VIA MEDICA

ORIGINAL ARTICLE

Cardiology Journal
2008, Vol. 15, No. 3, pp. 226–229

ISSN 1897–5593

Outcome of patients with stable angina pectoris treated with or without percutaneous coronary intervention

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Abstract

Background: *To assess the outcome of patients with stable angina pectoris treated with percutaneous coronary intervention versus medically treated patients.*

Methods: *Eighty patients with stable angina pectoris and coronary stenosis as confirmed in coronary angiography were treated with (n = 31) or without (n = 49) percutaneous coronary intervention in our department. All patients received optimal medical therapy and were followed up for a period of 24 months.*

Results: *Baseline clinical characteristics, including risk factors of coronary heart disease and coronary lesion type did not differ between the two groups (all p > 0.05). There was no significant difference in major adverse cardiac events (22.4%* vs. *22.6%) during the 24 month follow-up between the two groups (p > 0.05).*

Conclusions: *Percutaneous coronary intervention did not provide extra benefit in this group of patients with stable angina pectoris receiving standard medical treatment in terms of 24 months major adverse outcomes.* (Cardiol J 2008; 15: 226–229)

Key words: stable angina pectoris, percutaneous coronary intervention, medication, outcome

Introduction

Percutaneous coronary intervention (PCI) is commonly used in patients with acute coronary syndrome and patients with stable angina pectoris. Although PCI reduces the incidence of death and myocardial infarction in patients who present with acute coronary syndromes [1, 2], the long term effects of PCI in patients with stable angina pectoris need further elucidation [3]. The present study was designed to assess the long term outcome in patients with stable angina pectoris treated with or without PCI on top of optimal medical therapy. The primary end-point was major adverse cardiac event including: fatal or non-fatal myocardial infarction, death of any cause, repeat target lesion revascularization (either by PCI or coronary artery bypass graft surgery) and re-hospitalization.

Methods

Study population

Patients with stable angina pectoris and proximal coronary stenosis of at least 70% in at least one of major epicardial coronary arteries, who were admitted to our hospital from June 2003 to June 2005 were included in this study. Exclusion criteria included acute coronary syndrome, refractory heart failure or cardiogenic shock, ejection fraction of less than 30%, or revascularization within the preceding 6 months. The study was approved by the institutional ethics committee for human subjects. Informed consent was obtained from all patients. A total of 80 patients were included in this study. Thirty one patients received PCI on top of standard medical treatment and 49 patients received standard medical treatment alone. Patient selection for

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Received: 3.09.2007 Accepted: 12.02.2008

PCI was based on patient preference rather than clinical or angiographic data. All patients were receiving optimal medical treatment and all patients completed the 24-month follow-up.

According to current guidelines, patients undergoing PCI received aspirin and clopidogrel. Patients implanted with bare metal stents [BMS, Mutilinkvision, zata (Guidant), Coroflex/Theca (Braun), S7 (Medtronic)] were treated with clopidogrel (75 mg/d) for at least 3 months and patients implanted with drug eluting stents [DES, Cypher (Cordis), Firebird (Microport)] were treated with clopidogrel (75 mg/d) for at least 9 months and all patients received life-long aspirin (100 mg/d) if not contraindicated.

Percutaneous transluminal intervention procedure

Percutaneous coronary intervention procedures were performed according to standard clinical practice either by radial or by femoral approach. Lesion length, % of stenosis and lesion type were evaluated with on-line quantitative coronary angiography according to ACC/AHA guidelines [4]. All patients received aspirin (100–300 mg/d), clopidogrel (300 mg/d) and nitrate before the procedure, and 10,000 IU of heparin was administered intravenously at the beginning of the procedure, followed by additional boluses as needed to maintain the clotting time at 300 s.

Follow-up

All patients were seen in an outpatient facility or contacted by telephone monthly after discharge. Data on major adverse cardiac events (MACE), including fatal or non-fatal myocardial infarction, death of cardiac or non-cardiac origin, repeat target lesion revascularization by PCI or coronary artery bypass graft surgery and re-hospitalization were collected.

Statistic analysis

Continuous variables were expressed as mean ± SD and unpaired Student's t-test was used for comparison of continuous parameters between groups. Categorical data were compared using the χ^2 test. Results were considered statistically significant when p value was < 0.05.

Results

Baseline characteristics

Age, gender and risk factors for coronary heart disease were similar between the two groups (Table 1).

Table 1. Baseline clinical characteristics.

	Medication (n = 49)	PCI (n = 31)
Age (year)	65.8 ± 5.8	64.4 ± 5.4
Male	28 (57.1%)	17 (54.8%)
Hypertension	39 (79.6%)	26 (83.9%)
Diabetes	10 (20.4%)	8 (25.8%)
Hyperlipidemia	43 (87.8%)	27 (87.1%)
Smoking	18 (36.7%)	12 (38.7%)
Family history	13 (26.5%)	10 (32.3%)

PCI — percutaneous coronary intervention

Angiographic characteristics

Successful coronary angiogram was achieved in all patients without major complications.

Number of diseased vessels, lesion type and location, % of stenosis and stenosis length were all comparable between the two groups (Table 2).

Stent implantation

Thirty-one out of 80 patients underwent stent implantations (14 with single vessel disease, 8 with double vessel disease and 9 with triple vessel disease). Fifty-eight stents were implanted in 54 lesions [19 DES stents in 17 lesions (3 for type A lesion, 10 for type B, 4 for type C) and 39 BMS stents in 37 lesions (29 for type A, 7 for type B, 1 for type C)]. Type B and C lesions were implanted chiefly with DES while most of type A lesions were implanted

Table 2. Angiographic characteristics.

	Medication (n = 49)	PCI (n = 31)
No. of diseased vessels:		
One	23 (46.9%)	14 (45.2%)
Two	13 (26.5%)	8 (25.8%)
Three	13 (26.5%)	9 (29.0%)
Lesion type:		
A	29 (44 ± 57.1)	19 (32 ± 59.3)
B	13 (24 ± 31.2)	8 (17 ± 31.5)
C	7 (9 ± 11.7)	4 (5 ± 9.3)
Lesion location:		
LAD	36 (38 ± 49.4)	25 (25 ± 46.3)
LCx	15 (17 ± 22.1)	10 (14 ± 25.9)
RCA	17 (22 ± 28.6)	13 (15 ± 27.8)
Stenosis (%)	75 ± 6.8	77 ± 6.0
Lesion length [mm]	13.58 ± 6.29	13.47 ± 5.52

PCI — percutaneous coronary intervention; LAD — left anterior descending; LCx — left circumflex; RCA — right coronary artery

Table 3. Stent implantation.

	DES (n = 19)	BMS (n = 39)
Lesion type:		
A	3 (15.8%)	29 (74.3%)*
B	12 (63.2%)	9 (23.1%)*
C	4 (21.1%)	1 (2.6%)*
Lesion location:		
LAD	9 (47.4%)	18 (46.2%)
LCx	5 (26.3%)	10 (25.6%)
RCA	5 (26.3%)	11 (28.2%)
Stenosis (%)	78 ± 6.2	76 ± 5.8
Lesion length [mm]	17.57 ± 6.08	11.47 ± 4.96*
Stent length [mm]	19.95 ± 5.49	13.87 ± 4.62*
Stent diameter [mm]	2.95 ± 0.23	3.08 ± 0.29
Reference vessel diameter [mm]	3.06 ± 0.24	3.27 ± 0.28

*p < 0.05 *vs*. DES; DES — drug eluting stent; BMS — bare metal stent; LAD — left anterior descending; LCx — left circumflex; RCA — right coronary artery

with BMS (all p < 0.05, Table 3). Lesion location, % of stenosis, reference vessel diameter and stent diameter were comparable between patients who received BMS and DES, while lesion length and stent length were significantly greater in DES group (p < 0.05, Table 3).

Medication and follow-up results

Medical treatment was similar between the two groups except that there was significantly more nitrate use in the medical treatment group during the 24-month follow-up period (Table 4). Mortality rate during follow-up was similar between the two groups (p > 0.05); 1 patient in the PCI group died of cerebral stroke (1/31, 3.2%) at 19 months post PCI and 3 patients (3/49, 6.1%) in the medical treatment group died of acute myocardial infarction (at 12 months), lung cancer (at 16 months) and cerebral stroke (at 18 months), respectively. Incidence of total MACE was similar between the two groups (p > 0.05, Table 5). Incidence of total MACE between BMS and DES was also similar (3/9 *vs*. 4/18, p = 0.384).

Discussion

Our study demonstrates that the clinical efficiency of optimal medical treatment versus PCI on top of medical treatment is similar during the 24-month follow-up in patients with stable angina pectoris.

Table 4. Drug use in-hospital and during follow up

	Medication only (n = 49)		PCI (n = 31)	
	In-hospital	24 months	In-hospital	24 months
Nitrates	44 (89.8%)	36 (73.5%)	28 (90.3%)	17 (54.8%)*
Beta-blocker	27 (55.1%)	27 (55.1%)	18 (58.1%)	17 (54.8%)
Aspirin	49 (100%)	49 (100%)	31 (100%)	31 (100%)
Ca-antagonist	24 (49%)	24 (49%)	14 (45.2%)	14 (45.2%)
ACEI/ARB	43 (87.8%)	42 (85.7%)	29 (93.5%)	28 (90.3%)
Statin	47 (95.9%)	46 (93.8%)	30 (96.7%)	30 (96.7%)

*p < 0.05 vs. medication; PCI — percutaneous coronary intervention; ACEI — angiotensin-converting enzyme inhibitors; ARB — angiotensin receptor blocking agents

Table 5. Clinical follow up

	Medication (n = 49)		PCI (n = 31)	
	12 month	24 month	12 month	24 month
MACE	8 (16.3%)	11 (22.4%)	4 (12.9%)	7 (22.6%)
Fatal myocardial infarction	1 (2.0%)	1 (2.0%)	0 (0%)	0 (0%)
Nonfatal myocardial infarction	0 (0%)	1 (2.0%)	0 (0%)	0 (0%)
Noncardiac death	0 (0%)	2 (4.1%)	0 (0%)	1 (3.2%)
Re-hospitalization	7 (14.3%)	8 (16.3%)	4 (12.9%)	6 (19.4%)

PCI — percutaneous coronary intervention; MACE — major adverse cardiac events

The reason for the absence of the extra benefits in patients with stable angina pectoris receiving PCI could be partly explained by the plaque characteristics in these patients. It was shown that coronary plaques in patients with stable angina pectoris are mostly stable plaques characterized by thick fibrous caps, small lipid cores, more smooth-muscle cells, fewer macrophages, and more collagen. Such lesions typically result in ischemia and stable angina symptoms but rarely cause acute coronary syndromes [5, 6]. It is plaque characteristics rather than the degree of stenosis that are the major determinants of the development of an acute coronary syndrome. In another respect, the rapid progress of modern medical therapy for the treatment of coronary heart disease significantly improved the prognosis. In the preceding 3 decades, multiple large, well designed, randomized clinical trials have established a survival benefit for 4 different classes of drugs among a broad spectrum of coronary artery disease patients. Aspirin; lipid lowering drugs (especially statins); beta-blockers; and angiotensin-converting-enzyme inhibitors and/or angiotensin receptor blocking agents; have all been shown to enhance survival, as well as reduce other objective adverse outcomes of coronary artery disease [7]. Consistent with the recently reported study of 2287 patients [8], our findings support the current guidelines [9, 10], in that the optimal medical therapy without routine PCI can be implemented safely in the majority of patients with stable coronary artery disease. Moreover, experimental and clinical studies have shown the beneficial role of modern medical therapy on plaque stability [11]. It is of note that a subset of patients with stable angina pectoris might need PCI for symptom control and some patients might develop acute coronary syndrome which also warrant PCI during the disease process and these patients need to be carefully monitored and PCI should be performed when indicated.

Most of the patients in the present study were aged around 65 years and hence most patients were free of active occupational stress (in contrast to younger patients with initial diagnosis of acute coronary syndrome who are usually treated with PCI). Some factors might have influenced patient preference towards the PCI treatment: fear of PCI, physical activity level, financial as well as educational status. Subgroup analysis might have been helpful to elucidate the possible associations concerning patient preference with regard to PCI treatment, however, this could not be answered by the present study due to limited patient number.

Conclusions

In conclusion, at present, medical therapy is a safe and effective treatment option for patients with stable angina pectoris and PCI on top of medical therapy does not provide extra benefit in these patients.

Acknowledgements

The authors do not report any conflict of interest regarding this work.

References

1. Fox KA, Poole-Wilson PA, Henderson RA et al. Interventional versus conservative treatment for patients with unstable angina or non-ST-elevation myocardial infarction: the British Heart Foundation RITA 3 randomised trial. Randomized Intervention Trial of unstable Angina. Lancet, 2002; 360: 743–751.
2. Fox KA, Anderson FA, Dabbous OH et al. Intervention in acute coronary syndromes: do patients undergo intervention on the basis of their risk characteristics? The Global Registry of Acute Coronary Events (GRACE). Heart, 2007; 93: 177–182.
3. Morrison D. PCI versus CABG versus medical therapy in 2006. Minerva Cardioangiol, 2006; 54: 643–672.
4. Smith SC Jr., Dove JT, Jacobs AK et al. ACC/AHA Guidelines for Percutaneous Coronary Intervention (revision of the 1993 PTCA guidelines) — executive summary: A report of the American College of Cardiology/American Heart Association task force on practice guidelines (Committee to revise the 1993 guidelines for percutaneous transluminal coronary angioplasty) endorsed by the Society for Cardiac Angiography and Interventions. Circulation, 2001; 103: 3019–3041.
5. Ambrose JA, Winters SL, Arora RR et al. Angiographic progression of coronary artery disease and the development of myocardial infarction. J Am Coll Cardiol, 1988. 12: 56–62.
6. Little WC, ConstantinescuM, Applegate RJ et al. Can coronary angiography predict the site of a subsequent myocardial infarction in patients with mild-to-moderate coronary artery disease? Circulation, 1988; 78 (5 Part 1): 1157–1166.
7. Naidu SS, Polin GM, Selzer F, Laskey WK, Jacobs AK, Williams DO, Wilensky RL. Outcome of percutaneous coronary intervention in unstable angina pectoris versus stable angina pectoris in two different time periods. Am J Cardiol, 2006; 98: 447–452.
8. Boden WE, O'Rourke RA, Teo KK et al. Optimal medical therapy with or without PCI for stable coronary disease. N Engl J Med, 2007; 356: 1503–1516.
9. Gibbons RJ, Abrams J, Chatterjee K et al. ACC/AHA 2002 guideline update for the management of patients with chronic stable angina — summary article: A report of the American College of Cardiology/American Heart Association Task Force on practice guidelines (Committee on the Management of Patients With Chronic Stable Angina). J Am Coll Cardiol, 2003; 41: 159–168.
10. Smith SC Jr., Feldman TE, Hirshfeld JW Jr. et al. ACC/AHA/ /SCAI 2005 Guideline Update for Percutaneous Coronary Intervention — summary article: A report of the American College of Cardiology/American Heart Association task force on practice guidelines (ACC/AHA/SCAI writing committee to update the 2001 guidelines for percutaneous coronary intervention). J Am Coll Cardiol, 2006; 47: 216–235.
11. Mezzetti A. Pharmacological modulation of plaque instability. Lupus, 2005; 14: 769–772.