $p < 0.05$).Plasma BNP level in Group I decreased significantly both in base line vs. 6 hours after procedure and baseline vs. 12 hours after procedure (105.2 ± 73.7 pg/ml vs. 81.3± 67.1 pg/ml; $p < 0.05$ and 105.2 ± 73.7 pg/ml vs. 89.9 ± 62.3pg/ml ; $p<0.05$ respectively). With direct stenting BNP levels decreased significantly with no change in Troponin I level, while with conventional stenting Troponin I levels increased significantly.

Key words Brain natriuretic peptide, Direct stenting, Conventional stenting.

Introduction

Direct coronary stenting without balloon predilatation has become a popular method now a days in percutaneous coronary intervention techniques. Two recent large randomized trials[1,2] have shown that the direct stenting is technically feasible yielding similar angiographic outcomes as the standard conventional technique with less duration of procedure, less resource utilization, less radiation exposure and a trend towards less major adverse cardiac event (MACE) rate. Concerning post procedural early complication of percutaneous revascularization procedures like transient ischaemia, or minor myocardial injury due to temporary compromisation of threatened coronary circulation during balloon inflation, the conventional method of stenting with balloon predilatation has got potentials of early minor myocardial injury due to micro vascular injury, prolonged ischaemic insult and distal microembolization provoked by predilatation ballooning. As direct stenting does not need balloon predilatation,minor myocardial injury may be reduced or restored by this procedure as shown by some studies[3, 4] These studies have used Troponin T or I and CKMB as cardiac biomarker for minor myocardial injury. In recent days, plasma BNP has been emerged as a very sensitive biomarker for left ventricular function assessment both for diagnostic and prognostic purpose Several studies have shown that BNP level increases in acute myocardial infarction and in acute coronary syndromes suggesting its acute synthesis and release in these conditions and thus acts as an index of the extent or severity of the ischaemic insult as well as the degree of underlying impairment in left ventricular function.[5-8] Considering all these facts, the present study was designed to measure BNP along with other cardiac biomarkers like CKMB and Troponin I over a period of 12 hours undergoing DS or CS in order to assess the level of change of BNP and also CKMB and Troponin I to evaluate the evidences of transient myocardial ischaemia or minor myocardial injury and its subsequent effect on ventricular function.

Materials and Methods

Patient selection

This prospective nonrandomized study consists of 40 patients with coronary artery disease who underwent elective stent implantation. Presence of single lesion of low or intermediate risk type in one major vessel (left anterior descending, left circumflex, right coronary artery, diagonal

branch, obtuse marginal branch), echocardiographically measured left ventricular ejection fraction more than 40% with no evidence of left ventricular hypertrophy (wall thickness of left ventricle <12 mm in the M mode echocardiography) and normal level of troponin T or I and CKMB before stent implantation were included in our study. Patients with renal dysfunction, recent myocarditis, and pericardial disease, cardiomyopathy, or severe valvular disease, cirrhosis of liver were excluded.

The total number of 40 patients who have fulfilled our criteria, were then divided into Group I for direct stenting patients (n = 20) and group II for conventional stenting (n =20). Hospital Ethics Committee has approved the study protocol. All patients signed an informed consent prior to participating in the study.

Study protocol

The patients, selected for elective stent implantation in appropriate single lesion, were evaluated initially by echocardiography (Vingmed, System 3, USA). A cardiologist monitored the presence or absence of major cardiac risk factors and clinical evaluation according to NYHA classification. Basal electrocardiographic (ECG) evaluation was performed in both group of patients included in our study. Continuous ECG monitoring was done during the intervention and up to 12 hours after intervention for exclusion of any acute ischaemic changes. A significant ST-segment depression was defined as horizontal or down sloping depression of ST- segment > 0.1 mV and 80 msec after the J point that persisted more than 1 minute. Systemic blood pressure by brachial cuff and heart rate were also monitored in a regular fashion up to 12 hours after the procedure. Blood samples were drawn from an antecubital vein just before the procedure, just after the procedure and 6 and 12 hours after the procedure for the measurement of CKMB, Troponin I and BNP:

Angiographic and intervention procedure:

Two separate interventional cardiologists have performed the intervention procedure of our study. The standard Seldinger technique was used usually to cannulate the femoral artery and whenever alternative arterial access was needed, the radial artery. PCI was performed with a 0.014″ guide wire after obtaining multiple views of the coronary lesion. Preference of interventionist determined the procedure to be performed by conventional or direct stenting. Initial balloon dilatation in the CS group was carried out with percutaneous transluminal coronary angioplasty balloons inflated at nominal pressures (6-10 atmospheres). Multiple balloon inflations were performed if needed. Stenting in both groups was accomplished with the use of second generation (NIR, SciMed Life System, Maple Grove, MN, USA.), pre-loaded tubular stents, which were implanted at high pressure (> 10 atmospheres). Stent-artery ratio was 1.1:1. DS was accomplished with the delivery balloon and a high-pressure single inflation. Post-dilation was not performed in any patients in either group. Quantitative coronary angiography (QCA) was performed with the use of an automatic edge detection system (General Electric DLX Angiographic Systems, GE Medical Systems Europe, Dedex, France). Successful stent placement was defined as a ≥ 20% reduction in the lumen diameter stenosis, resulting in a final residual stenosis within the stent of <50% by visual estimation with achievement of Thrombolysis in Myocardial Infarction (TIMI) flow grade 3, without in hospital occurrence of death, Q wave myocardial infarction (MI) or a complication requiring immediate coronary artery bypass graft surgery.

In all the procedures, length and diameter of the stent, amount of contrast used, predilatation balloon inflation time and maximal inflation pressure in case of group II with conventional stent and in-stent balloon inflation pressure for both groups were noted.

Medical Therapy:

All the patients received preprocedural oral aspirin (100-325 mg) and a bolus dose of oral clopidogrel (225 mg) with maintenance dose of 75mg clopidogrel (once a day) for 4 weeks at least after the procedure. A bolus dose of 10,000 IU of sodium heparin was administered during the procedure to maintain an activated clotting time > 300 seconds for every case. No patient received glycoprotein IIb/IIIa inhibitor. All the patients in both groups used a beta blocker or a calcium channel blocker and an angiotensin converting enzyme inhibitor in a regular fashion as prescribed previously for coronary artery disease.

Measurement of Brain Natriuretic Peptide, Troponin I and CKMB:

Blood samples for BNP measurement were taken from both group of patients before intervention, 6 and 12 hours after the intervention. Each 5 ml sample of blood was collected by venipuncture from the antecubital vein into EDTA tubes at room temperature. Then each sample was centrifuged and plasma was removed, allocated, and frozen at -70°C before analysis.

Blood samples for measurement of CKMB and Troponin I were taken from antecubital vein before the stenting procedure, just after and 6 and 12 hours after the intervention. CKMB (normal reference value<24 IU/L) levels were measured with a Hitachi 017 (Boeringer, Mandien, Germany) analyzer with the automatic enzyme immunonoassay method. Cardiac Troponin I was measured using the Access Immunoassay Systems (BECKMAN Coulter) with a normal reference value <0.04 ng/mL.

Statistical Analysis

Statistical analysis was performed using the statistical software program (Statistical Package for Social Science Windows; SPSS 11.5, München, Germany). Continuous variables were expressed as the mean ± standard deviation (SD). Categorical variables were expressed as numbers and percentage and compared by the Chi square test. A p value <0.05 was considered statistically significant. Plasma BNP concentration, CKMB and Troponin I were compared over the time course using two-way analysis of variance (ANOVA) repeated measure with student Newman- Keels multiple comparison test.

Results

Baseline clinical characteristics of the patients in two groups are described in Table I.

Table I: Patient Characteristics

Patient characteristics	Group I (DS) (n=20)	Group II (CS) (n=20)	P value
Age (years)	57.9± 10.1	57.9± 8.0	NS
Sex (male)	17(85%)	15(75%)	NS
Cardiovascular risk factors			
Dyslipidemia	9(45%)	9(45%)	NS
Diabetes mellitus	4(20%)	1(5%)	NS
Hypertension	10(50%)	8(40%)	NS
Family history of coronary artery disease	5(25%)	7(35%)	NS
Smoking	13(65%)	11(55%)	NS
Echocardiographic parameter			
Left ventricular Ejection Fraction (%)	48± 4%	46± 4%	NS
New York Heart Association Classification			
NYHA Class I	15(75%)	14(70%)	NS
NYHA Class II	5(25%)	6(30%)	NS

NS = nonsignificant (p value > 0.05)

All patients underwent only single vessel single lesion procedure. There were no significant differences in the mean age, gender and presence of cardiovascular risk factors between the two groups. Baseline echocardiographically measured left ventricular ejection fraction was 48% and 46% in group I (DS) and group II (CS) respectively. Left ventricular dimensions were within normal limits in both group of patient. Continuous ECG monitoring showed no significant new ischaemic changes in preintervention and post intervention up to 12 hours period of time. No subjective complaints of angina (post intervention) were noted. There were no significant changes in arterial blood pressure and heart rate in both groups of patients over the time course (Table.2 and 3).

Table II: Homodynamic parameters in Group I (DS)

Homodynamic parameters	Group I (DS) n = 20				
	Baseline	Post intervention	6 hours after intervention	12 hours after intervention	P value
Heart rate (beats/min)	71 ±10	70 ±9	69± 10	70± 8	NS
Systolic blood pressure (mmHg)	125± 15	123± 16	120± 16	120± 14	NS
Diastolic blood pressure (mmHg)	69± 8	67± 9	66±9	68±9	NS

NS = nonsignificant (p value > 0.05)

Table III : Homodynamic parameters in Group II (CS)

Homodynamic parameters	Group I (CS) n = 20				
	Baseline	Post intervention	6 hours after intervention	12 hours after interventi on	P value
Heart rate (beats/min)	70±10	69 ±12	67±10	69 ±18	NS
Systolic blood pressure (mmHg)	120± 15	120± 14	118± 10	117 ±10	NS
Diastolic blood pressure (mmHg)	68± 8	67± 9	66± 11	65± 13	NS

NS = nonsignificant (p value > 0.05)

Percutaneous coronary interventions by DS or CS group of patients were successful, according to the ACC/AHA Guidelines for Percutaneous Coronary Intervention, achieving both procedural and clinical success. The angiographical and interventional data in both group of patients showed no significant differences (Table 4).

There were no significant changes of CKMB level in both groups in the time course. No significant change was observed in Troponin I level in Group I patients with DS but there was significant rise of Troponin I in group II patients 12hours after the intervention while compared with the baseline preintervention level (0.084±0.085 ng/ml vs 0.032±0.028 ng/ml with p value < 0.05). Concerning plasma BNP levels, Group I patients with DS have shown significant decrease of BNP level both in 6 hours and 12 hours after the DS implantation with p value <0.05 while compared with the basal level.

Table IV : Angiographic and Interventional Parameters

Angiographic and Interventional parameters	Group I (DS) n = 20	Group II (CS) n = 20	P value
Vessel of intervention			
Left anterior descending artery	8(40%)	10(50%)	**NS**
Left Circumflex artery	2(10%)	3(15%)	**NS**
Right Coronary	8(40%)	6(30%)	**NS**
Diagonal branch	-	-	**NS**
Obtuse marginal branch	2(10%)	1(5%)	**NS**
Type of lesion			
Low risk	14(70%)	15(75%)	**NS**
Intermediate risk	6(30%)	5(25%)	**NS**
Stenosis (%)	83.00±3.14	84.00±3.40	**NS**
Procedural variables of stent implantation			
Stent length (mm)	10.9±2.5	12.4±4.3	**NS**
Stent diameters (mm)	3.1±0.3	2.9±0.4	**NS**
Maximal in stent inflation pressure (atmosphere)	10.4±1.5	10.4±1.3	**NS**
Predilatation parameters in CS			
Total inflation time (seconds)	-	25.0±5.7	**NS**
Maximal balloon inflation pressure (atmosphere)	-	9.9±2.2	**NS**
Amount of contrast media (ml)	186.0±10.5	200.0±10.6	**NS**

NS = nonsignificant (p value > 0.05)

No significant change observed in BNP level in group II patients with CS along its time course up to 12 hours after the intervention. Biochemical parameters are shown in Table (5-6)

Table V-A : Biochemical parameters: CKMB and Troponin I

Biochemical parameters	Group I (DS) n = 20				
	Base line	Post intervention	6 hours after intervention	12 hours after intervention	P value
CKMB (IU/L)	22.05±7.78	20.95±7.06	23.55±8.43	22.65±6.57	**NS**
Troponin I (ng/ml)	0.030±0.025	0.029±0.021	0.031±0.024	0.033±0.030	**NS**

NS = no significant (p value > 0.05)

Table V-B : Biochemical parameters: CKMB and Troponin I

Biochemical parameters	Group II (CS) n = 20				
	Base line	Post intervention	6 hours after intervention	12 hours after intervention	P value
CKMB (IU/L)	21.90±10.83	19.45±9.41	19.60±10.59	21.60±13.50	**NS**
Troponin I (ng/ml)	0.033±0.028	0.035±0.03	0.039±0.034	0.084±0.085*	**NS**

* = P < 0.001 12 hours after intervention us baseline Troponin I level is statistically significant NS = no significant (p value > 0.05)

Table – 6: Biochemical parameters: BNP level

Group	Baseline BNP (pg/ml)	6 hours after intervention BNP 9pg/ml)	12 hours after intervention BNP (pg/ml)	P Value
Group I (DS) (n = 20)	105.2±73.7	81.3±67.1*	89.9±62.3?	0.007
Group II (CS) (n = 20)	89.1±54.4	90.4±64.4	92.8±46.9	0.555

* = P < 0.01 6 hours after intervention vs. baseline BNP level is statistically significant. ♥= P < 0.05 12 hours after intervention vs. baseline BNP level is statistically significant.

Discussion

The extensive use of stents in the treatment of coronary artery disease led interventional cardiologists to simplify the procedure by introducing the concept of stenting without predilatation i.e. the direct stenting. Several studies have confirmed the safety and feasibility of this modified procedure with success rates greater than 90%. Two large randomized studies [1,2] have shown that there are significant reductions in procedure time, fluoroscopy time, use of contrast and over all procedural cost in case of direct stenting while compared with conventional stenting with balloon predilatation. One very recently performed randomized trial[9] where sirolimus drug eluting stent have been implanted in small atherosclerotic coronary lesion either by conventional or direct stenting procedure, have suggested that reduced long term restenosis and even a considerable lower major adverse cardiac event (MACE) rate are possible in case of direct stenting. As it is proved that percutaneous coronary intervention by balloon angioplasty and conventional stenting have got early complications like transient myocardial ischaemia or minor myocardial injury, the possible mechanisms of these early complications have been depicted as due to minor vessel trauma, microembolization of plaque micro particles and intravascular friable material, side branch occlusions, coronary dissection, transient abrupt vessel closure and elastic recoil of the lesioned vessel. Several studies [10,11] have shown the change of novel cardiac markers like CKMB and Troponin T or I level after apparently successful balloon angioplasty supporting the fact of procedural minor myocardial injury.

After the advent of direct stenting technique without balloon predilatation, few studies [3,4] have been performed to compare the incidence of minor myocardial injury or transient ischaemia in between the direct stenting and conventional stenting procedure. One of the two large randomized trial [1] described earlier, have suggested that the DS procedure has got the possible advantage of reduced embolization which is one of the major pathophysiologic cause of myocardial injury, whereas the other one[2] depicted that the DS has the advantage of lesser immediate complications due to the shorter procedural time associated with a lesser degree of ischemic insult. Three other previous studies [3,4,12] have compared the DS with CS to find out the early incidence of minor myocardial injury induced by procedural acute transient ischaemic insult affecting the coronary microcirculation. Although one of these two studies has shown the higher incidence of Troponin I release post procedurally in case of CS procedure, in other study there were no significant difference.

Several studies demonstrate the relation of B-type natriuretic peptide levels with the severity of heart failure, and it underscores the prognostic importance of this peptide in

a number of settings. The value of natriuretic peptides has already been recognized by their inclusion in the recent European guidelines for the diagnosis of chronic heart failure.[13] Being an acute phase reactant; BNP has been emerged as an excellent diagnostic and prognostic marker supported by several recent studies, in acute coronary syndromes [14,7], and in acute myocardial infarction.[5,6] B-type natriuretic peptide has a putative role in the counter regulatory response to ischaemia. Therefore, it may act as an index of the extent or severity of the ischaemic insult, as well as the degree of underlying impairment in left ventricular function.[7,8,15,16] So, serial BNP changes along with the other conventional markers like Troponin I or T and CKMB, may be a helpful informative means for post procedural ischaemic insult of cardiac ventricles after stent implantation.. Although two studies [17,18] have been performed to see the changes of BNP or BNP and atrial natriuratic peptide (ANP) immediately after the balloon angioplasty, to our knowledge, no studies have been done to see the serial changes of BNP immediately before and after the stenting procedure either by CS or DS technique.

In our study, we have selected the patients of both groups with conventional and direct stenting with almost homogeneous patient characteristics. Echocardiographic evaluation of left ventricular ejection fraction and left ventricular dimension, angiographic and intervention characteristics like the type of lesion, vessel distribution and stenosis rate, stent length, balloon pressure, etc. had no significant difference in between two groups. The amount of contrast used for intervention was 186.0±10.5 ml in DS group and 200.0±10.6 ml in CS group. There were no significant changes of systemic blood pressure or electrocardiographic changes in both groups of patients along the time course of the procedure and up to 12 hours after the procedure.

In our study, there were no significant changes in CKMB levels in both groups while compared with the respective basal level. These findings resemble the findings of some previous studies [19,20] supporting the fact that in case of minor myocardial injury without any acute complicated ischaemia, CKMB level is not a novel marker to judge the minor ischaemic insult. And as there was no evidence of side branch occlusion in case of stenting in both groups, no rise of CKMB level provides the logical ground for this finding. But in case of Troponin I, CS group of patients have shown a statistically significant increase in Troponin I level while compared with their basal preintervention Troponin I (0.038±0.028 ng/ml VS 0.084±0.085 ng/ml, p value< 0.05). But in case of DS group of patients the serial changes were not significant comparing with the basal level. Several previous studies[21,22] suggested that Troponin I or T in response to transient ischaemic insult or minor myocardial injury is much more a sensitive marker in comparison to CKMB and following conventional stenting Troponin I or T may be raised. In case of CS procedure, this study has supported the previous observations. In our study Troponin I level in case of direct stenting 12 hours after the intervention was a little bit higher (0.033±0.030 ng/ml) in comparison to its pre intervention value of (0.030±0.025 ng/ml) with no statistical significance. This finding has been supported by one recent study, suggesting that the post intervention minor myocardial injury is less in case of direct stenting (DS).

We have measured the plasma BNP level in both group of patient of baseline, before the procedure and 6 and 12 hours after the procedure. We did not measure BNP level just after procedure. One previous study [11] have shown that BNP increased 1.2 fold after left heart catheterization with coronary artery disease, though few other studies have over thrown this concept showing no specific changes of BNP level before and just after coronary angiography. One study [17] even showed that just after the percutaneous balloon angioplasty BNP level has been reached its peak level, whereas in other study peak level of BNP was achieved 24 hours after balloon angioplasty.[18] In our study, we have thought of elimination of the chances of contrast volume loaded changes of this highly sensitive marker, BNP and measured the BNP level 6 hours after the procedure giving time for haemodynamic adjustment. BNP level has been raised minimally in case of group II patients with CS where the BNP level were (89.1±54.4 pg /ml) in baseline, (90.4±64.4 pg/ml) 6 hours after intervention and (92.8± 46.9 pg/ml) 12 hours after intervention, with no statistical significance. On the contrary, in group I with DS, plasma BNP level were significantly decreased 6 hours and 12 hours after intervention in comparison to basal BNP level. Considering BNP as a highly sensitive biomarker and its acute diagnostic and prognostic efficacy, we have included the patients in both groups with homogeneous demographic and angiographic characteristics. Our study population has also maintained the homogeneousity in respect to intervention procedural and clinical success parameters, as described in ACC/AHA guidelines for PCI. [23] There were no side branch occlusions in both groups. Considering the serial changes in Troponin I level in both groups, our study has shown the similar results as seen in some previous studies that the transient ischemic insult or minor myocardial injury probably due to distal microembolization is much more prevalent in conventional stenting group and DS has got the potential advantage of lowering this minor myocardial injury. This observation has been supported by the serial changes of BNP level in our study as previous studies have suggested its secretion and release in response to transient ischemic insult in an acute manner without any change of haemodynamic variables. Statistically significant decrease in BNP level with no statistically significant change in Troponin I level up to 12 hours after intervention may explain decreased evidence of minor myocardial injury in DS group in our study. On the other hand in CS

group we have expected a significant rise in BNP level in response to higher evidence of minor myocardial injury as evidenced by the significant rise of Troponin I level 12 hours after the intervention procedure. The same thought was applicable for DS group of patients where logically the significant decrease of plasma BNP level is not only a suggestive evidence of lower incidence of minor myocardial injury but also, at the same time a clue to acute improvement of left ventricular functional status for what BNP is an established biomarker. One recent study [24] has shown that conventional stenting with balloon predilatation in comparison to balloon PTCA has significantly improved the left ventricular diastolic function within 48 hours after intervention due to increased coronary flow reserve and improved coronary microcirculation. Several studies [25,26] have also shown the immediate antiischaemic effectiveness of stenting in left ventricular systolic and diastolic function by increasing the coronary flow reserve and improving the coronary microcirculation thus decreasing the area of ischaemic myocardium. So, in our study, we have hypothesized that in CS group, immediate effective establishment of coronary circulation of the lesioned vessel may have the role of not reaching expected significantly increased level of BNP despite of higher evidence of minor myocardial injury. The significant decrease in BNP level in DS group may also be explained in the same way not only by the lower incidence of the minor myocardial injury, but also by the much more effective early reperfusion of the ischaemic myocardium.

One of the limitations of our study is the small number of patients in each group. We did not have also the invasive hemodynamic measurement of atrial and ventricular pressure. Much more prolonged observation of BNP level at least up to 48 hours might be helpful for better prediction.

In conclusion, the present study demonstrated that direct stenting may reduce the incidence of minor myocardial injury in comparison to conventional method. Early improvement of left ventricular functional status as suggested by the plasma BNP level changes might also be more in direct stenting in comparison to conventional stenting with balloon predilatation.

References

1. L.Martinez Elbal, JM Ruiz-Nodar,J Zueco et al.Direct coronary stenting versus stenting with balloon predilatation:immediate and follow up results of a multicentre, prospective, randomized study.The DISCO trial. Eur Heart J 2002;23:633-40.
2. TRENDS:Tetra Randomized European Direct Stenting Study. TCT Mannual 2002.
3. Timurkaynak T, Ozdemir M, Cengel A. Myocardial injury after apparently successful coronary stenting with or without balloon dilatation: direct versus conventional stenting. J Invas Cardiol 2002; 14: 167-70.
4. Atmaca Y, Ertas F, Gülec S. Effect of direct stent implantation on minor myocardial injury. J Invas Cardiol 2002; 14: 443-46.
5. Horio T,Shimada A, Kohno M, et al.Serial changes in atrial and brain natriuretic peptides in patient with acute myocardial infarction treated with early coronary angioplasty. Am Heart J 1993;126:292-99.
6. Arakawa N,Nakamura M,Aoiki H.Plasma brain natriuretic peptide concentration predict survival after acute myocardial infarction. J Am Coll Cardiol 1996;27:1656-61.
7. de Lemons JA, Morrow DA, Benfiey JH, et al. The prognostic value of B-type natriuretic peptide in patients with acute coronary syndrome. N Engl J Med. 2001; 345: 1014-21.
8. Sabatine MS,Morrow DA,de lemos JA,et al.Elevation of B-type natriuretic peptide in the setting of myocardial ischaemia. Circulation 2001;104(suppl II):II-485.abstract.
9. Joachim schofer,Michael Schluter,Anthony H Gershlick, et al.Sirolimus eluting stents for treatment of patients with long atherosclerotic lesions in small coronary arteries;double blind ,randomised controlled trial (E-SIRIUS).Lancet 2003;362:1093-99.
10. Talasz H, Genser N, Mair J, et al. Side branch occlusion during percutaneous transluminal coronary angioplasty. Lancet 1992; 339:1380-82.
11. Noll B, Goke B, Simon B. Maisch B: Cardiac natriuretic peptide hormones during cardiac pacemaker stimulation and left heart catheterization. Clin Invest 1992; 70:1057-60.
12. Atmaca Y, Altin T, Ozdol C, et al. Direct stent Implantation in Acute Coronary Syndrome. J Invas Cardiol 2002; 14: 308-12.
13. Remme WJ,Swedberg K.Guidelines for the diagnosis and treatment of chronic heart failure. Eur Heart J 2001;2:1527-60.
14. Kikuta K,Yasue H,Yoshimura M,et al.Increased plasma levels of B-type natriuretic peptide in patients with unstable angina. Am Heart J1996;132:101-07.
15. Canon CP,McCabe CH,Wilcox RG,et al.Oral glycoprotein IIb/IIIa inhibition with orbofiban in patients with unstable coronary syndromes(OPUS-TIMI 16)trial. Circulation 2000;102:149-56.
16. 79. 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;105:1760-63.

17. Tateishi J,Masutani M,Ohyanagi M.Transient increase in plasma brain (B- type) natriuretic peptide after percutaneous transluminal coronary angioplasty. Clin Cardiol 2000;23:776-80.
18. Kyriakides ZS,Markianos M,Michalis L.Brain natriurtic peptide increases acutely and much more prominently than atrial natriuretic peptide during coronary angioplasty. Clin Cardiol 2000;23:285-88.
19. Hoffmann R, Minz GS, Dusaillant GR, Popma JJ, Pichard AD, Satler LF, Kent KM, Griffin L, Leon MB. Patterns and mechanisms of in-stent restenosis. A serial intravascular ultrasound study. Circulation 1996; 94:1247-54.
20. Bonan R, Paiement P, Scortichini D, Cloutier MJ, Leung TK, Coronary restenosis in a swine model:evaluation of restenosis injury index. Am Heart J 1993; 126:1334-40.
21. Karim MA, Shinn M, Oskarsson H, et al. Significance of troponin T and CKMB mass release after percutaneous transluminal coronary angioplasty. Am J Cardiol 1995; 76:521-23.
22. Ohnishi N, Iwasaki K, Kusachi S, et al. Low incidence of minor myocardial damage associated with coronary stenting detected by serum troponin T comparable to that with baloon coronary angioplasty. Jpn Heart J 1998; 39:139-46.
23. ACC/AHA Guidelines for Percutaneous Coronary Intervention(Revision of the 1993 PTCA guideline). J Am Coll Cardiol 2001;37:2239 i-lxvi.
24. Christiana M, Schannwell, Markus Schneppenheim. Parameters of left ventricular diastolic function 48 hours after coronary angioplasty and stent implantation. J Invas Cardiol 2003; 15: 326-33.
25. Wilson RF,Jhonson MR,Marcus ML, et al.The effect of coronary angioplasty on coronary flow reserve .Circulation 1988;77:873-85.
26. Lewis JF,Verani MS,Poliner LR,et al.Effects of transluminal coronary angioplasty on left ventricular systolic function at rest and during exercise.Am Heart J 1985;109:792-98.

Corrigendum

Trimetazindine MR in Patients with Stable Angina in Bangladesh
TRIUMPH-BANGLA Study Group
MN ISLAM, MA BASHAR, RS MAHMUD, N FARID, AW CHOWDHURY,
KHQ ISLAM, MS BARI, SC DHAR, MS HOQUE, MT RAHMAN,
AK CHOUDHURY, TC GHOSE, PK DAS, TF KAHN

January 2008, Vol. 4. Number I, Page 9-13.
Several printing mistakes was detected after printing the article.
A corrected version of the article has been published as reprint.

Editor